Research paper

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Point-of-care ultrasound: a viable alternative for assessing ulnar neuropathy in rheumatoid arthritis?

Adel Azzam, Mohamed Mansy, Abdel Hamid Ghazaly, Hegazy Al-Tamimy

Department of Rheumatology and Rehabilitation, Faculty of Medicine, Al-Azhar University, Egypt

Corresponding author: Adel Azzam; e-mail: adelazzam25@yahoo.com

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Abstract

Aim of the study: This study seeks to evaluate the effectiveness of ultrasound measurements of the ulnar nerve cross-sectional area in comparison to electrodiagnostic tests for identifying ulnar nerve entrapment at the elbow in rheumatoid arthritis. Patients and methods: This study was designed as a cross-sectional observational analysis involving 90 individuals, divided into three groups: Group A consisted of 30 individuals diagnosed with rheumatoid arthritis without clinical signs of ulnar neuropathy at the elbow; Group B included 30 individuals with rheumatoid arthritis exhibiting clinical indicators of ulnar neuropathy at the elbow; and Group C comprised 30 healthy controls. Each participant underwent a thorough medical history assessment, along with both clinical and neurological evaluations. Additionally, ultrasound and electrophysiological assessments of the ulnar nerve were performed. Results: There was no significant demographic difference between the groups, except for age, which was notably lower in Group A compared to Group B. Additionally, abnormalities in nerve conduction studies and cross-sectional area were found to be significantly greater in Group B (p <0.0001). The cross-sectional area demonstrated diagnostic accuracy rates of 52.22%, 62.22%, and 78.89% for identifying ulnar neuropathy at Guyon's canal, the medial epicondyle, and based on the elbow-to-wrist ratio, respectively. Conclusion: Ultrasonography exhibits high diagnostic accuracy, especially with the cross-sectional area at the medial epicondyle and the elbow-to-wrist cross-sectional area ratio serving as important indicators for ulnar nerve entrapment in patients with rheumatoid arthritis.

Introduction

Peripheral neuropathy ranks among the most common extra-articular manifestations associated with rheumatoid arthritis (RA)[1]. In its initial phases, peripheral neuropathy may be asymptomatic or may present with a variety of signs such as pain, tingling sensations, and muscle weakness. These symptoms can sometimes overlap with those of arthritis[2].

Patients with RA may experience different forms of peripheral neuropathy, including entrapment neuropathy, sensory-motor neuropathy, and mononeuritis multiplex[3]. The underlying factors contributing to neuropathy in RA patients include nerve entrapment, adverse effects of medications, vasculitis, and, though very infrequently, amyloidosis[4].

Electrodiagnostic studies are useful tools for identifying the location of nerve entrapment, assessing the severity and type of nerve injury (whether axonal or demyelinating), and predicting disease outcomes. These tests may also be necessary to exclude other causes of muscle atrophy, such as radiculopathy or thoracic outlet syndrome. However, due to their limited availability, high cost, and the discomfort associated with these electrophysiological tests, ultrasound may serve as a valuable alternative or complementary approach for confirming nerve entrapment and diagnosing unusual innervations[5].

This study aims to evaluate and compare the effectiveness of neuromuscular ultrasound versus electrophysiological assessments in diagnosing ulnar nerve entrapment among patients with RA.

Patients and methods

This observational cross-sectional study involved 90 participants who were randomly selected from the Rheumatology and Rehabilitation Departments at our University Hospitals, between May 2023 and October 2024. The participants were categorized into three distinct groups:

- Group A (RA only) consisted of thirty individuals diagnosed with RA who showed no signs of ulnar neuropathy based on clinical neurological assessment.

- Group B (RA with ulnar neuropathy at the elbow (UNE)) included thirty RA patients exhibiting clinical indicators of ulnar nerve entrapment at the elbow, with the diagnosis established according to the criteria of Beekman *et al.*[6].
- Group C (Control group) comprised thirty healthy volunteers of similar age, serving as the control group.

