

# Prevalence, Pattern and Clinical Correlates of Peripheral Neuropathy in Rheumatoid Arthritis: A Cross-Sectional Nerve Conduction Study

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## ABSTRACT

**Background:** Rheumatoid arthritis (RA) is a chronic immune-inflammatory systemic disease that, besides synovial joints, can affect the peripheral nervous system. Peripheral neuropathy in RA is frequently under-recognised clinically because neuropathic symptoms are often attributed to joint disease, and neurological examination can be difficult in patients with severe articular involvement.

**Objective:** To study the prevalence and pattern of peripheral neuropathy in patients with RA using nerve conduction studies (NCS), and to examine its association with disease parameters and other clinical and laboratory variables.

**Materials and Methods:** This cross-sectional study was conducted over eight months in the Department of Medicine and Rheumatology, Government Kilpauk Medical College, Chennai, among 60 consecutive patients diagnosed with RA by the 1987 revised ACR classification criteria. All patients underwent clinical evaluation, relevant laboratory investigations and bilateral NCS of the median, ulnar, common peroneal, posterior tibial and sural nerves. Polyneuropathy was diagnosed when at least three abnormal NCS parameters were present. Associations were assessed using Fisher's exact test and the unpaired t-test, with multivariate logistic regression used to examine independent associations; a two-tailed p value <0.05 was considered significant.

**Results:** Peripheral neuropathy was present in 26 of 60 patients (43.3%). The commonest pattern was sensorimotor neuropathy (42.3%), followed by sensory neuropathy (23.1%), motor neuropathy (19.2%), carpal tunnel syndrome (7.7%) and mononeuritis multiplex (7.7%). Patients with neuropathy were significantly older (mean 45.85 vs 36.65 years,  $p < 0.0001$ ), had higher ESR (mean 39.77 vs 28.97 mm/hr,  $p = 0.0007$ ), longer RA duration (mean 8.23 vs 5.06 years,  $p = 0.0004$ ), and higher rheumatoid factor (96.2% vs 67.6%,  $p = 0.0064$ ) and anti-CCP antibody positivity (30.8% vs 8.8%,  $p = 0.0288$ ) than those without neuropathy. On multivariate logistic regression, age >40 years, ESR >40 mm/hr, anti-CCP

antibody positivity and RA duration >10 years remained independently associated with peripheral neuropathy. Gender, haemoglobin, complete blood counts, CRP, steroid use and neuropathic symptom reporting showed no significant association.

**Conclusion:** Peripheral neuropathy, often subclinical, is common in patients with RA and is associated with older age, higher ESR, longer disease duration and anti-CCP antibody positivity. Nerve conduction studies may be a useful tool for detecting subclinical neuropathy in RA patients with these risk factors, particularly as subjective neuropathic symptoms correlate poorly with electrophysiological findings in this population.

**Keywords:** Rheumatoid arthritis; Peripheral neuropathy; Nerve conduction study; Anti-cyclic citrullinated peptide antibody; Cross-sectional study.

## INTRODUCTION

Rheumatoid arthritis (RA) is a chronic immune-inflammatory systemic disease that primarily affects the synovial joints but is well recognised to have extra-articular manifestations [1]. Its global prevalence is estimated at approximately 0.8% (range 0.3–2.1%), with Indian data suggesting a somewhat lower prevalence of 0.65–0.75% [2].

Although RA is primarily a joint disease, extra-articular manifestations can involve any organ system and may occasionally precede the onset of arthritis itself. The most common neurological manifestation is entrapment neuropathy secondary to proliferative synovitis [3], and up to one-third of patients may develop neurological problems during their lifetime [3]. Peripheral nerve involvement in RA spans a spectrum from compressive mononeuropathy, the commonest pattern, to distal sensory and combined sensorimotor polyneuropathy, a spectrum first systematically described in early electrophysiological case series [4,7].

The underlying pathophysiology of rheumatoid neuropathy is not fully understood, but ischaemia secondary to vasculitis, with associated axonal loss, and humoral mechanisms such as immune-complex deposition and complement fixation in small-vessel walls are thought to be important contributing factors [5,6].

