ORIGINAL ARTICLE

The Utility of Ultrasound in Identifying Carpal Tunnel Syndrome among Individuals with Systemic Lupus Erythematosus

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Abstract

Background: ***The most prevalent form of entrapment neuropathy globally is carpal tunnel syndrome (CTS), and its frequency in people with systemic lupus erythematosus (SLE) is similar to that of the general population.***

Aim and Objectives: ***The purpose of this research is to assess how well ultrasonography performs as a CTS diagnostic tool in lupus patients.***

Patients and Methods: ***Sixty SLE patients were included in the study and divided into two groups. Thirty individuals in one group had clinical symptoms of CTS, while thirty patients in the other group did not. Every participant had a comprehensive examination that included electrodiagnostic tests, clinical assessments, and ultrasonographic scanning.***

Results: ***Higher disease activity scores, aberrant nerve conduction study (NCS) results, and a significant increase in the median nerve's cross-sectional area (CSA) as shown by ultrasonography at the carpal tunnel entry were all seen in patients with SLE and CTS. For different CSA cutoff values, the best sensitivity (93.33%) and specificity (100%) were observed at 10.5 mm².***

Conclusion: ***In individuals with lupus who exhibit classic symptoms of CTS, ultrasonography may offer a rapid and accurate method of making the diagnosis.***

Keywords: Ultrasound; NCS; CTS; lupus

1. Introduction

A prevalent condition, CTS significantly impairs the quality of life for people who suffer from it. CTS is caused by a number of variables, including aging, being overweight, and the usage of vibrating tools .[1] Its onset is also significantly influenced by autoimmune illnesses, especially SLE. Autoimmune inflammatory conditions such as SLE are believed to have underlying processes associated with the existence of autoantibodies.[2,3] Nevertheless, the precise causes of neurological dysfunction in autoimmune conditions remain elusive .[4,5] Several factors, particularly alterations in the flexor retinaculum and inflammation of the synovial membrane, are believed to be linked to the development of CTS among individuals with SLE .[6]

A wide range of clinical symptoms that might impact multiple bodily systems are characteristic of SLE .[7] It therefore has the potential to impact both the central and peripheral neurological systems for a long time before additional signs emerge. Furthermore, it is possible for CTS to manifest as a potential initial clinical sign of SLE .[8]

NCS has historically been the main technique used to diagnose CTS .[9, 10] however, tests such as these can produce wildly varying outcomes.[10,11] and may result in a substantial amount of expense and discomfort. Despite the development of several diagnostic techniques and guidelines for determining CTS .[12,13,14] recent evidence indicates that physical examinations by themselves are not enough to properly identify patients for CTS .[15,16]

Accepted 15 April 2025.
Available online 30 June 2025

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https://doi.org/10.21608/aimj.2025.446615

The application of ultrasonography is an additional diagnostic technique .[17]

The image quality of ultrasound scans has been significantly enhanced owing to improvements in ultrasonography transducer innovation. However, there is still much disagreement about the best anatomical locations to assess the median nerve and the proper definitive cutoff threshold for CTS .[10,18,19] The purpose of this study was to assess the diagnostic precision of ultrasonography in detecting CTS in lupus patients as well as its relationship to other diagnostic techniques, including electrodiagnostic testing and clinical evaluations.

2. Patients and methods

We selected a sample of 60 SLE patients from our university hospitals' rheumatology and rehabilitation departments. Based on the existence of clinical symptoms linked to CTS, these individuals were divided into two groups. Thirty patients in the first group, known as the symptomatic group, had clinically verified CTS, whereas thirty patients in the second group did not have any symptoms of CTS.

This study used the CTS-6, a standardized screening tool for CTS created by the American Association of Surgery. Each of the symptoms was given a value of a single point. A cumulative score of at least two points[20] was used to diagnose symptomatic CTS.

Exclusion from the research was based on predetermined criteria. Individuals who had a history of diabetes, thyroid issues, rheumatic conditions besides lupus, previous fractured wrists or trauma resulting in injury to the nerves, or neurological dysfunction independent of CTS were not considered eligible.

