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Electrophysiological abnormalities in patients with psoriasis vulgaris and their association with disease duration and severity: a cross-sectional study

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ABSTRACT

Background. Psoriasis vulgaris is a chronic immune-mediated inflammatory skin disease with potential systemic involvement. Recent research suggests a possible association between psoriasis and peripheral nervous system abnormalities, although this relationship remains incompletely understood.

Objectives. This study aimed to investigate the occurrence of electrophysiological abnormalities, particularly generalized peripheral neuropathy (polyneuropathy), in patients with psoriasis vulgaris and to assess their association with disease duration and severity in Basrah, southern Iraq.

Methods. This cross-sectional study included 120 patients diagnosed with psoriasis vulgaris. Neurological examinations, nerve conduction studies, and electromyography were performed to evaluate peripheral nerve function.

Results. The mean age of the participants was 36.96 ± 15 years, and 60.8% were male. Most patients had mild psoriasis (83.3%), while 16.7% had moderate disease. Neurological symptoms were reported by 56.7% of participants. Electrophysiological abnormalities were detected in 59.2%, with carpal tunnel syndrome being the most common (24.2%). Peripheral neuropathy was identified as an isolated finding in 5.8% and in combination with carpal tunnel syndrome in 8.3%, giving an overall prevalence of 14.2%. Disease duration differed significantly across electrophysiological diagnostic groups, with the longest duration observed in patients with peripheral neuropathy. Psoriasis severity showed no significant association with electrophysiological findings.

Conclusion. Electrophysiological abnormalities were common among patients with psoriasis vulgaris, with disease duration showing a significant association with electrophysiological outcomes, unlike disease severity. However, the cross-sectional design, absence of a control group, and lack of patients with severe psoriasis limit causal interpretation and generalizability.

Keywords: psoriasis vulgaris, peripheral neuropathy, electrophysiological studies

INTRODUCTION

Plaque psoriasis (psoriasis vulgaris) is a chronic, immune-mediated inflammatory skin disease characterized by well-defined erythematous plaques covered with silvery-white scales. It is the most common type of psoriasis, accounting for approximately 80–90% of all psoriasis cases. Psoriasis vulgaris is a chronic relapsing-remitting disease, with exacerbations frequently triggered by infections, psychological stress, medications, or physical trauma. Clinical features vary from mild, stable lesions in some patients to extensive, severe plaques in others, affecting quality of life [1].

The clinical manifestations of psoriasis are diverse. Cutaneous lesions may appear as macules, papules, plaques, or pustules. Moreover, the condi-

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tion is not limited to the skin and nails; about 30% of affected patients develop psoriatic arthritis, while uveitis and inflammatory bowel disease may also occur. The course of the illness varies among patients, with dermatological involvement ranging from localized to more widespread disease [2].

Patients with psoriatic arthritis have an increased incidence of neuropathies, most notably carpal tunnel syndrome. This comorbidity can significantly affect pain processing, physical function, and overall quality of life, particularly in patients with higher levels of disease activity. These observations suggest a possible link between systemic inflammation and neurological impairment [3].

Recent laboratory and clinical findings suggest a relationship between neurogenic inflammation and psoriasis, although the prevalence and characteristics of electrophysiological abnormalities in these patients remain poorly understood. Increasing evidence suggests that patients with psoriasis may develop various forms of polyneuropathy. The most commonly reported form is symmetrical, length-dependent neuropathy that predominantly affects sensory nerves and is associated with minimal or no functional limitations. Additionally, a previous study identified electrophysiologically confirmed polyneuropathy in approximately 9% of individuals with psoriasis.

The frequency and characteristics of generalized peripheral neuropathy in patients with psoriasis remain unclear [4]. It is important to distinguish generalized polyneuropathy from focal entrapment neuropathies, such as carpal tunnel syndrome, and from radiculopathy, as these represent distinct electrophysiological entities [5].

Accordingly, the present study aimed to investigate electrophysiological abnormalities in individuals with psoriasis vulgaris, focusing on generalized peripheral neuropathy/polyneuropathy as the primary neurological outcome, and to examine their relationship with disease duration and severity.

MATERIALS AND METHODS

Study design

This study used a cross-sectional design and included 120 patients diagnosed with psoriasis vulgaris. No participant had concomitant psoriatic arthritis. The study was conducted in Basrah Governorate, southern Iraq, among patients attending the dermatology outpatient clinics at Al Sayab Teaching Hospital and Al Basrah Teaching Hospital.

During the study period, patients with psoriasis who attended the dermatology clinics were screened according to predefined inclusion and exclusion criteria. Those meeting the eligibility criteria underwent detailed evaluations, including clinical and neurological examinations, laboratory investigations, and electrophysiological testing with nerve conduction studies (NCS) and electromyography (EMG). Individuals who did not meet the criteria or had incomplete datasets required for analysis were excluded. Only participants who met the eligibility criteria and had complete study data were included in the final analysis. The number of patients who were screened but subsequently excluded was not systematically documented and, therefore, is not reported.