Peripheral neuropathy in RA is frequently under-recognised in routine clinical practice because patients often attribute neurological symptoms to their joint disease, and

neurological examination can be difficult in the presence of severe articular involvement [5]. This study was therefore undertaken to evaluate the prevalence and pattern of peripheral neuropathy in patients with RA using nerve conduction studies, and to correlate its presence with disease parameters and other clinical and laboratory variables.

## Aims and Objectives

- To study the prevalence of peripheral neuropathy in rheumatoid arthritis.
- To determine the pattern of neuropathy in rheumatoid arthritis using nerve conduction studies.
- To correlate neuropathy with disease parameters and other extra-articular involvement

## MATERIALS & METHODS

### Study Design and Setting

This was a cross-sectional, observational, single-centre study conducted in the Department of Medicine and Rheumatology, Government Kilpauk Medical College and Hospital, Chennai, over a period of eight months from the date of ethical clearance.

### Study Population and Sampling

Sixty consecutive patients with a diagnosis of RA, with or without clinical evidence of neuropathy, attending the outpatient department or admitted to the hospital during the study period and satisfying the inclusion and exclusion criteria, were enrolled using a convenience (consecutive-

sampling) approach. A formal a priori sample-size calculation was not performed; the sample size was determined pragmatically by the number of eligible patients identified within the study period, and this is acknowledged as a limitation in the Discussion.

**Inclusion criteria:** Patients already diagnosed with RA as per the 1987 Revised American College of Rheumatology (ACR, formerly American Rheumatism Association) Classification Criteria [8], correlated with laboratory data.

**Exclusion criteria:** Patients with diabetes mellitus, renal failure, chronic alcoholism, retroviral disease (HIV), liver disease, thyroid disease, pregnancy, or current use of medications known to be associated with peripheral neuropathy, were excluded, as these are recognised confounding causes of peripheral neuropathy independent of RA.

### ***Clinical and Laboratory Evaluation***

All patients underwent thorough clinical and laboratory evaluation, including haemoglobin, total and differential leucocyte count, erythrocyte sedimentation

rate (ESR), platelet count, C-reactive protein (CRP), rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibody status, in addition to recording of disease duration and current medication (steroid and DMARD use).

### ***Nerve Conduction Studies***

All patients underwent nerve conduction studies (NCS) performed using a Medelec-Synergy EMG instrument, with skin temperature maintained between 31–32°C (nerve conduction velocity is known to increase by approximately 2.4 m/s per degree Celsius between 29–38°C skin temperature) [9-11]. Motor NCS, including F-waves, were performed bilaterally for the median, ulnar, common peroneal and posterior tibial nerves using the belly-tendon method. Sensory NCS were performed bilaterally for the median, ulnar, superficial peroneal and sural nerves, studied antidromically. H-reflex latency was measured bilaterally. All amplitudes were measured base-to-peak, and findings were compared against the institutional laboratory's reference values (Table 1).

**Table 1: Reference values used for nerve conduction study parameters**

| Nerve                          | Latency (ms) | Amplitude | Conduction velocity (m/s) |
|--------------------------------|--------------|-----------|---------------------------|
| Motor – Median                 | <4.2         | >5 mV     | >50                       |
| Motor – Ulnar                  | <6.2         | >2 mV     | >40                       |
| Motor – Deep Peroneal          | <5           | >5 mV     | >40                       |
| Motor – Posterior Tibial       | <5           | >5 mV     | >40                       |
| F-wave – Median                | <30          | –         | –                         |
| F-wave – Ulnar                 | <30          | –         | –                         |
| F-wave – Deep Peroneal         | <50          | –         | –                         |
| F-wave – Posterior Tibial      | <50          | –         | –                         |
| Sensory – Median               | <3.6         | >20 µV    | –                         |
| Sensory – Ulnar                | <3.6         | >10 µV    | –                         |
| Sensory – Superficial Peroneal | <4.2         | >5 µV     | –                         |
| Sensory – Sural                | <3.9         | >10 µV    | –                         |
| H-reflex                       | <32          | –         | –                         |

Polyneuropathy was diagnosed when at least three abnormal NCS parameters were present. Carpal tunnel syndrome (CTS) was suspected when median compound muscle action potential (CMAP) latency exceeded 4.2 ms or median sensory nerve action potential (SNAP) latency exceeded 3.6 ms, and was confirmed using the mid-palm

technique (median nerve stimulated at the palm, 14 cm from the wrist); CTS was diagnosed when the palm-to-wrist latency ratio was below 50%.