In addition to a comprehensive assessment of their medical history, every individual had a physical examination along with a comprehensive neurological assessment. The degree of disease activity in the individuals with SLE was evaluated using the SLE disease activity index (SLEDAI). Additionally, the Boston Carpal Tunnel Questionnaire, a validated self-identified instrument designed to gauge the extent of discomfort and physical constraints in individuals having CTS, was completed by all individuals with SLE .[21]

Ultrasound Evaluation

For this study, an ultrasound device (APLIO400-Toshiba, California, US) fitted with a LA 3-14 MHz linear transducer was employed. With their hands resting on a firm surface and their fingers slightly bent, the individuals being examined were seated comfortably, confronting the examiner. The median nerve's CSA measurement was taken at its broadest point, immediately before it entered the carpal tunnel (Figure 1). At the hyperechoic epineural border, the ultrasonography measured the CSA perpendicular to the nerve's route in order to record its shape. CSA measures were taken for patients over the course of four months in an outpatient setting by a certified rheumatologist (AIA) who has over a decade of work experience with musculoskeletal ultrasonography.

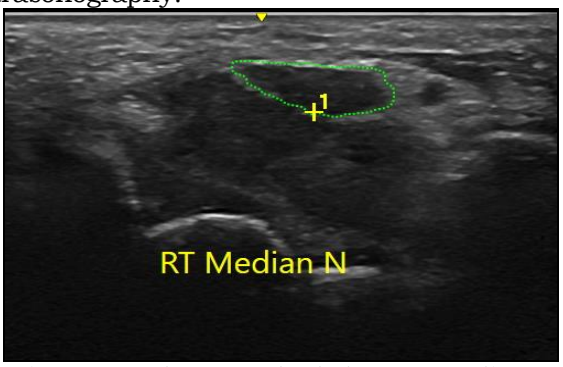

Figure 1. Ultrasound of the RT median nerve depicting the CSA at the carpal tunnel inlet.

Electrophysiological assessments

Electrophysiological evaluations were conducted on a cohort of 60 patients through nerve conduction studies following standard protocols.

This research was sanctioned by the local medical ethics board, and informed consent was secured from all participants before the study commenced.

Statistical Analysis

All data were collected, tabulated, and statistically analyzed using IBM Corp., released in 2015. IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp. For quantitative data after testing normality using the Shapiro-Wilk test: normally distributed data presented with mean ± SD and skewed data presented as median (IQR). Qualitative data were expressed as numbers with percentages. The chi-square and Fisher's exact tests were used for qualitative data, while the independent sample t-test and Mann-Whitney U test were used for quantitative data. Spearman's rank correlation coefficient analysis was used to assess the association between measured parameters. The cutoff value was calculated using ROC to discriminate between symptomatic and asymptomatic SLE patients. All tests were two-sided. P-value ≤ 0.05 was considered statistically significant, p-value > 0.05 was considered statistically insignificant.

3. Results

The research involved 60 individuals diagnosed with SLE, categorized into two groups based on the presence of clinical symptoms of CTS. The initial characteristics of the groups are

summarized in Table 1. There were no significant differences in average age, height, weight, body mass index (BMI), or gender between the two groups (P>0.05). However, the duration of SLE was notably longer in the asymptomatic group compared to the symptomatic group (P=0.01).

Table 1. Demographic data among the studied groups

VARIABLES		SYMPTOMATIC (N=30)	ASYMPTOMATIC (N=30)	P VALUE
AGE *(YEARS)*	*Mean ± SD*	32.9 ± 10.3	36.9 ± 11.1	0.32[1]
	Range	(20 – 47)	(20 – 56)	
SEX *(N. %)*	Male	4 (13.3%)	0 (0%)	
	Female	26 (86.7%)	30 (100%)	0.113
HEIGHT *(CM)*	*Mean ± SD*	169.1 ± 7.52	164.9 ± 4.14	0.07[1]
	Range	(160 – 184)	(158 – 170)	
WEIGHT *(KG)*	*Mean ± SD*	89.3 ± 25.5	77.1 ± 12.4	0.11[1]
	Range	(51 – 128)	(52 – 100)	
BMI *(KG/M²)*	*Mean ± SD*	27.9 ± 5.62	26.9 ± 2.41	0.52[1]
	Range	(18.8 – 37.8)	(23.4 – 30.1)	
DURATION *(YEARS)*	*Median (IQR)*	3 (2.75 – 5)	12 (6 – 12.5)	0.01[2]

*1Independent sample T test, 2Mann-Whitney U test, 3Fisher exact test, Non-significant: P >0.05, Significant: P ≤0.05.

