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MR Imaging and Electrophysiological Evaluation in Carpal Tunnel Syndrome

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The objective of this study was to compare the MRI findings of wrists in patients diagnosed with CTS with those of the healthy controls, and to evaluate the correlation between the MRI differences and the electrophysiological findings in the patient group. This study involved 55 wrists, 30 of which were clinically and electrophysiologically diagnosed with CTS and 25 healthy controls. These 55 wrists were evaluated electrophysiologically, and in terms of median nerve diameter, ratio of median nerve diameter at psiform bone level to distal radio-ulnar joint level, the flexor retinaculum bulging ratio and the median nerve intensity by MRI.

When the patient group, which were clinically and electrophysiologically diagnosed with CTS, and the healthy control group were compared, a significant difference ($p<$ 0.001) was observed between the two in terms of median nerve diameters (at psiform bone level: 8.47 $\pm$ 1.41mm and 2.91 $\pm$ 1.01 mm, distal radio-ulnar joint level: 4.04 $\pm$ 1.06 mm and 2.42 $\pm$ 0.95 mm), ratio of median nerve diameter at psiform bone level to distal radio-ulnar joint level (2.17 $\pm$ 0.54 and 1.25 $\pm$ 0.12), their flexor retinaculum bulging ratios (26.21 $\pm$ 5.98% and 7.27 $\pm$ 4.53%) and their median nerve intensities. In the patient group, no significant correlation between MRI and the electrophysiological findings was found ($p>0.05$).

According to the data obtained from the study, we believe that the MRI examination of structural changes that occur in the carpal tunnel, neighboring structures and the median nerve would be useful in the diagnosis of CTS, especially in cases with suspected clinical and electrophysiological diagnosis.

Key Words: Carpal tunnel syndrome, magnetic resonance imaging, electrophysiology

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INTRODUCTION

Although clinical diagnosis and electrophysiological methods have been used primarily for the diagnosis of Carpal Tunnel Syndrome (CTS), Magnetic Resonance Imaging (MRI) has recently been used to a greater extent as an important and additional method of diagnosis. Since MRI can apparently define the density differences of soft tissues, it has taken a place among the diagnostic methods of CTS. The possibility of detecting the median nerve and structural changes in the region by MRI has an important place in CTS diagnosis and in the examination of etiological factors, especially inflammatory, systemic, metabolic and physiological reasons for the structural changes in the region, which might give rise to non-specific tenosynovitis.[1] MRI imaging is the best way of imaging CTS, as it can define the locus of entrapment in the carpal tunnel.[2]

The objective of this study was to compare MRI findings of the wrists of CTS patients with those of a healthy control group, and to evaluate the correlation between the MRI differences and the electrophysiological findings in the patient group.

MATERIALS AND METHODS

The study involves 30 wrists, which were clinically and electrophysiologically diagnosed with CTS, and 25 healthy controls.

At clinical diagnosis, the following recruitment criteria[3] were used.

- Sensory disorders in the median sensory innervation region (thumb,index and middle fingers) or complaints of pain
- Increase of complaints with repeated flexion and extension movements of wrists at night.
- Positive Tinnel's sign.
- Positive Phalen test results.
- Thenar atrophy.

The following criteria were used for the electrophysiological diagnosis of CTS:

In sensory nerve conduction studies[3-5];
- Median sensory nerve action potential (SNAP) peak distal latency of longer than 3.2 msec,
- In the ring electrodes recording of 2nd and 5th digits, the difference between the SNAP peak distal latencies of the median and ulnar nerves was 0.5 msec or longer,
- During the examination of the 4th digit, the difference between the SNAP peak distal latencies of the median and ulnar nerves was 0.4 msec or longer,
- The speed of median nerve sensory conduction was slow (<50 m/s) in the wrist segment.

In the motor nerve conduction studies[6];
- Median nerve compound muscle action potential (CMAP) distal latency of longer than 4.2 msec.
- The speed of median nerve motor conduction was slow (<50 m/s) in the wrist segment.
- Spontaneous pathological activity was recorded in the needle EMG of the abductor pollicis brevis muscle.