Male and female patients aged 18–60 years with a confirmed diagnosis of psoriasis were eligible for inclusion in the study. Diagnostic evaluation included electrophysiological assessment using nerve conduction studies (NCS) and electromyography (EMG) to confirm the presence of peripheral neuropathic involvement. The study was conducted over a 15-month period, from October 1, 2024, to January 1, 2026.

Participants were classified according to disease severity using the Psoriasis Area and Severity Index (PASI) score. Some enrolled patients were also receiving biologic therapy as part of their treatment.

Patients with autoimmune diseases, diabetes mellitus, infections such as leprosy, tumors, bone marrow disorders, or chronic systemic diseases, including kidney disease, liver disease, and hypothyroidism, were excluded. Diabetes mellitus was defined by either a previous clinical diagnosis or abnormal HbA1c values. HbA1c testing was performed for all participants, and the results were interpreted according to the laboratory's established reference ranges. Thyroid function was assessed in all participants using thyroid-stimulating hormone (TSH) testing.

In addition, participants with other recognized causes of peripheral neuropathy were excluded, including medication-induced neuropathy, nerve injury, pre-existing diagnoses of compression disorders, and vitamin B12 deficiency (assessed through medical history).

Information on body mass index (BMI) and relevant social history was collected for all participants. Each participant underwent a detailed clinical evaluation that included current psoriasis symptoms, disease duration, and treatment type. Treatment exposure was recorded based on the therapy received at the time of assessment, including biologic agents, methotrexate, phototherapy (UV), topical therapy, or combinations of these treatments.

The specific biologic agents received by participants [such as Altebrel (etanercept), adalimumab, ixekizumab, infliximab, or golimumab] and the duration of treatment were documented. Those not receiving psoriasis therapy at the time of assessment were classified as untreated.

Additional information was collected regarding the presence of other skin conditions and symptoms suggestive of peripheral neuropathy.

Neurological symptoms were evaluated using a structured questionnaire developed specifically for the study and administered to all participants before electrophysiological testing. Information on neurological complaints (sensory and motor), such as numbness, pain, tingling, and combinations of these symptoms, was collected. A standardized neurological examination was also performed, but its findings were not documented as an independent outcome measure.

Before participation, the study objectives were explained to all patients, and informed consent was obtained. Electrophysiological testing was performed using a Nihon Kohden Neuropack S3 system at a room temperature of approximately 25°C. Heating during winter and air conditioning during summer were used to maintain this temperature. Both surface and needle electrodes were used for data collection.

Motor nerve conduction studies were performed on the median, ulnar, peroneal, and tibial nerves. Median compound muscle action potentials (CMAPs) were recorded from the abductor pollicis brevis with stimulation at the wrist and antecubital fossa, while ulnar CMAPs were obtained from the abductor digiti minimi with stimulation at the wrist and elbow. These stimulation sites were also used for F-wave assessment.

Peroneal studies included recordings from the extensor digitorum brevis and tibialis anterior with stimulation at the ankle and below the fibular head. Tibial CMAPs were recorded from the abductor hallucis brevis with stimulation at the medial ankle and popliteal fossa, with the distal stimulation site also used for tibial F-wave recording.

Late responses were evaluated using F-wave studies of the ulnar and tibial nerves, with ten F-waves recorded to determine F-wave latency. Sensory conduction was evaluated using antidromic recordings: ulnar sensory nerve action potentials (SNAPs) from digit 5 with wrist stimulation, median SNAPs from digit 2 with wrist stimulation between the flexor carpi radialis and palmaris longus tendons, and sural SNAPs posterior to the lateral malleolus with stimulation at the posterolateral calf.

Needle electromyography (EMG) was performed on selected muscles using standard insertion and activation techniques. In the upper limb, the first dorsal interosseous was examined with needle placement between the first and second metacarpophalangeal joints and activation by index finger abduction. The triceps brachii (lateral head) was studied with needle insertion midway between the lateral epicondyle and shoulder, with activation by elbow extension, while the deltoid was assessed through medial shoulder insertion and activation by shoulder abduction.

Lower limb recordings included the extensor hallucis longus with needle insertion above the ankle and activation by great toe extension, the tibialis anterior with insertion lateral to the tibial crest and activation by ankle dorsiflexion, the vastus medialis with medial thigh insertion and activation by knee extension, and the gastrocnemius with posterior calf insertion and activation by plantarflexion or standing on tiptoe.

Reference values for motor nerve conduction studies were as follows: median nerve distal latency ≤4.4 ms, amplitude ≥4 mV, and conduction velocity ≥49 m/s; ulnar nerve latency ≤3.3 ms, amplitude ≥6 mV, and velocity ≥49 m/s; peroneal nerve (extensor digitorum brevis) latency ≤6.5 ms, amplitude ≥2 mV, and velocity ≥44 m/s; peroneal nerve (tibialis anterior) latency ≤6.7 ms, amplitude ≥3 mV, and velocity ≥44 m/s; and tibial nerve latency ≤5.8 ms, amplitude ≥4 mV, and velocity ≥41 m/s.