### ***Ethical Considerations***

The study was conducted after obtaining approval from the Institutional Ethics

Committee, Government Kilpauk Medical College, Chennai [Protocol ID. No. 03/2017; Meeting held on 03.03.2017]. Written informed consent was obtained from all participants, and patient anonymity and confidentiality were maintained throughout the study.

### Statistical Analysis

Data were entered and analysed using SPSS version 20. Categorical variables were compared using Fisher's exact test, and continuous variables were compared between patients with and without peripheral neuropathy using the unpaired t-test, with results expressed as mean  $\pm$  standard deviation. Multivariate logistic regression was used to assess the independent association of clinical and laboratory variables with the presence of peripheral neuropathy, adjusting for the other variables listed in Table 11, and results are expressed as adjusted odds ratios (OR) with 95% confidence intervals (CI). A two-tailed p value  $<0.05$  was considered statistically significant.

### RESULT

Sixty patients with RA were included in the study. Peripheral neuropathy (PN) was detected on nerve conduction study in 26 patients (43.3%), while 34 patients (56.7%) had no electrophysiological evidence of neuropathy (Table 2).

**Table 2: Prevalence of peripheral neuropathy (n=60)**

| Status      | Number | Percentage |
|-------------|--------|------------|
| PN positive | 26     | 43.33%     |
| PN negative | 34     | 56.67%     |
| Total       | 60     | 100.00%    |

### Pattern of Neuropathy

Among the 26 patients with PN, sensorimotor neuropathy was the commonest pattern (11/26, 42.3%), followed by sensory neuropathy (6/26, 23.1%), motor neuropathy (5/26, 19.2%), carpal tunnel syndrome (2/26, 7.7%) and mononeuritis multiplex (2/26, 7.7%) (Table 3).

**Table 3: Pattern of peripheral neuropathy on nerve conduction study (n=26)**

| Type of neuropathy      | Number | Percentage |
|-------------------------|--------|------------|
| Sensorimotor neuropathy | 11     | 42.31%     |
| Sensory neuropathy      | 6      | 23.08%     |
| Motor neuropathy        | 5      | 19.23%     |
| Carpal tunnel syndrome  | 2      | 7.69%      |
| Mononeuritis multiplex  | 2      | 7.69%      |
| Total                   | 26     | 100.00%    |

### Clinical and Laboratory Correlates of Peripheral Neuropathy

Patients with PN were significantly older (mean  $45.85 \pm 8.99$  years) than those without PN (mean  $36.65 \pm 6.87$  years; unpaired t-test,  $p<0.0001$ ). The PN-positive group was predominantly in the 41–50-year age band (46.2%), while the PN-negative group was predominantly in the 31–40-year band (58.8%) (Table 4).

**Table 4: Age distribution by peripheral neuropathy status**

| Age group (years) | PN positive (%) | PN negative (%) | Overall (%) |
|-------------------|-----------------|-----------------|-------------|
| $\leq 30$         | 1 (3.85)        | 5 (14.71)       | 6 (10.00)   |
| 31–40             | 6 (23.08)       | 20 (58.82)      | 26 (43.33)  |
| 41–50             | 12 (46.15)      | 8 (23.53)       | 20 (33.33)  |
| 51–60             | 6 (23.08)       | 1 (2.94)        | 7 (11.67)   |
| 61–70             | 1 (3.85)        | 0 (0.00)        | 1 (1.67)    |
| Total             | 26 (100.00)     | 34 (100.00)     | 60 (100.00) |

Gender distribution did not differ significantly between groups: 73.1% of the PN-positive group and 88.2% of the PN-negative group were female (Fisher's exact test,  $p=0.1329$ ). Mean haemoglobin ( $11.68$  vs  $11.59$  g%,  $p=0.8231$ ) and complete blood

count parameters (total count, polymorphs, lymphocytes, monocytes and platelets; all  $p>0.05$ ) also did not differ significantly between groups.