Cases that had at least one of the criteria of clinical, sensory or motor nerve conduction were included in the study as having a diagnosis of CTS.

Cases that had been diagnosed with polineuropathy, plexopathy, radiculopathy or proximal compression syndrome by clinical and/or electrophysiological examination were excluded from the study. The electrophysiological assessment was carried out with the Neurostar, Medelec MS 92 B, Oxford Instruments, London.

In nerve conduction studies, the sweep speed was 50 msec/div., 10 msec/div., 10-100 msec/div.; and the sensitivity was 5 mv/div, 20 μv/div, 1000 μv/div. (in motor, sensory nerve conduction studies and needle EMG, respectively)

In median nerve sensory conduction studies, stimulation was made at the 2nd digit and recording was made at a distance of 12 cm at the volar surface of the wrist; and in motor nerve studies, stimulation was made at wrist and elbow and recording was made at a distance of 8 cm at the pollicis brevis muscle. In the examination of the 4th digit, stimulation was made at the 4th digit with a ring electrode and recording was made 11 cm distant at the wrist palmar surface medially and laterally. In studies of ulnar nerve conduction, stimulation was made at the 5th digit and recording some 10 cm distant at the wrist volar surface. Palmar stimulation was performed on the hand palmar surface at the 4th metacarpal space and the recording was made some 8 cm distant at the wrist volar surface. Surface electrodes were used for stimulation and recording activities in this study,

- Proton density +T2 turbo spin echo axial.
- T1 spin echo axial.
- T2 turbo spin echo coronal.
- Flash 2D axial, sequences have been applied, by using Magnetom Impact, 1 tesla MRI device.

In the study, preferred parameters were a section thickness of 3 mm, FOV 10 cm, NEX 2, and MATRIX 256X256, using a surface coil. Since there is little fatty tissue in the carpal tunnel, these sequences did not require fat suppression. T1-weighted axial images were obtained in order to evaluate the wrist anatomy in detail, or T2-weighted coronal cross-sections were used to assess the prospective bone pathologies. Because median nerve oedema gives a bright signal (hyperintensity), and possible fluid accumulations in the carpal tunnel can be demonstrated, the T2 sequence was chosen for the examination.

The imaging was performed while the patient lay in the prone position with his arm fixed above and in front of his head in a supine position and the elbow in slight flexion. The patient was given soft pillows when necessary to ensure a comfortable convenient position.

The sections were taken axially towards the wrist and at a slightly oblique angle to compensate for the modest radial or ulnar inclination

of the carpal bones. This best performed by orienting the axial images perpendicular to capitate bone.

The following parameters were evaluated during the MRI examination[2];

-Median nerve diameter: Measured at the levels of the distal radio-ulnar joint and at the psiform bone and the ratio of the two diameters was determined.

- Median nerve density
- Flexor retinaculum bulging ratio: A sraight line (a) between the attachments of the flexor retinaculum to the tubercle of the trapezium and the hook of the hamate. Then, to represent the palmar displacement of the flexor retinaculum, we determined the length of a perpendiculer line (b) from line TH to the palmar apex of the flexor retinaculum. The bulging ratio was calculated as a/b.

Statistical analyses was performed using the independent sample t test, Pearson Chi-square and Pearson correlation tests, using the SPSS (Statistical Package for Social Sciences) programme.

RESULTS

The mean age of the CTS diagnosed patient group was 48.69 ± 2.12, and there were 28 women and 2 men. The mean age of the control group, including 25 volunteers was 50.20 ± 8.21 and there were 23 women and 2 men. No statistically significant difference was observed between the two groups in terms of age and gender ($p > 0.05$). However, there was a statistically significant difference between them in terms of median nerve diameters (at the levels of the psiform bone and at the distal radio-ulnar joint), the diameter ratios and the flexor retinaculum bulging ratios ($p < 0.001$) (Table 1). The intensity evaluation, showed hyperintensity in 25 of 30 wrists of the patient group and 5 were isointense; and hyperintensity in 4 of 25 controls and isointensity in 21 ($p < 0.001$) (Table 2). The study found no correlation between the median nerve diameter or the flexor retinaculum bulging ratio and the SNAP distal peak latency or the SNAP amplitude (Table 3).