Late responses included ulnar F-wave latency ≤32 ms and tibial F-wave latency ≤56 ms. Sensory conduction values were defined as ulnar peak latency ≤3.1 ms, amplitude ≥17 μV, and velocity ≥50 m/s; median peak latency ≤3.5 ms, amplitude ≥50 μV, and velocity ≥50 m/s; and sural peak latency ≤4.4 ms, amplitude ≥6 μV, and velocity ≥40 m/s [6,7].

Neuropathic disorders were diagnosed according to established electrodiagnostic criteria. Peripheral neuropathy was identified based on electrophysiological abnormalities that varied according to the underlying pathology. Axonal involvement was characterized by reduced amplitudes with relatively preserved conduction velocities and latencies, together with EMG evidence of denervation and reinnervation.

Demyelinating involvement (demyelinating neuropathy) was identified by marked slowing of conduction velocity and prolonged distal latencies, while amplitudes were generally maintained and EMG morphology remained normal. These distinctions formed the basis for differentiating axonal from demyelinating processes.

Carpal tunnel syndrome (CTS) was diagnosed using established electrodiagnostic criteria, with particular attention to prolongation of the median sensory peak latency across the wrist. In this study, a peak latency exceeding 3.5 ms at the second digit was considered abnormal and suggestive of CTS, consistent with the reference values reported by Preston and Shapiro, who identified ≤3.5 ms as the upper limit of normal. The evaluation formed part of the routine median nerve conduction study across the wrist.

Radiculopathy was primarily identified through EMG findings of denervation in muscles supplied by the affected root, including fibrillations, positive

sharp waves, reduced recruitment, and polyphasic or prolonged motor unit action potentials (MUAPs). Nerve conduction studies in such cases were often within normal limits [6].

Electrophysiological studies were conducted by a specialized neurophysiologist. The examiner was informed of the study protocol.

Statistical analysis

Data were analyzed using SPSS software, version 26 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages.

The primary outcome was the presence and type of electrophysiological abnormalities of the peripheral nervous system, assessed by neurological examination, nerve conduction studies, and electromyography. The abnormalities evaluated included peripheral neuropathy/polyneuropathy and carpal tunnel syndrome.

The primary analysis examined the association between psoriasis disease duration and electrophysiological abnormalities, particularly peripheral neuropathy. A secondary analysis assessed the association between psoriasis severity (mild versus moderate) and electrophysiological abnormalities.

For comparisons of continuous variables between two groups, the independent-samples t-test was used for normally distributed data, whereas the Mann–Whitney U test was used for non-normally distributed data. For comparisons among more than two groups, one-way analysis of variance (ANOVA) was applied to normally distributed data, followed by appropriate post hoc pairwise comparisons when significant differences were detected.

The Kruskal–Wallis test was used for non-normally distributed continuous variables across more than two groups. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Correlations were assessed using Pearson's correlation coefficient for normally distributed variables and Spearman's rank correlation coefficient for non-normally distributed variables. A two-sided p-value ≤ 0.05 was considered statistically significant.

Ethical considerations and approvals

The study was approved by the Basrah Health Directorate's study approval committee (approval No. 2044, dated September 25, 2024).

RESULTS

Table 1 shows the sociodemographic and lifestyle characteristics of the participants. The mean age was 36.96 ± 15 years, and the mean BMI was 25.99 ± 4.6 kg/m^2. Males accounted for 60.8% of the sample. Almost half of the participants were employed (47.5%), while 30% were not employed and 22.5% were students. Most participants were nonsmokers (65%) and did not consume alcohol (95.8%).

TABLE 1. General characteristics of patients with psoriasis (n = 120)

Characteristic	Category	Value
Age (years)	Mean ± SD	36.96 ± 15
Sex	Male, n (%)	73 (60.8 %)
	Female, n (%)	47 (39.2 %)
BMI (kg/m²)	Mean ± SD	25.99 ± 4.6
Occupation	Working, n (%)	57 (47.5%)
	Not working, n (%)	36 (30%)
	Student, n (%)	27 (22.5%)
Smoking	Smoker, n (%)	42 (35%)
	Non-smoker, n (%)	78 (65%)
Alcohol consumption	No, n (%)	115 (95.8%)
	Yes, n (%)	5 (4.2%)

BMI: body mass index; SD: standard deviation.

Table 2 summarizes the clinical characteristics, treatment distribution, and neurological symptoms. The mean duration of psoriasis was 47.92 ± 113.6 months, with a mean PASI score of 6.44 ± 4.9. Most patients had mild psoriasis (83.3%), while 16.7% had moderate disease; no cases of severe psoriasis were observed.