Individuals with a history of elbow injuries, fractures, surgeries, or congenital or post-traumatic elbow deformities were excluded from the study. Additionally, those with known neurological disorders associated with peripheral neuropathy, such as Guillain-Barré syndrome, as well as other comorbid conditions (excluding RA) like diabetes mellitus, hypothyroidism, various connective tissue diseases, upper limb swelling, and pregnancy, were also not included.

Methods

Ultrasound assessments were performed using an 8–13 MHz linear array transducer (APLIO 400 Model, Toshiba US machine, California, USA) to evaluate all patients. Each participant underwent a comprehensive medical history review, along with clinical and neurological evaluations. The Disease Activity Score (DAS28) was employed to measure RA activity across 28 joints[7].

For the ultrasonographic assessment of the ulnar nerve, participants were positioned supine, with the arm extended, the shoulder slightly abducted, and the wrist in a neutral position to facilitate nerve tracing. The scanning of the ulnar nerve began distally at Guyon's canal, with the probe placed transversely at the medial aspect of the wrist (Fig. 1).

The ulnar nerve appeared as a rounded or oval, honeycomb-like formation, positioned just medial to the ulnar artery, adjacent to the pisiform bone, and beneath the tendon of the flexor carpi ulnaris. The nerve was then traced along the ulnar groove, where it exhibited a honeycomb structure, typically appearing as a hypoechoic monofascicular entity. At this point, the ultrasound probe was oriented transversely along an imaginary line connecting the olecranon process of the ulna and the medial epicondyle of the humerus.

To assess the cross-sectional area (CSA) of the ulnar nerve, electronic calipers were applied around the nerve's perimeter, just within the hyperechoic boundary of the nerve sheath. The free-hand outlining technique was used instead of the ellipse tool, and the outlining was done without zoom magnification. CSA was measured at two locations: within Guyon's canal, using the pisiform bone as a bony landmark, and at the tip of the medial epicondyle (Fig. 2).

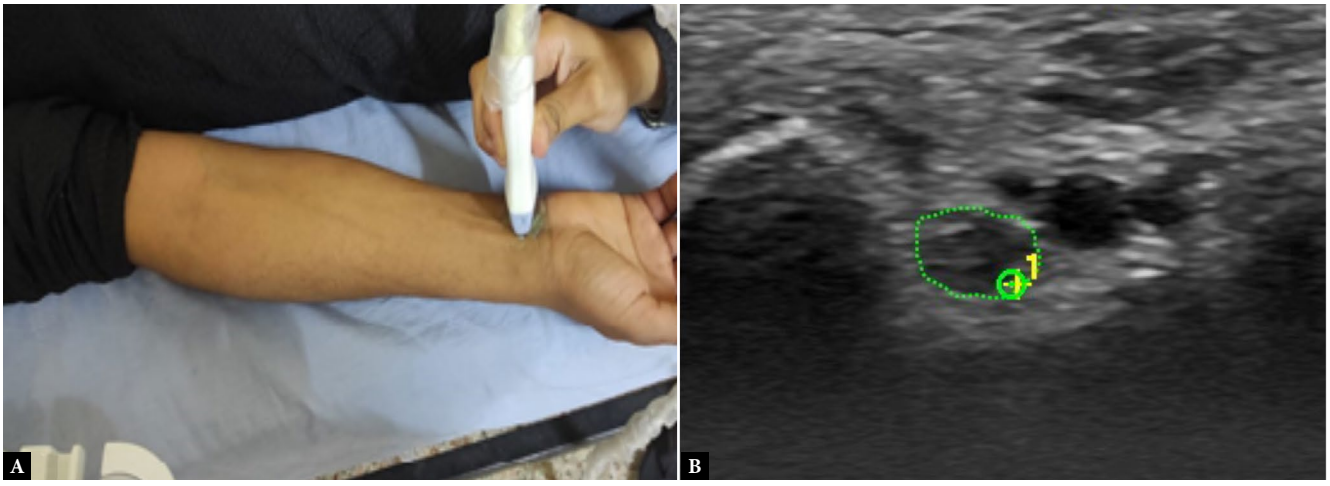

Fig. 1. *Ulnar nerve at Guyon's canal.* **A.** *Probe positioned along the medial aspect of the wrist.* **B.** *Corresponding short-axis ultrasound image shows the ulnar nerve (circled) between the ulnar artery and the pisiform bone*