Mean ESR was significantly higher in the PN-positive group ( $39.77 \pm 14.48$  mm/hr)

than in the PN-negative group ( $28.97 \pm 8.63$  mm/hr;  $p=0.0007$ ) (Table 5). CRP positivity did not differ significantly between groups (57.7% vs 55.9%;  $p=0.8891$ ).

**Table 5: Erythrocyte sedimentation rate (ESR) by peripheral neuropathy status**

| ESR distribution | PN positive | PN negative | Overall |
|------------------|-------------|-------------|---------|
| Mean (mm/hr)     | 39.77       | 28.97       | 33.65   |
| SD               | 14.48       | 8.63        | 12.63   |

Unpaired t-test,  $p=0.0007$

Rheumatoid factor (RF) positivity was significantly more frequent in the PN-positive group (25/26, 96.2%) than in the PN-negative group (23/34, 67.6%; Fisher's exact test,  $p=0.0064$ ) (Table 6). Anti-CCP antibody positivity was also significantly more frequent in the PN-positive group (8/26, 30.8%) than in the PN-negative group (3/34, 8.8%;  $p=0.0288$ ) (Table 7).

**Table 6: Rheumatoid factor (RF) status by peripheral neuropathy status**

| RF status | PN positive (%) | PN negative (%) | Overall (%) |
|-----------|-----------------|-----------------|-------------|
| Negative  | 1 (3.85)        | 11 (32.35)      | 12 (20.00)  |
| Positive  | 25 (96.15)      | 23 (67.65)      | 48 (80.00)  |
| Total     | 26 (100.00)     | 34 (100.00)     | 60 (100.00) |

Fisher's exact test,  $p=0.0064$

**Table 7: Anti-CCP antibody status by peripheral neuropathy status**

| Anti-CCP status | PN positive (%) | PN negative (%) | Overall (%) |
|-----------------|-----------------|-----------------|-------------|
| Negative        | 18 (69.23)      | 31 (91.18)      | 49 (81.67)  |
| Positive        | 8 (30.77)       | 3 (8.82)        | 11 (18.33)  |
| Total           | 26 (100.00)     | 34 (100.00)     | 60 (100.00) |

Fisher's exact test,  $p=0.0288$

Mean duration of RA was significantly longer in the PN-positive group ( $8.23 \pm 3.73$  years) than in the PN-negative group ( $5.06 \pm 2.84$  years;  $p=0.0004$ ) (Table 8). Steroid use did not differ significantly between groups (26.9% vs 38.2%;  $p=0.3573$ ), whereas DMARD use was significantly more frequent in the PN-positive group (26/26, 100%) than in the PN-negative group (24/34, 70.6%;  $p=0.0022$ ). Reported neuropathic symptoms did not differ significantly between groups (19.2% vs 5.9%;  $p=0.1198$ ), consistent with poor correlation between subjective symptoms and electrophysiological findings in this population.

**Table 8: Duration of RA by peripheral neuropathy status**

| Duration of RA distribution | PN positive | PN negative | Overall |
|-----------------------------|-------------|-------------|---------|
| Mean (years)                | 8.23        | 5.06        | 6.43    |
| SD                          | 3.73        | 2.84        | 3.60    |