Biologic therapy was the most frequently used systemic treatment (31.7%), with Altebrel being the predominant agent (22.5%). Other biologics, including adalimumab, ixekizumab, infliximab, and golimumab, were prescribed less commonly. The mean duration of biologic therapy was 6.3 ± 14.66 months. Topical therapy (27.5%), methotrexate (14.2%), phototherapy (13.3%), and combined phototherapy and topical therapy (10.0%) were also represented, while 3.3% of participants were untreated at the time of assessment.

Neurological symptoms were reported in 68 participants (56.7%). Numbness was the most frequent complaint (33.3%), followed by combined symptoms (20%). Pain and tingling were less frequent (1.7% each). In contrast, 52 participants (43.3%) reported no symptoms.

Table 3 summarizes the nerve conduction study findings in patients with mild psoriasis (n = 100) compared with those with moderate psoriasis (n = 20). The electrophysiological measurements were largely similar between the two groups.

TABLE 2. Psoriasis duration, PASI score, disease severity, treatment, and neurological symptoms (n = 120)

Characteristic	Category	Value
Psoriasis duration (months)	Mean ± SD	47.92 ± 113.6
PASI score	Mean ± SD	6.44 ± 4.9
Biologic treatment duration (months)	Mean ± SD	6.3 ± 14.66
Psoriasis severity	Mild, n (%)	100 (83.3%)
	Moderate, n (%)	20 (16.7%)
Treatment	Biologic therapy, n (%)	38 (31.7%)
	Altebrel (etanercept), n (%)	27 (22.5%)
	Adalimumab, n (%)	5 (4.2%)
	Ixekizumab, n (%)	4 (3.3%)
	Infliximab, n (%)	1 (0.8%)
	Golimumab, n (%)	1 (0.8%)
	Topical therapy, n (%)	33 (27.5%)
	Methotrexate, n (%)	17 (14.2%)
	Phototherapy (UV), n (%)	16 (13.3%)
	Phototherapy and topical therapy, n (%)	12 (10%)
	No treatment, n (%)	4 (3.3%)
Neurological symptoms	No symptoms, n (%)	52 (43.3%)
	Symptoms present, n (%)	68 (56.7%)
	Numbness, n (%)	40 (33.3%)
	Pain, n (%)	2 (1.7%)
	Tingling, n (%)	2 (1.7%)
	Combined symptoms, n (%)	24 (20%)

PASI: Psoriasis Area and Severity Index; SD: standard deviation; UV: ultraviolet

TABLE 3. Nerve conduction study findings according to psoriasis severity (n = 120)

Nerve	Parameter	Side	Mild psoriasis (n = 100)	Moderate psoriasis (n = 20)	p-value
Median motor nerve	D.L	RT	3.612 ± 1.119	3.135 ± 0.4	0.081
	A	RT	9.829 ± 2.191	10.205 ± 1.44	0.341
	CV	RT	58.567 ± 6.99	57.565 ± 6.35	0.554
	D.L	LT	4.616 ± 8.73	3.333 ± 0.54	0.513
	A	LT	8.957 ± 2.24	10.095 ± 2.46	0.044
	CV	LT	57.647 ± 9.51	58.715 ± 4.08	0.624
Ulnar motor nerve	D.L	RT	2.69 ± 6.3	2.46 ± 0.48	0.724
	A	RT	9.10 ± 1.90	9.79 ± 1.75	0.139
	CV	RT	59.92 ± 9.95	63.19 ± 16.16	0.236
	D.L	LT	3.22 ± 6.75	2.41 ± 0.29	0.594
	A	LT	9.126 ± 1.98	9.16 ± 1.73	0.943
	CV	LT	58.7 ± 12.87	59.66 ± 8.41	0.752
Peroneal nerve	D.L	RT	4.215 ± 1.16	4.615 ± 2.17	0.431
	A	RT	4.461 ± 2.1	4.815 ± 3.36	0.655
	CV	RT	47.255 ± 6.36	47.36 ± 9.576	0.693
	D.L	LT	5.037 ± 5.95	4.19 ± 2.17	0.533
	A	LT	4.217 ± 1.89	3.53 ± 2.24	0.154
	CV	LT	46.887 ± 5.29	45.325 ± 9.08	0.465
Tibial nerve	D.L	RT	4.024 ± 1.066	3.985 ± 0.593	0.820
	A	RT	10.178 ± 4.52	9.545 ± 5.22	0.579
	CV	RT	45.645 ± 7.47	43.295 ± 7.472	0.175
	D.L	LT	3.849 ± 1.11	3.65 ± 0.71	0.313
	A	LT	10.768 ± 6.91	10.76 ± 5.82	0.996
	CV	LT	44.385 ± 4.56	41.03 ± 9.68	0.018
Median sensory nerve	D.L	RT	2.44 ± 0.5	2.395 ± 0.26	0.529
	A	RT	40.155 ± 17.85	47.86 ± 24.46	0.102
	CV	RT	53.567 ± 12.6	54.355 ± 6.69	0.787
	D.L	LT	2.452 ± 0.41	2.42 ± 0.39	0.734
	A	LT	45.106 ± 21.84	47.15 ± 30.436	0.722
	CV	LT	53.0 ± 9.81	53.0 ± 4.89	0.995
Ulnar sensory nerve	D.L	RT	2.017 ± 0.22	2.11 ± 0.12	0.015
	A	RT	39.207 ± 15.4	42.36 ± 20.96	0.436
	CV	RT	53.347 ± 6.48	51.93 ± 6.35	0.372
	D.L	LT	2.089 ± 0.29	2.22 ± 0.31	0.072
	A	LT	39.775 ± 20.01	44.86 ± 18.53	0.296
	CV	LT	52.429 ± 8.76	49.865 ± 5.29	0.210
Sural nerve	D.L	RT	2.647 ± 2.19	2.4 ± 0.45	0.617
	A	RT	14.04 ± 6.33	14.09 ± 6.4	0.975
	CV	RT	52.869 ±15.89	49.5 5.58	0.352
	L	LT	2.273 ± 2.24	2.45 ± 0.38	0.581
	A	LT	13.911 ± 7.28	13.41 ± 5.48	0.771
	CV	LT	50.813 ± 12.36	50.445 ± 5.857	0.897
Ulnar F-wave		RT	29.378 ± 5.75	29.585 ± 2.26	0.875
		LT	29.153 ± 4.187	30.205 ± 2.23	0.278
Tibial F-wave		RT	50.746 ± 7.06	52.8 ± 6.31	0.229
		LT	51.73 ± 7.09	53.295 ± 6.4	0.336