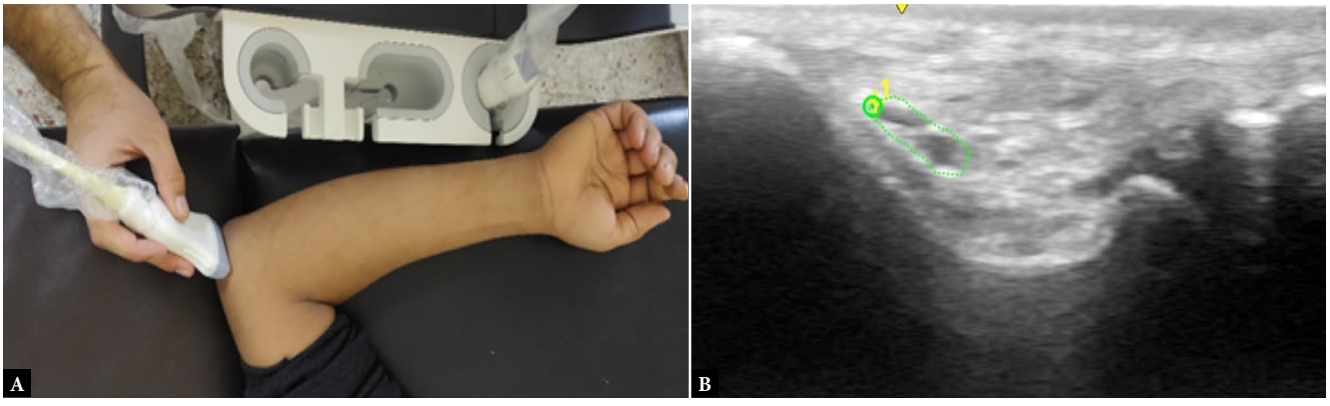

Fig. 2. *Ulnar nerve at the medial epicondyle.* **A.** *Position of the patient during the examination of the ulnar nerve at the medial epicondyle.* **B.** *Short-axis ultrasound image at the medial epicondyle shows the ulnar nerve (circled) at the cubital tunnel*

All examinations were conducted and analyzed by a qualified rheumatologist (AIA), a member of the European League Against Rheumatism. Subsequently, another examiner, blinded to the patients' clinical and electrophysiological data, reviewed the images offline. In cases where discrepancies arose in the findings, a third examiner was consulted to make the final determination.

Electrodiagnostic assessments were conducted utilizing the Neuropack MEP-9400A/K EMG/EP measurement system (Nihon Kohden, Japan). The patient was positioned supine, with the elbow flexed at 135 degrees, the shoulder slightly externally rotated in a right-angle abduction, and the wrist in a neutral position. A standard motor nerve conduction study of the ulnar nerve was performed, recording from the abductor digiti minimi. The active electrode was positioned over the muscle belly, while the reference electrode was placed 3 cm distal to the active electrode, over the fifth metacarpophalangeal joint.

A diagnosis of ulnar nerve neuropathy at the elbow was made in accordance with the American Academy of Neurology's 1991 summary statement, based on one of the following criteria:

1. A reduction in conduction velocity exceeding 10 m/s;
2. Evidence of conduction block at the elbow indicated by a decrease in amplitude of 20% or more;
3. A conduction velocity of less than 50 m/s across the elbow segment.

The local medical ethics committee granted approval for this prospective research, and all participants provided informed consent prior to the commencement of the study.

Statistical analysis

Data were checked, entered, and analyzed using SPSS version 23.0 for data processing. The statistical techniques applied in this study included expressing qualitative variables as counts and percentages, while quantitative variables were represented as mean ± standard deviation (SD). For comparative analysis, ANOVA, Mann-Whitney U, and Chi-square tests were utilized. A *p*-value of less than 0.05 was considered indicative of statistically significant findings.