Additionally, there were no notable differences in laboratory results among the groups examined. The levels of ESR, CRP, ANA, anti-dsDNA, and proteinuria did not show any significant variation between the groups of SLE patients. Conversely, the SLEDAI scores were markedly elevated in the symptomatic group compared to the asymptomatic group (P=0.049). In terms of the Boston carpal tunnel questionnaire, both the severity of symptoms and functional status were significantly greater in the symptomatic group relative to the asymptomatic group (P<0.001). (Table 2)

Table 2. Clinical data among the studied groups

VARIABLES		SYMPTOMATIC (N=30)	ASYMPTOMATIC (N=30)	P VALUE
ESR *(MM/HR)*	*Mean ± SD*	49.2 ± 18.3	63.6 ± 20.5	0.052[1]
	Range	(12 – 66)	(30 – 96)	
CRP *(MG/DL)*	*Median (IQR)*	10.1 (6 – 18)	16 (8.95 – 70)	0.22[2]
ANA *(N. %)*	Negative	0 (0%)	0 (0%)	
	Positive	30 (100%)	30 (100%)	1.00[4]
ANTI-DSDNA *(N. %)*	Negative	8 (26.7%)	6 (20%)	
	Positive	22 (73.3%)	24 (80%)	0.67[3]
PROTEINURIA *(MG/DAY)*	*Median (IQR)*	2300 (260 – 2900)	300 (190 – 525)	0.11[2]
SLEDAI	*Median (IQR)*	14 (5.5 – 20)	6 (4 – 9.5)	0.049[2]
SYMPTOMS SEVERITY	*Median (IQR)*	3 (3 – 4)	1 (1 – 1)	<0.001[2]
FUNCTIONAL STATUS	*Median (IQR)*	4 (3 – 4)	1 (1 – 1)	<0.001[2]

*1Independent sample T test, 2Mann-Whitney U test, 3Chi-square test, 4Fisher exact test, Non-significant: P >0.05, Significant: P ≤0.05.

In terms of NCS data, patients with SLE who also had CTS exhibited notably increased median motor latency, reduced amplitude, and decreased conduction velocity compared to those SLE patients without CTS (P<0.001). Additionally, the CSA of the median nerve at the entrance of the carpal tunnel, as measured by ultrasound, was significantly larger in the symptomatic group than in the asymptomatic group (11.1 ± 0.74 vs 8.94 ± 1.02, P<0.001) (Table 3).

Table 3. Nerve conduction study and US findings among the studied groups

VARIABLES		SYMPTOMATIC (N=30)	ASYMPTOMATIC (N=30)	P VALUE
NCS: LATENCY *(MS)*	*Median (IQR)*	5.6 (5.03 – 6.35)	3.58 (3.15 – 3.79)	<0.001[2]
AMPLITUDE *(MV)*	*Median (IQR)*	3.1 (2.45 – 3.58)	6.08 (4.71 – 8.34)	<0.001[2]
VELOCITY *(M/S)*	*Mean ± SD*	35.5 ± 6.81	59.46 ± 5.86	<0.001[1]
	Range	(23 – 45)	(50 – 69.3)	
ULTRASOUND: CSA	*Mean ± SD*	11.1 ± 0.74	8.94 ± 1.02	<0.001[1]
	Range	(10.1 – 12.3)	(7.1 – 10.3)	

*1Independent sample T test, 2Mann-Whitney U test, Non-significant: P >0.05, Significant: P ≤0.05 .

In the correlative analysis, a notable relationship was found between the CSA of the median nerve at the carpal tunnel entrance, and all parameters of NCS. Specifically, the CSA exhibited a strong positive correlation with the motor distal latency of the median nerve (r=0.825, P<0.001), while showing negative correlations with both the median motor amplitude (r=-0.784, P<0.001) and conduction velocity (r=-0.866, P<0.001). Furthermore, a significant positive correlation was observed between the CSA and the (SLEDAI) (r=0.356, P=0.045), as well as with the severity of CTS symptoms (r=0.873, P<0.001) and CTS functional status (r=0.845, P<0.001) (Table 4).

Table 4. Correlation of CSA with NCS, SLEDAI, symptoms severity and functional status among the studied patients

	CSA	
Variable	*r*	*P*
Latency	0.825	<0.001
Amplitude	-0.784	<0.001
Velocity	-0.866	<0.001
SLEDAI	0.356	0.045
Symptoms severity	0.873	<0.001
Functional status	0.845	<0.001

*Spearman rank correlation test, Non-significant: P >0.05, Significant: P ≤0.05 (Bold).