NCS: nerve conduction study; DL: distal latency (ms); A: amplitude (mV for motor nerves; μV for sensory nerves); CV: conduction velocity (m/s); RT: right; LT: left. F-wave latency is expressed in milliseconds.
Statistical test: Independent-samples t-test.

Most motor, sensory, and F-wave parameters showed no significant differences, suggesting that greater psoriasis severity was not associated with widespread changes in peripheral nerve conduction. Only a limited number of variables showed statistically significant differences between the groups.

Patients with moderate psoriasis had a higher left median motor nerve amplitude than those with mild disease. In patients with moderate psoriasis, the left tibial motor nerve conduction velocity was lower, while the right ulnar sensory nerve distal latency was prolonged.

Although these differences reached statistical significance, they were isolated and were not accompanied by similar changes in related electrophysiological parameters. Overall, when nerve conduction parameters were assessed by disease severity, the electrophysiological profiles of patients with mild and moderate psoriasis were largely similar.

TABLE 4. Needle electromyography findings according to psoriasis severity (n = 120)

Muscle	EMG parameter	Side	Mild psoriasis (n = 100)	Moderate psoriasis (n = 20)	p-value
FDI	A	RT	0.559 ± 0.469	0.734 ± 0.622	0.244
	MUAP duration	RT	11.41 ± 1.63	11.88 ± 2.17	0.273
	A	LT	0.582 ± 0.435	0.743 ± 0.623	0.282
	MUAP duration	LT	11.74 ± 1.41	12.47 ± 1.71	0.044*
DEL	A	RT	0.621 ± 0.47	0.905 ± 0.73	0.111
	MUAP duration	RT	12.26 ± 1.5	12.98 ± 2.4	0.221
	A	LT	0.62 ± 0.45	0.95 ± 0.78	0.085
	MUAP duration	LT	12.5 ± 1.5	13.2 ± 2.5	0.277
TRI	A	RT	0.68 ± 0.55	0.94 ± 0.71	0.125
	MUAP duration	RT	12.2 ± 1.5	12.9 ± 2.1	0.205
	A	LT	0.63 ± 0.41	0.97 ± 0.77	0.071
	MUAP duration	LT	12.09 ± 1.4	13.2 ± 2.06	0.029*
TA	A	RT	0.88 ± 0.69	1.04 ± 0.79	0.356
	MUAP duration	RT	12.9 ± 2.2	13.5 ± 2.5	0.276
	A	LT	0.81 ± 0.67	1.02 ± 0.75	0.212
	MUAP duration	LT	12.6 ± 2.3	13.4 ± 2.7	0.157
EHL	A	RT	0.92 ± 0.71	1.0 ± 0.74	0.664
	MUAP duration	RT	12.9 ± 2.1	13.5 ± 2.4	0.250
	A	LT	0.87 ± 0.66	1.03 ± 0.77	0.332
	MUAP duration	LT	12.8 ± 2.2	13.4 ± 2.6	0.307
MG	A	RT	0.78 ± 0.62	1.03 ± 0.73	0.169
	MUAP duration	RT	12.01 ± 1.3	12.8 ± 1.7	0.051
	A	LT	0.71 ± 0.54	1.03 ± 0.77	0.094
	MUAP duration	LT	12.8 ± 2.2	13.4 ± 2.6	0.019*
VM	A	RT	0.7 ± 0.49	0.88 ± 0.66	0.263
	MUAP duration	RT	11.9 ± 1.4	12.8 ± 1.96	0.067
	A	LT	0.68 ± 0.46	0.86 ± 0.6	0.237
	MUAP duration	LT	12.1 ± 1.4	12.7 ± 1.9	0.219