Results

In the current study, an analysis of age revealed a notable difference between Group A and Group B, with average ages of 34.27 ± 11.04 years and 41.77 ± 11.67 years, respectively ($p1 = 0.047$). However, no significant age differences were found between Group B and Group C ($p3 = 0.703$) or between Group A and Group C ($p2 = 0.055$). The distribution of sex was consistent across the groups, with females making up 70% of Group A, 73.33% of Group B, and 63.33% of Group C ($p = 0.703$).

In terms of residence type, there were no significant differences; urban residents accounted for 70% in Group A, 60% in Group B, and 63.33% in Group C ($p = 0.719$). When examining physical characteristics, Group B exhibited a significantly greater weight (83.63 ± 13.5 kg) compared to Group A (77.27 ± 13.67 kg, $p1 = 0.155$) and Group C (77.57 ± 11.72 kg, $p2 = 0.995$). Height ($p = 0.593$) and BMI ($p = 0.216$) did not show significant differences among the groups (Tab. 1).

Notable variations were found in the levels of rheumatoid factor (RF) and C-reactive protein (CRP) among the different groups. A markedly larger percentage of individuals in Group B (93.33%) tested positive for RF when compared to Group A (80%) and Group C (13.33%) ($p < 0.0001$). Furthermore, Group B exhibited significantly elevated CRP levels (26.33 ± 18.57) compared to Group A (18.73 ± 18.61) ($p = 0.047$), indicating a heightened inflammatory response in patients with RA accompanied by UNE. Additionally, the Disease Activity Score (DAS28) was significantly greater in Group B (3.77 ± 1.05) than in Group A (3.1 ± 1.21) ($p = 0.0185$), pointing to increased disease activity among RA patients with UNE (Tab. 2).

Tab. 1. Comparison between study groups regarding demographic data

	RA only (group A) ($n = 30$)	RA with UNE (group B) ($n = 30$)	Controls (Group C) ($n = 30$)	*p* value
Age (years)	34.27 ± 11.04	41.77 ± 11.67	39.27 ± 12.87	0.055
	p1 = 0.047*, *p2* = 0.250, *p3* = 0.703			
Female	21 (70%)	22 (73.33%)	19 (63.33%)	0.703
Male	9 (30%)	8 (26.67%)	11 (36.67%)	
Urban	21 (70%)	18 (60%)	19 (63.33%)	0.719
Rural	9 (30%)	12 (40%)	11 (36.67%)	
Weight (kg)	77.27 ± 13.67	83.63 ± 13.5	77.57 ± 11.72	0.115
	p1 = 0.155, *p2* = 0.995, *p3* = 0.183			
Height (cm)	167.53 ± 7.37	166.7 ± 6.66	168.63 ± 7.57	0.593
	p1 = 0.898, *p2* = 0.830, *p3* = 0.565			
BMI (kg/m²)	27.75 ± 4.94	29.64 ± 5.13	27.51 ± 5.03	0.216
	p1 = 0.330, *p2* = 0.981, *p3* = 0.245			

p1 – Group A & B; *p2* – Group A & C; *p3* – Group B & C; BMI – body mass index
* Indicates a *p* value <0.05, denoting statistically significant results

Tab. 2. Comparison of clinical and laboratory data between study groups

	Group A (*n* = 30)	Group B (*n* = 30)	Group C (*n* = 30)	*p* value
RF	24 (80%)	28 (93.33%)	4 (13.33%)	<0.0001*
Anti CCP ab	25 (83.33%)	28 (93.33%)	-	0.234
RA duration (years)	9.67 ± 6.79	11.6 ± 6.92	-	0.253
ESR	49 ± 29.63	57.87 ± 17.44	-	0.126
CRP	18.73 ± 18.61	26.33 ± 18.57	-	0.047
DAS 28	3.1 ± 1.21	3.77 ± 1.05	-	0.018

RF – rheumatoid factor; CRP – C-reactive protein; DAS28 – disease activity score
* Indicates a *p* value < 0.05, denoting statistically significant results

The various categories of medications, including glucocorticoids, methotrexate, biologics (such as Adalimumab and Etanercept), and Janus kinase inhibitors like Baricitinib, did not reveal any notable differences among the groups (Tab. 3).