Table 1. Comparison of Measurement Results Obtained by MRI in the Patient and Control Groups

Median nerve diameter (mm)	Case group (Medium± SD)	Control group (Medium ± SD)	p
PS	8.47 ± 1.41	2.91 ± 1.01	<0.001
RU	4.04 ± 1.06	2.42 ± 0.95	<0.001
PS / RU rate	2.17 ± 0.54	1.25 ± 0.12	<0.001
FRB (%)	26.21 ± 5.98	7.27 ± 4.53	<0.001

PS, Median nerve diameter at psiform bone level; RU, Median nerve diameter distal radio-ulnar joint level; FRB, Flexor retinaculum bulging ratio.

Table 2. MRI and Intensity Evaluation in Case and Control Groups

MRI evaluation	Case group	Control group	Total
Hyperintensity	25	4	29
sointensity	5	21	26
Total	30	25	55

Pearson Chi-square: 22.26.
$p < 0.001$.

Table 3. MRI and Electrophysiology Correlations

		PS	RU	FRB (%)	SNAP-dl	SNAP-amp
PS	Pearson Correlation		,712	,203	,323	-,188
	Sig. (2-tailed)		,000**	,354	,132	,390
RU	Pearson Correlation	,712		,048	,211	-,311
	Sig. (2-tailed)	,000**		,827	,333	,148
FRB (%)	Pearson Correlation	,203	,048		,024	-,045
	Sig. (2-tailed)	,354	,827		,915	,839
SNAP-dl	Pearson Correlation	,323	,211	,024		-,700
	Sig. (2-tailed)	,132	,333	,915		,000**
SNAP-amp	Pearson Correlation	-,188	-,311	-,045	-,700	
	Sig. (2-tailed)	,390	,148	,839	,000*	

**Correlation is significant at the 0.001 level (2-tailed).
PS, Median nerve diameter at Psiform bone level; RU, Median nerve diameter at joint level; FRB, Flexor retinaculum bulging ratio; SNAP-dl, sensory nerve action potential peak distal latency.
SNAP-amp, sensory nerve action potential amplitude.

DISCUSSION

The realization that MRI is a method of imaging soft tissues has increased its importance in the evaluation of the carpal tunnel and the median nerve.[7] The reason for the use of MRI in the evaluation of CTS in wrists is that it can clearly detect contrast differences in soft tissues.[8]

The possibility to of detecting slight and more dramatic changes in signal density using MRI has increased its utility in cases where electrophysiological examination is negative or suspicious, in the assessment of etiological factors, and in confirmation of electrophysiological diagnoses.

The use of MRI in CTS has at a diagnostic method has increased significantly in recent years. MRI examination is needed for the diagnosis of CTS in cases where clinical symptoms are indefinite and electrophysiological methods are negative.[9,10]

Recently, MRI has been used frequently in dynamic examinations[11,12] to evaluate the canal diameter, and to perform postoperative patient assessments[13,14] and painful wrist evaluations.[15-17]

In the present study, median nerve diameters were measured by MRI at the levels of the psiform bone and the distal radioulnar joint. Mesgarzadeh et. al stated that the imaging of median nerve expansion could best be managed at the level of the psiform bone.[18]

When we measured the median nerve diameters in patients diagnosed with CTS and in the control group at both of these levels, it was observed that the diameter expanded significantly in patients with CTS. The results of median nerve measurement indicated a dramatic diameter expansion of the median nerve in the proximal carpal tunnel, which is consistent with results in the literature. Mesgarzadeh et al.[2] found that this ratios was 2.4 in the patient group and 1.1 in the control group. Monagle et al.[19] found diameter of the median nerve within and proximal to the carpal tunnel was approximately 50% larger in patients with CTS than in asymptomatic subjects. It seems to us that this significant diameter expansion of the median nerve in the carpal tunnel proximally is due to the proximal diffusion of inflammation in the median nerve.

Kleindienst et al.[20] demonstrated that the median nerve significantly expanded in the proximal canal during the early phase. Allmann et al.[21] determined in 1997 that a significant diameter

expansion took place in the median nerve at the levels of the psiform bone and the radio-ulnar joint. Another parameter used in studies is the flattening ratio of the median nerve.[19,20,22] We did not use of this parameter as an evaluation criterion in our study.