EMG: electromyography; A: amplitude (mV); MUAP: motor unit action potential; MUAP duration: milliseconds; RT: right; LT: left; FDI: first dorsal interosseous; DEL: deltoid; TRI: triceps brachii; TA: tibialis anterior; EHL: extensor hallucis longus; MG: medial gastrocnemius; VM: vastus medialis.
Statistical test: Independent-samples t-test.

TABLE 5. Nerve conduction study and electromyography findings (n = 120)

NCS and EMG findings	n (%)	Sensorimotor PNP	Sensory PNP
Normal	49 (40.8%)	—	—
CTS	29 (24.2%)	—	—
Radiculopathy	10 (8.3%)	—	—
Peripheral neuropathy	7 (5.8%)	7 (5.8%)	0 (0%)
Combined radiculopathy and CTS	15 (12.5%)	—	—
Combined PNP and CTS	10 (8.3%)	8 (6.7%)	2 (1.7%)
Total	120 (100%)		

NCS: nerve conduction study; EMG: electromyography; PNP: peripheral neuropathy; CTS: carpal tunnel syndrome. Percentages are calculated using the full sample (n = 120), except where explicitly indicated otherwise.

Significant variations were noted in left median motor amplitude, left tibial motor conduction velocity, and right ulnar sensory distal latency, but these changes were isolated rather than systematic. No evidence emerged to suggest a generalized deterioration in motor, sensory, or proximal nerve conduction function with increasing disease severity.

Table 4 presents a comparison of needle electromyographic (EMG) findings between patients with mild psoriasis (n = 100) and those with moderate psoriasis (n = 20). Significant differences were confined to MUAP duration measurements.

Patients with moderate psoriasis showed longer MUAP durations in several muscles compared with those with mild disease. In the first dorsal interosseous muscle, the left-sided MUAP duration was significantly longer in patients with moderate psoriasis.

A similar pattern was seen in the left triceps muscle, where MUAP duration was prolonged, and in the left medial gastrocnemius, which also demonstrated a significantly longer reported MUAP duration among patients with moderate psoriasis.

Despite these differences in MUAP duration, amplitudes did not differ significantly between the groups across all three muscles. No significant changes were observed in the deltoid, tibialis anterior, extensor hallucis longus, or vastus medialis.

Overall, MUAP amplitudes did not differ significantly between patients with mild and moderate psoriasis in any of the examined muscles. Significant differences in MUAP duration were limited to the left first dorsal interosseous, left triceps, and left medial gastrocnemius muscles. These abnormalities were restricted to a small number of muscles and were not accompanied by changes in MUAP amplitude.

Additionally, no consistent pattern of abnormalities was observed across the other examined muscles. Together, these findings do not support the presence of widespread neuromuscular dysfunction associated with increasing psoriasis severity.

Table 5 presents the nerve conduction study (NCS) and electromyography (EMG) findings among 120 participants. Normal electrophysiological results were identified in 49 patients (40.8%). Among the abnormal findings, carpal tunnel syndrome (CTS) was the most frequently observed isolated diagnosis, affecting 29 patients (24.2%). This was followed by the coexistence of radiculopathy and

TABLE 6. Comparison of psoriasis disease duration across electrophysiological diagnostic groups

NCS and EMG findings	n	Mean rank
Normal	49	49.67
CTS	29	59.07
Radiculopathy	10	65.55
Peripheral neuropathy	7	93.57
Combined radiculopathy and CTS	15	77.50
Combined PNP and CTS	10	64.00
Total	120	—

Kruskal–Wallis test: H = 15.193; df = 5; p = 0.01. NCS: nerve conduction study; EMG: electromyography; CTS: carpal tunnel syndrome; PNP: peripheral neuropathy.

TABLE 7. Comparison of electrophysiological findings according to psoriasis severity

Finding	Mild (n = 100)	Moderate (n = 20)	Total (n = 120)
Normal	42 (42%)	7 (35%)	49 (40.8%)
PNP	5 (5%)	2 (10%)	7 (5.8%)
Radiculopathy	10 (10%)	0 (0%)	10 (8.3%)
CTS	23 (23%)	6 (30%)	29 (24.2%)
Radiculopathy + CTS	14 (14%)	1 (5%)	15 (12.5%)
PNP + CTS	6 (6%)	4 (20%)	10 (8.3%)
Total	100 (100%)	20 (100%)	120 (100%)

PNP: peripheral neuropathy; CTS: carpal tunnel syndrome.
Reported p = 0.138; reported effect size = 0.262.
Fisher's exact test.