Notable disparities were found in various neurophysiological metrics across the groups. Group B exhibited a considerably larger percentage of abnormal NCS results (76.67%) compared to Group A at 10% and Group C (6.67%) (p <0.0001). Additionally, Group B demonstrated significantly extended motor latency at the wrist, measuring 4.12 ± 1.71 ms, when compared to Group A (2.66 ± 0.45 ms) and Group C (2.79 ± 0.53 ms) (p <0.0001). The above elbow motor amplitude was also significantly reduced in Group B (5.29 ± 1.76 mv) than in Group A (6.67 ± 1.66 mv) (p = 0.005). Furthermore, Group A had a notably higher motor conduction velocity from the wrist to below the elbow (61.17 ± 8.35 m/s) compared to Group B (53.37 ± 10.16 m/s) ($p1$ = 0.006), and from below to above the elbow (60.7 ± 7.64 m/s) versus Group B (51.9 ± 9.42 m/s) ($p1$ = 0.001). Other measured parameters, including motor amplitude at the wrist and below the elbow, did not reveal significant differences across the groups (Tab. 4).

Table 5 indicates that the mean CSA at Guyon's canal did not exhibit significant differences among the groups (p = 0.083). Conversely, the CSA at the medial epicondyle was markedly greater in Group B (12.49 ± 2.1) compared to Group A (7.75 ± 1.46, $p1$ <0.0001) and the control group (8.2 ± 1.57, $p3$ <0.0001). No significant difference was noted between Group A and the control group ($p2$ = 0.581). The overall p-value was highly significant (p <0.0001). Furthermore, the CSA ration from the medial epicondyle to Guyon's canal was significantly elevated in Group B (1.99 ± 0.31) in comparison to Group A (1.31 ± 0.2, $p1$ <0.0001) and the control group (1.26 ± 0.21, $p3$ < 0.0001). No significant difference was observed between Group A and the control group ($p2$ = 0.751). The overall p-value remained highly significant (p <0.0001).

In Tab. 6, the cutoff value for a CSA greater than 6.05 mm² at Guyon's canal exhibited limited diagnostic value, reflected by an area under the curve (AUC) of 0.5631. The sensitivity was 60.71%, while the specificity was 48.39%. The positive predictive value (PPV) stood at 34.69%, while the negative predictive value (NPV) was 73.17%, resulting in an overall accuracy of 52.22% (p = 0.339).

Conversely, a CSA threshold exceeding 8 mm² at the medial epicondyle revealed robust predictive capabilities, achieving an AUC of 0.8453, sensitivity of 85.71%, and specificity of 51.61%. The PPV was 44.44%, while the NPV was 88.89%, yielding an accuracy rate of 62.22% (p <0.0001). Additionally, the elbow-to-wrist CSA ratio greater than 1.495 demonstrated remarkable diagnostic effectiveness, with an AUC of 0.8762, sensitivity of 82.14%, specificity of 77.42%, PPV of 62.16%, NPV of 90.57%, and an accuracy of 78.89% (p <0.0001) (Fig. 3).

Discussion

Ulnar neuropathy ranks as the second most prevalent type of compression neuropathy occurring at the elbow, following carpal tun-

Tab. 3. Comparison of medications used in both study groups

	Group A (*n* = 30)	Group B (*n* = 30)	*p* value
GCs 5 mg	20 (66.67%)	24 (80%)	0.250
GCs 10 mg	7 (23.33%)	2 (6.67%)	0.072
MTX 15 mg	17 (56.67%)	10 (33.33%)	0.071
MTX 25 mg	5 (16.67%)	8 (26.67%)	0.355
Biology			
Adalimumab 40 mg	5 (16.67%)	4 (13.33%)	0.723
Etanercept 50 mg	4 (13.33%)	4 (13.33%)	0.99
JAKi			
Baricitinib 4 mg	7 (23.33%)	2 (6.67%)	0.072