In performing ROC analysis (Receiver Operating Characteristic) to identify the ideal cutoff point for distinguishing between symptomatic and asymptomatic patients, it was found that a median nerve CSA exceeding 10.5

mm² yielded the greatest sensitivity (93.33%) and perfect specificity (100%). This resulted in an area under the curve (AUC) of 0.993, as illustrated in Table 5 and Figure 2.

Table 5. ROC curve analysis of CSA in detecting symptoms

VARIABLES	CUT POINT	SENSITIVITY	SPECIFICITY	PPV	NPV	AUC
CSA	10.5	93.33%	100%	100%	93.75%	0.993

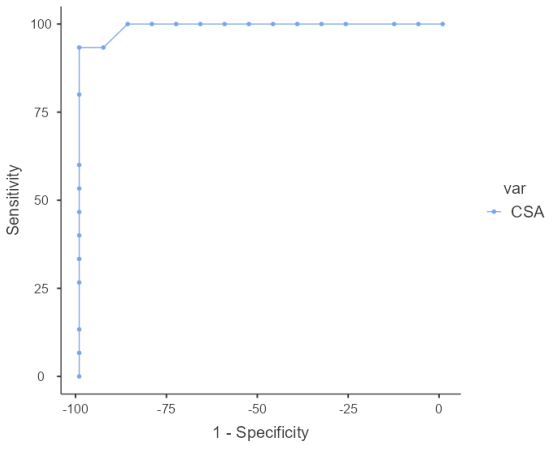

Figure 2: ROC curve analysis of CSA in detecting symptoms

4. Discussion

Among compressive neuropathies, CTS is exceedingly popular and has a major impact on the peripheral nervous system .[22] The majority of diagnoses occur in people between the ages of 40 and 60, and it affects females more often than males .[23, 24] Early peripheral nervous system consequences of SLE have been proposed, and CTS may manifest early in the course of the disease .[25]

Given that there was a significant association between clinically verified CTS and an elevated CSA at the carpal tunnel entry, our research indicates that evaluating the median nerve's CSA by ultrasonography may improve the diagnosis of CTS in patients with SLE. These results are consistent with other research that supports the use of ultrasonography in determining the presence of CTS .[26,27] Multiple research investigations have attempted to develop novel sonographic parameters to facilitate the identification of the median nerve among those with CTS .[22]

Physical trauma or elevated pressure in the carpal tunnel could lead to CTS in individuals with SLE, which might end in entrapment of the median nerve and potential ischemic injury .[28, 29] As a result, the compression in the carpal tunnel could lead to nerve enlargement, which ultrasonography may reveal .[17] In individuals suffering from SLE, CTS could occur as a result of other conditions such as vascular inflammation, amyloidosis, and unfavorable drug reactions .[30] Furthermore, the start of CTS in these individuals may be related to an overabundance of connective tissue growth factor (CTGF) .[31]

According to our receiver operating characteristic (ROC) analysis, a median nerve CSA greater than 10.5 mm² at the carpal tunnel entrance demonstrates good sensitivity as well as specificity, underscoring its diagnostic dependability for CTS. Some researchers advocate a cutoff range of 9 to 11 mm² (32), while others state that the usual CSA range at the carpal tunnel entrance is between 8 and 14 mm² .[19] Several variables, such as image resolution, equipment settings, and individual size, along with assessment methods, may contribute to this heterogeneity.

In this study, the average CSA for the asymptomatic SLE group was 8.94 ± 1.02 mm². However, our research identified a cutoff of 10.5 mm², which yielded the highest sensitivity (93.33%) and specificity (100%), with an area under the curve of 0.993. Furthermore, Ratasvuori et al.[22] proposed that the ideal cutoff should be above 11 mm².

Electrodiagnostic testing is usually performed on individuals suspected of having CTS prior to invasive procedures .[9] Numerous hand specialists argue that neurophysiological testing is not necessary for establishing a diagnosis in all individuals with CTS .[33 -36] When a diagnosis is necessary, ultrasonography delivers an immediate and straightforward substitute. Additionally, electrodiagnostic testing may overlook the pathologic conditions underlying CTS, such as ganglion cysts, which ultrasonography can identify .[22]

4. Conclusion

Our findings suggest that ultrasonography may be an effective alternative to neuropsychological tests for assessing lupus patients with CTS. This study could identify individuals who would benefit from future electrophysiological testing. More research is needed to better understand the relationship between disease severity, ultrasonography findings, and patient satisfaction after treatment.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

Funding

No Funds : Yes

Conflicts of interest

There are no conflicts of interest.

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