CTS in 15 patients (12.5%), while isolated radiculopathy was noted in 10 patients (8.3%).

Peripheral neuropathy (PNP) was identified as an isolated finding in 7 patients (5.8%), all of whom had the sensorimotor subtype; no cases of isolated sensory neuropathy were detected. Additionally, 10 patients (8.3%) presented with both PNP and CTS, including 8 cases (6.7%) of sensorimotor neuropathy and 2 cases (1.7%) of sensory neuropathy.

In total, 17 patients (14.2%) demonstrated electrophysiological evidence of peripheral neuropathy, either alone or in combination with CTS. The majority of these cases (15 patients, 88.2%) showed a sensorimotor pattern, while a smaller proportion (2 patients, 11.8%) had purely sensory neuropathy.

Table 6 compares psoriasis disease duration across the different NCS/EMG diagnostic groups. Patients with normal electrophysiological findings had the lowest mean disease duration, while those with isolated peripheral neuropathy had the highest.

The mean disease duration was 42.33 ± 56.1 months among participants with normal electrophysiological findings, 58.07 ± 60.4 months among those with carpal tunnel syndrome (CTS), 123.4 ± 189 months among those with radiculopathy, 251.14 ± 285.1 months among those with peripheral neuropathy (PNP), 95.47 ± 89.2 months among those with combined radiculopathy and CTS, and 80.8 ± 77.7 months among those with combined PNP and CTS.

Disease duration differed significantly among the electrophysiological diagnostic groups (Kruskal–Wallis H = 15.193, df = 5, p = 0.01).

Table 7 shows the relationship between disease severity and electrophysiological findings. In patients with mild psoriasis (n = 100), normal NCS/EMG findings represented the largest proportion, occurring in 42 patients (42%). Carpal tunnel syndrome (CTS) was the second most frequent finding, identified in 23 patients (23%). The coexistence of radiculopathy and CTS was observed in 14% of patients with mild psoriasis, whereas isolated peripheral neuropathy (PNP) and combined PNP and CTS were observed less commonly.

Among patients with moderate psoriasis (n = 20), CTS was identified in 30%. Compared with patients with mild psoriasis, those with moderate disease showed a relatively greater proportion of neuropathic involvement, particularly combined PNP and CTS. This combination was detected in 20% of patients with moderate psoriasis, compared with 6% of those with mild disease.

Notably, isolated radiculopathy was absent among patients with moderate psoriasis. There was no statistically significant association between psoriasis severity (mild versus moderate) and electrophysiological findings (p = 0.138). The reported effect size was 0.262, indicating a small-to-moderate association.

DISCUSSION

This study examined the occurrence of electrophysiological abnormalities in patients with psoriasis vulgaris and their possible association with disease duration and severity. The findings revealed that neurological symptoms were common among patients with psoriasis, although electrophysiological evidence of generalized peripheral neuropathy (PNP) was identified in only a limited number of cases. The most commonly observed isolated electrophysiological abnormality was carpal tunnel syndrome. Moreover, longer disease duration was observed among participants with generalized PNP compared with those with normal electrophysiological findings. In contrast, disease severity showed no statistically significant overall association with electrophysiological abnormalities. These findings should be interpreted with caution, as the cross-sectional design does not allow conclusions regarding temporal or causal relationships. Similarly, the differences observed in nerve conduction parameters between severity groups cannot be taken as evidence that psoriasis severity directly contributes to electrophysiological changes. Moreover, the absence of patients with severe psoriasis limits the ability to assess whether such abnormalities are more common in individuals with advanced disease.

These results suggest a possible link between psoriasis and peripheral nerve abnormalities, consistent with the understanding of psoriasis as a multisystem inflammatory disorder [8].

The study included 120 patients with psoriasis vulgaris. The mean age was 36.96 ± 15 years, and males accounted for 60.8% of the study population. The prevalence of psoriasis generally increases with advancing age, reaching its highest levels in individuals aged approximately 60–70 years before showing a gradual decline in older age groups. Previous studies have reported conflicting findings regarding sex differences in psoriasis prevalence, although some have noted a slight male predominance [9].

Regarding disease characteristics, the mean duration of psoriasis was 47.92 ± 113.6 months, reflecting considerable variability.

The mean PASI score was 6.44 ± 4.9, reflecting generally mild disease activity. Most patients (83.3%) were classified as having mild psoriasis, while 16.7% had moderate disease, and no cases of severe psoriasis were identified. The predominance of mild disease may be related to ongoing treatment, as many patients were receiving therapies such as biologic agents, methotrexate, topical medications, or phototherapy, which may have contributed to improved disease control and lower PASI scores [10]. However, given the cross-sectional design of the study and the fact that treatment effectiveness was not specifically assessed, it cannot be determined whether treatment exposure led to the relatively low PASI scores observed. Moreover, the study setting may have influenced the distribution of disease severity, as participants were recruited from outpatient clinics where patients with psoriasis may have relatively stable or adequately controlled disease.