GCs – glucocorticoids; MTX – methotrexate; JAKi – Janus kinase inhibitors

Tab. 4. Comparison between different study groups regarding NCS

	Group A (*n* = 30)	Group B (*n* = 30)	Group C (*n* = 30)	*p* value
NCS abnormality	3 (10%)	23 (76.67%)	2 (6.67%)	<0.0001*
Distal motor latency (mS)				
Wrist	2.66 ± 0.45	4.12 ± 1.71	2.79 ± 0.53	<0.0001*
p1 <0.0001*, *p2* = 0.887, *p3* <0.0001*				
Motor amplitude (mv)				
Wrist	7.07 ± 1.6	6.99 ± 3.16	6.6 ± 1.6	0.053
p1 = 0.990, *p2* = 0.707, *p3* = 0.785				
Below elbow	6.65 ± 1.58	6.12 ± 1.91	6.42 ± 1.47	0.063
p1 = 0.447, *p2* = 0.86, *p3* = 0.767				
Above elbow	6.67 ± 1.66	5.29 ± 1.76	6 ± 1.29	0.005*
p1 = 0.003*, *p2* = 0.244, *p3* = 0.206				
Conduction velocity (m/s)				
Wrist to below elbow	61.17 ± 8.35	53.37 ± 10.16	65.7 ± 9.71	<0.0001*
	p1 = 0.006*, *p2* = 0.166, *p3* <0.0001*			
Below to above elbow	60.7 ± 7.64	51.9 ± 9.42	63.37 ± 9.73	<0.0001*
	p1 = 0.001*, *p2* = 0.495, *p3* <0.0001*			

NCS – nerve conduction studies; *p1* – Group A & B; *p2* – Group A & C; *p3* – Group B & C
* Indicates a *p* value < 0.05, denoting statistically significant results

Tab. 5. Comparison between groups regarding sonographic CSA measurements

	Group A (*n* = 30)	Group B (*n* = 30)	Group C (*n* = 30)	*p* value
CSA (mm²)				
At Guyon's canal	6.01 ± 0.97	6.32 ± 0.72	6.54 ± 0.95	0.083
p1 = 0.393, *p2* = 0.068, *p3* = 0.616				
At medial epicondyle	7.75 ± 1.46	12.49 ± 2.1	8.2 ± 1.57	<0.0001*
p1 <0.0001*, *p2* = 0.581, *p3* <0.0001*				
Elbow-to-wrist ratio	1.31 ± 0.2	1.99 ± 0.31	1.26 ± 0.21	<0.0001*
	p1 <0.0001*, *p2* = 0.751, *p3* <0.0001*			

CSA – cross-sectional area; *p1* – Group A & B; *p2* – Group A & C; *p3* – Group B & C
* Indicates a *p* value < 0.05, denoting statistically significant results

Tab. 6. Receiver-operating characteristic (ROC) curve for predicting UNE among all cases

	Cutoff	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	Kappa Agreement	*p* value
CSA at Guyon's canal (mm²)	>6.05	0.563	60.71	48.39	34.69	73.17	52.22	0.075	0.339
CSA at ulnar groove (mm²)	>8	0.845	85.71	51.61	44.44	88.89	62.22	0.297	<0.0001*
CSA Elbow-to-wrist ratio	>1.495	0.876	82.14	77.42	62.16	90.57	78.89	0.547	<0.0001*

CSA – cross-sectional area; NPV – negative predictive value; PPV – positive predictive value
* Indicates a *p* value < 0.05, denoting statistically significant results

nel syndrome. This condition can result in significant functional limitations, particularly affecting fine motor skills[8,9]. Given that neurophysiological testing is often costly, less accessible, and can be uncomfortable for patients, ultrasound may serve as a useful alternative or complementary method for confirming entrapment and identifying abnormal innervations[10]. This study aims to evaluate the effectiveness of neuromuscular ultrasound in comparison to electrophysiological assessments in diagnosing ulnar neuropathy in patients with RA.