Unpaired t-test,  $p=0.0004$

**Table 9: Summary of clinical and laboratory associations with peripheral neuropathy**

| Variable                              | Test used       | p value       | Significant ( $p<0.05$ ) |
|---------------------------------------|-----------------|---------------|--------------------------|
| Age                                   | Unpaired t-test | $<0.0001$     | Yes                      |
| Gender                                | Fisher's exact  | 0.1329        | No                       |
| Haemoglobin                           | Unpaired t-test | 0.8231        | No                       |
| Complete blood count (all parameters) | Unpaired t-test | $>0.05$ (all) | No                       |
| ESR                                   | Unpaired t-test | 0.0007        | Yes                      |
| CRP                                   | Fisher's exact  | 0.8891        | No                       |
| Rheumatoid factor                     | Fisher's exact  | 0.0064        | Yes                      |
| Anti-CCP antibody                     | Fisher's exact  | 0.0288        | Yes                      |
| Duration of RA                        | Unpaired t-test | 0.0004        | Yes                      |
| Steroid use                           | Fisher's exact  | 0.3573        | No                       |
| DMARD use                             | Fisher's exact  | 0.0022        | Yes                      |
| Neuropathic symptoms                  | Fisher's exact  | 0.1198        | No                       |

### Multivariate Analysis

On multivariate logistic regression adjusting for the variables listed in Table 10, age >40 years (adjusted OR 2.43, 95% CI 1.47–4.01,  $p=0.0112$ ), ESR >40 mm/hr (adjusted OR 2.65, 95% CI 1.67–4.79,  $p=0.0457$ ), anti-CCP antibody positivity (adjusted OR 3.49,  $p=0.0322$ ) and RA duration >10 years

(adjusted OR 2.07,  $p=0.0145$ ) remained significantly and independently associated with peripheral neuropathy. Gender, haemoglobin, CRP status, RF status, steroid intake and DMARD intake did not retain statistical significance on multivariate analysis (Table 10).

**Table 10: Multivariate logistic regression analysis of factors associated with peripheral neuropathy in RA**

| Variable                          | Adjusted OR | 95% CI    | p value |
|-----------------------------------|-------------|-----------|---------|
| Age (>40 years)                   | 2.43        | 1.47–4.01 | 0.0112  |
| Gender (Male)                     | 1.75        | 1.04–2.93 | 0.1141  |
| Haemoglobin (>11 g%)              | 1.02        | 0.85–2.87 | 0.1026  |
| ESR (>40 mm/hr)                   | 2.65        | 1.67–4.79 | 0.0457  |
| CRP positive status               | 1.75        | 1.04–2.93 | 0.4421  |
| RF positive status                | 2.00        | 1.21–3.32 | 0.0966  |
| Anti-CCP antibody positive status | 3.49        | 0.88–5.87 | 0.0322  |
| Duration of RA (>10 years)        | 2.07        | 0.74–4.47 | 0.0145  |
| Steroid intake                    | 2.15        | 0.87–3.39 | 0.6832  |
| DMARD intake                      | 2.94        | 0.91–5.05 | 0.0751  |

### DISCUSSION

The relationship between peripheral neuropathy and demographic, clinical and laboratory findings in RA has not been consistently characterised in the literature, with reported prevalence varying considerably between studies, partly because of differing inclusion criteria and neuropathy-assessment methods (Table 11). In the present study, electrophysiological

evidence of peripheral neuropathy, including entrapment neuropathy, was found in 43.3% of RA patients — a frequency within the range reported in comparable electrophysiological series, and substantially higher than the prevalence of idiopathic polyneuropathy (approximately 8%) reported in a population-based study of an unselected elderly population [12].

**Table 11: Comparison of electrophysiological studies of peripheral neuropathy in RA**

| Study                | No. of patients | SM | S  | M | MM | CTS | Associated factors                          |
|----------------------|-----------------|----|----|---|----|-----|---------------------------------------------|
| Fleming et al. [21]  | 102             | 0  | 15 | 3 | 0  | 53  | Not specified                               |
| Agarwal et al. [19]  | 108             | 25 | 28 | 0 | 7  | 11  | Absence of DTR, presence of vasculitis      |
| Lanzillo et al. [20] | 40              | 26 | 0  | 0 | 0  | 3   | Not specified                               |
| Nadkar et al. [4]    | 31              | 6  | 0  | 4 | 4  | 1   | Not specified                               |
| Bayrak et al. [22]   | 60              | 8  | 2  | 0 | 0  | –   | Duration of RA, NSS, NDS, DAS28             |
| Present study        | 60              | 11 | 6  | 5 | 2  | 2   | Age, ESR, anti-CCP antibody, duration of RA |

SM: sensorimotor neuropathy; S: sensory neuropathy; M: motor neuropathy; MM: mononeuritis multiplex; CTS: carpal tunnel syndrome; DTR: deep tendon reflex; NSS: neuropathy symptom score; NDS: neuropathy disability score; DAS28: 28-joint disease activity score.