A definite bulging in the flexor retinaculum is another important finding in the patient group compared to controls in our study. The bulging ratio was 26.21 ± 5.98% in the patient group and 7.27 ± 4.53% in the control group, and this was significantly different.[18] This result of the flexor retinaculum bulging ratio is identical to that obtained in previous studies.[10,11,13,21,23] The increase in the bulging ratio is presumed to be due to pressure augmentation in the tunnel, and thus to mechanical compression of the nerve.[10] Pressure augmentation is believed to be related to oedema developed secondarily by the compression of the median nerve and compression due to tenosynovitis in tendon sheath. The reason that bulging is most clearly seen at the os hamate level is because this region is a place where the flexor retinaculum is at its thickest. Since the flexor retinaculum can be definitely detected in this region and its borders can easily be followed, the most accurate evaluation can be made at this level. In addition, this level is a region where the carpal tunnel apparently narrows and the median nerve is maximally compressed.[18,19]

We found a striking increase of intensity in median nerve in 83.3% of the CTS cases when we evaluated the median nerve intensity. This rate was only 16% in the control group, and this difference between the two groups was significant. Britz et al. demonstrated a signal increase in the median nerve in 95% of cases clinically diagnosed with CTS.[24] The importance of the intensity increase in the median nerve in subclinical cases and during the early diagnosis of CTS has been emphasized,[20,25] as this increase is believed to accompany cases which have already progressed clinically and electrophysiologically.[10,23] Almann et al.[21] determined that the intensity in the median nerve decreased in 67% of postoperative cases, and Mesgarzadeh et al.[18,26] emphasized the importance of intensity increase in the median nerve in the diagnosis of CTS; they added that this expanded proximally towards the distal radio-ulnar joint in some cases.

In this study, we found that there was no correlation between the bulging ratio of the median nerve diameter or the flexor retinaculum, and the SNAP distal peak latency or the SNAP amplitude (Table 3). A striking point in the present study was the presence of a positive correlation between the median nerve diameters at the levels of the psiform bone and the distal radio-ulnar joint. This correlation could be interpreted as the retrograde swelling of the median nerve just proximal to the carpal tunnel. In many studies, different conclusions have been reached in terms of the correlation between MRI and clinical and electrophysiological findings. In particular, in some studies, no correlation between the MRI parameters, the clinical findings and median nerve electrophysiological dysfunctions were found.[19,27] In addition, the presence of a median nerve intensity increase accompanying serious clinical and electrophysiological findings has been emphasized.[10,22,23]

In 24% of the cases recruited in the study, no possible etiological factor of CTS was detected. However, the median nerve diameter ratios were 1.7 to 2.6 and the flexor retinaculum bulging ratios were 15 to 17%, and all patients showed median nerve hyperintensity. These cases were treated as being idiopathic because they had no associated pathology that might have caused CTS. In other cases, the most frequent etiological reasons were tenosynovitis, ganglion cyst, fat accumulation in the tunnel and effusion. We found more than one etiological factor in some cases. Particularly in cases with tenosynovitis, accompanied the effusion of wrist joints with tenosynovitis. The fact that there was no definite pathology in 24% of cases supports the thesis that CTS is related to congenital stenosis.[29]

In CTS, focal or diffused volume increases of inner-canal structures or the narrowing of the canal radius can seemingly occur without reason. In such cases, the congenital narrowness of the carpal tunnel might cause ischemic alterations to the median nerve. Dekels and Bleecker[28,29] proposed that the congenital narrowing of the carpal tunnel was one of the important reasons for idiopathic CTS. Lundberg et al.[30] indicated that endoneurial oedema development depends on ischemia, and that vascular mechanisms cause the

development of idiopathic CTS.

According to the results of this study, we believe that the imaging of structural alterations in the carpal tunnel, neighbouring structures and of the median nerve by MRI should be used in combination with electrophysiological evaluation in the diagnosis of CTS, in those cases with suspicious clinical and electrophysiological diagnoses.

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