In this study, the average ages recorded were 34.27 ± 11.04, 41.77 ± 11.67, and 39.27 ± 12.87 years, revealing a noteworthy difference be-

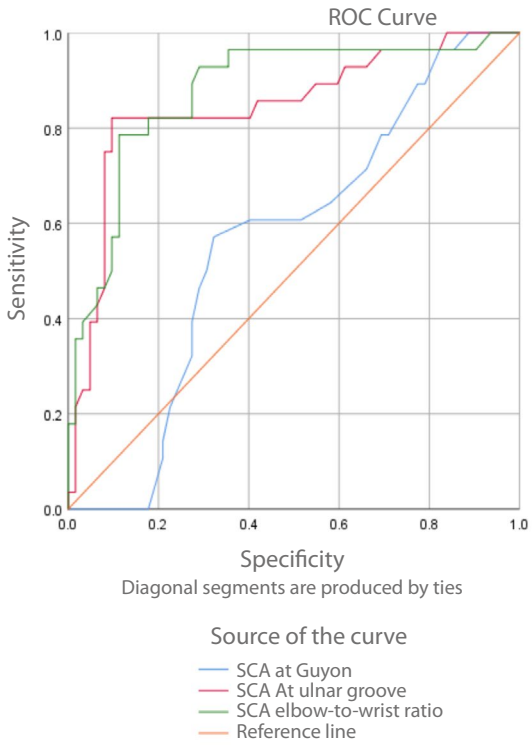

Fig. 3. *Roc curve analysis of UNE occurrence among all cases*

tween RA patients with and without ulnar neuropathy ($p1 = 0.047$). However, other demographic variables did not exhibit significant differences.

The observed link between advanced age and the occurrence of ulnar neuropathy in RA patients may be attributed to the prolonged duration of the disease in older individuals, which can result in cumulative joint deterioration, persistent inflammation, and reduced blood flow. These factors may elevate the likelihood of nerve compression or ischemic damage[11,12].

In our analysis, the BMI values were 27.75 ± 4.94, 29.64 ± 5.13, and 27.51 ± 5.03 kg/m², with no significant differences ($p = 0.216$). This contrasts with the findings of Joshua and Misri[13], who reported that a higher BMI is associated with an increased risk of ulnar neuropathy. They suggested that factors such as additional mechanical stress on the ulnar nerve, particularly at the elbow, postural habits that worsen nerve compression, and systemic inflammation linked to obesity may contribute to this association.

While the erythrocyte sedimentation rate levels in our RA patients with or without ulnar neuropathy did not show significant differences ($p = 0.126$), the CRP levels were notably elevated in those with UNE compared to the RA-only group ($p = 0.047$). Additionally, disease activity in RA, as measured by DAS28, was significantly greater in RA patients with UNE ($p = 0.018$).

In line with our findings, Ito and Kobayashi[14] conducted an analysis of the components of neuropathic pain in individuals with RA. Their study found that patients exhibiting likely or possible neuropathic pain had notably elevated levels of CRP compared to those with unlikely neuropathic pain.

The elevated levels of CRP and DAS28 scores observed in RA patients with ulnar neuropathy (Group B) suggest that heightened systemic and joint-specific inflammation may exacerbate damage to blood vessels and soft tissues, potentially leading to nerve compression or ischemic neuropathy[15]. This condition can impair nerve function and raise the risk of neuropathy in more active disease phases[16,17].

Our study revealed that RA patients with UNE displayed significantly prolonged motor latency, diminished amplitude above the elbow, and reduced conduction velocity in the elbow segment when compared to the other groups. These changes are likely due to axonal degeneration or demyelination stemming from ongoing synovial inflammation and vascular alterations associated with RA[18]. Additionally, the decreased motor amplitude above the elbow in the ulnar neuropathy group points to more pronounced nerve injury in this area, highlighting the specific impact of RA-related neuropathy on the function of the ulnar nerve[19,20].

In this study, we evaluated the CSA of the ulnar nerve through ultrasound at two distinct points: Guyon's canal at the level of the pisiform bone of the wrist and the tip of the medial epicondyle. To enhance the consistency of our measurements, we also introduced a ratio for standardization.

Our findings showed no notable differences in the CSA of the ulnar nerve at Guyon's canal across the different groups ($p = 0.083$), which aligns with the results reported by Bastawy *et al.*[21]. Furthermore, Atan Uzun *et al.*[22] found no statistically significant differences in the ulnar CSAs at the pisiform bone level when comparing patients with RA to control individuals.