In the present study, peripheral neuropathy showed a significant association with older age, higher ESR, longer RA duration and anti-CCP antibody positivity, while no significant association was found with gender, neuropathic symptom reporting, CRP status or type of medication on

multivariate analysis. Patients with peripheral neuropathy were older than those without, consistent with age being a recognised risk factor for polyneuropathy in general populations [13], in addition to any RA-specific contribution.

A notable finding of this study was that anti-CCP antibody positivity was associated with an increased likelihood of peripheral neuropathy. Anti-CCP antibody is an established marker of more severe RA [14] and has been associated with HLA class II susceptibility alleles and more severe disease manifestations [15]. While an association between anti-CCP antibody titre and extra-articular manifestations of RA has been suggested by some authors, data specifically relating anti-CCP antibody status to the development of peripheral neuropathy remain limited [16-18]; this study adds to that limited evidence base.

No significant association was found between neuropathic symptom reporting and electrophysiological evidence of neuropathy. This is consistent with the difficulty of distinguishing neuropathic symptoms from those of active arthritis on history alone, and suggests that subjective symptom screening may be an unreliable method for detecting neuropathy in this population; objective electrophysiological assessment, particularly in older patients and those positive for anti-CCP antibody, may therefore have a useful role.

### **Limitations**

This study has several limitations. First, the sample size (n=60) was determined pragmatically based on patients presenting during a fixed eight-month period at a single centre, without a prior formal sample-size calculation, which may limit statistical precision; this is reflected in the wide confidence intervals around some multivariate estimates, and some adjusted odds ratios and their corresponding confidence intervals and p-values in Table 10 should be interpreted with caution and verified against the original statistical analysis before any secondary use. Second, this was a single-centre, cross-sectional study, which limits generalisability and precludes assessment of the temporal relationship between RA disease activity and neuropathy onset; patients were not evaluated at a uniform time point relative to

symptom onset. Third, this study could not establish the definite mechanism of peripheral nerve involvement in individual patients - that is, whether neuropathy resulted from a direct RA-related process (e.g. vasculitic or immune-mediated nerve injury) or from secondary compressive effects of joint deformity - and this distinction would require further targeted evaluation. Longitudinal studies in larger, multicentric populations are needed to clarify the temporal and causal relationships between RA disease activity, anti-CCP antibody status and the development of peripheral neuropathy.

### **CONCLUSION**

In this cross-sectional study, peripheral neuropathy was common among patients with RA (43.3%) and was frequently subclinical. Gender, haemoglobin level, complete blood count parameters, CRP status, steroid use and reported neuropathic symptoms showed no significant association with peripheral neuropathy. On comparison between patients with and without peripheral neuropathy, the following associations were observed:

- Peripheral neuropathy was more common among patients in the older age group.
- ESR levels were higher among patients with peripheral neuropathy.
- Rheumatoid factor positivity was more frequent among patients with peripheral neuropathy.
- Anti-CCP antibody positivity was more frequent among patients with peripheral neuropathy.
- Duration of RA was longer among patients with peripheral neuropathy.
- DMARD use was more frequent among patients with peripheral neuropathy.

Age greater than 40 years, RA duration greater than 10 years, ESR greater than 40 mm/hr and anti-CCP antibody positivity characterised the patients at highest likelihood of peripheral neuropathy in this cohort. As a single-centre, cross-sectional study with a modest sample size, these

findings should be regarded as hypothesis-generating; they support a potential role for nerve conduction studies in identifying subclinical peripheral neuropathy in RA patients with these risk factors, particularly given the poor correlation between subjective neuropathic symptoms and electrophysiological findings, but require confirmation in larger, prospective, multicentric studies.

### **Declaration by Authors**

**Ethical Approval:** Approved

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