Conversely, at the medial epicondyle, RA patients with clinical signs of ulnar neuropathy exhibited a significantly greater CSA. Additionally, the elbow-to-wrist CSA ratio was markedly higher in RA patients with ulnar neuropathy compared to the other groups ($p < 0.0001$). These results align with previous studies indicating that ulnar nerve enlargement at the elbow, as observed through ultrasound, correlates with increased clinical severity[23–25].

Multiple studies have shown that individuals with ulnar nerve entrapment at the elbow exhibit a greater mean CSA at the elbow region, unlike at the level of the pisiform bone[21,26,27]. This suggests that the nerve swelling associated with ulnar nerve entrapment is localized specifically to the elbow area.

Furthermore, Rayegani and Raeissadat[5] evaluated the diagnostic effectiveness of ultrasound by measuring the CSA of the ulnar nerve at three distinct points: the medial epicondyle, 2 cm proximal, and 2 cm distal. Their study revealed that the CSA at the medial epicondyle and 2 cm below it was considerably greater in the patient group compared to the control group.

In the current study, the average CSA of the ulnar nerve among healthy participants was found to be 8.2 ± 1.57 mm² at the epicondyle level. This measurement aligns with previous research, which reported values ranging from 5.8 to 9.7 mm²[21,26,28–30].

In our study, we employed ROC curve analysis to identify key predictors for ulnar neuropathy within our cohort. We established a cutoff value of >1.495 for the elbow-to-wrist CSA ratio, which

resulted in an AUC of 0.876 and an accuracy rate of 78.89% (p <0.0001). Our findings align with those of Bastawy *et al.*[21], who assessed CSA at the medial epicondyle and at an unaffected location, calculating the ratio between these two measurements. Their study reported a cutoff value of 1.13. Despite differences in methodology and patient population, the CSA area ratios observed in both studies are notably comparable.

Similarly, a CSA greater than 8 mm² at the medial epicondyle demonstrated an AUC of 0.845 and an accuracy of 62.22% (p <0.0001). In contrast, the CSA at Guyon's canal showed limited diagnostic utility (AUC = 0.563, accuracy = 52.22%, p = 0.3399). A prior investigation indicated that a CSA measurement of at least 11 mm² at the medial epicondyle, rather than at the level of the pisiform bone, was a reliable predictor of ulnar neuropathy and the need for electrodiagnostic evaluation[26]. Furthermore, Bastawy *et al.*[21] found that a CSA exceeding 9 mm² at the medial epicondyle is highly effective for diagnosing ulnar neuropathy at the elbow.

In contrast, Rayegani and Raeissadat[5] reported that while the CSA of the ulnar nerve was greater in patients compared to controls at both the medial epicondyle and 2 cm distal to it, the most reliable single measurement was found to be 2 cm distal to the medial epicondyle. This measurement, with a threshold of 9 mm², demonstrated significant diagnostic utility, achieving a specificity of 80% and a sensitivity of 84%[5].

The variations in CSA cutoff levels observed in our study, compared to others, can be attributed to multiple factors. These include differences in the quality and resolution of ultrasound images, equipment settings, evaluation techniques, patient characteristics, and the unavailability of a high-frequency hockey stick probe, which could enhance the visualization of nerve fascicles.

A notable limitation of this study is the absence of evaluation of specific anatomical structures, such as the anconeus epitrochlearis, low-lying triceps, snapping triceps, and the Osborn ligament, each of which is relevant to ulnar nerve entrapment at the elbow. Another constraint is the limited sample size and the lack of multivariate analysis to control for potential confounding factors, such as age, gender, and socioeconomic status. By addressing these issues, subsequent larger multi-center studies may validate these findings and enhance the generalizability of the results across diverse populations with RA.

Conclusion

Electrodiagnostic tests and neuromuscular ultrasound are both valuable tools for identifying ulnar neuropathy in individuals with RA. Notably, ultrasound demonstrates high diagnostic precision, particularly with CSA measurements at the medial epicondyle and the elbow-to-wrist CSA ratio serving as significant indicators for ulnar neuropathy in this patient group.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organizations which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

Author contributions

Original concept of study: AA, HT. Writing of manuscript: AA. Analysis and interpretation of data: MM. Collection, recording and/or compilation of data: AA. Critical review of manuscript: AA, AHG.

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