The predominance of mild psoriasis observed in the present study is consistent with previous reports indicating that most patients have limited skin involvement. It has been estimated that approximately 50% of patients have mild disease affecting less than 3% of the body surface area, while around 78% have mild-to-moderate disease involving less than 10% of the body surface area. Severe and extensive psoriasis is considerably less common, which is consistent with the severity distribution observed in the present study [11].

Although most patients had mild psoriasis and relatively low PASI scores, neurological symptoms and electrophysiological abnormalities were detected in a considerable proportion of the study population. This observation suggests that peripheral nerve abnormalities may occur independently of the severity of cutaneous manifestations, including in patients with clinically mild disease [12].

Electrophysiological examination showed that only 40.8% of patients had entirely normal NCS and EMG findings, whereas 59.2% exhibited at least one electrophysiological abnormality. This finding indicates that electrophysiological abnormalities were frequently detected among patients with psoriasis, although their prevalence relative to individuals without psoriasis could not be determined. The most commonly observed isolated electrophysiological abnormality was carpal tunnel syndrome (CTS), identified in 24.2% of patients. The frequency of CTS may suggest a possible susceptibility to entrapment neuropathies among patients with psoriasis. Chronic inflammation, tissue edema, altered connective tissue structure, and musculoskeletal involvement may contribute to median nerve compression within the carpal tunnel [5,13].

The relatively high frequency of CTS observed in the current study suggests that focal nerve compression syndromes should be considered when evaluating neurological complaints in patients with psoriasis. This finding is consistent with previous electrophysiological studies describing entrapment neuropathies and other peripheral nerve abnormalities in patients with psoriasis [5,14]. Carpal tunnel syndrome is a focal entrapment neuropathy caused by compression of the median nerve at the wrist [15].

Radiculopathy was identified as an isolated diagnosis in 8.3% of patients. Although radiculopathy may result from mechanical rather than inflammatory causes, its presence may contribute to neurological symptoms and electrophysiological abnormalities in some patients. Radiculopathy should therefore be considered when interpreting neurological manifestations in patients with psoriasis, particularly in those presenting with radicular pain, sensory disturbances, or motor deficits [16].

Peripheral neuropathy was identified in 5.8% of patients as an isolated electrophysiological abnormality. This prevalence increased to 14.2% after including patients who had PNP in combination with carpal tunnel syndrome. The presence of peripheral neuropathy in a subset of patients is consistent with the possibility of neurological involvement in psoriasis, although the present study cannot establish a causal association. A considerable proportion of patients had more than one electrophysiological abnormality. The findings of the present study are consistent with previous research reporting an increased risk of peripheral neuropathy among patients with psoriasis compared with controls. Previous investigators have suggested that chronic immune-mediated inflammation may contribute to peripheral nerve dysfunction [4].

Combined radiculopathy and carpal tunnel syndrome were observed in 12.5% of cases, while the coexistence of PNP and carpal tunnel syndrome was identified in 8.3% of patients. These combinations involve distinct electrophysiological diagnoses. The

coexistence of multiple abnormalities may complicate the interpretation of neurological symptoms and electrophysiological findings. Therefore, generalized peripheral neuropathy (PNP), carpal tunnel syndrome (CTS), and radiculopathy should be regarded as separate diagnostic entities when evaluating neurological manifestations in patients with psoriasis.

The prevalence of neurological symptoms in the current study exceeded the prevalence of electrophysiologically confirmed generalized peripheral neuropathy. This discrepancy may have several explanations. Some neurological symptoms may be attributable to other electrophysiological abnormalities identified in the study, such as carpal tunnel syndrome or radiculopathy, rather than generalized peripheral neuropathy. Additionally, nerve conduction studies and electromyography may not detect all forms of peripheral nerve dysfunction. Small-fiber neuropathy is one possible explanation, as small-fiber abnormalities can produce sensory symptoms despite normal findings on routine NCS and EMG.

Small-fiber neuropathy was not directly evaluated in this study. Therefore, its presence cannot be excluded in patients who report neurological symptoms despite normal nerve conduction studies and electromyography. This possibility should be regarded as a potential explanation rather than an established finding.

The present study found a statistically significant difference in disease duration across electrophysiological diagnostic groups, with the highest mean rank observed among patients with isolated peripheral neuropathy. This finding may suggest that the chronic inflammatory burden associated with long-standing psoriasis could contribute to peripheral nerve dysfunction [17]. The observed relationship between disease duration and PNP may be explained by the cumulative effects of chronic inflammation. Long-term immune activation and sustained production of inflammatory cytokines may promote oxidative stress, vascular abnormalities, and neural injury, ultimately affecting peripheral nerve function. However, longitudinal studies are needed to determine whether the risk of neurological involvement increases with longer psoriasis duration [18].

The relationship between psoriasis severity and electrophysiological results was evaluated using the mild and moderate disease categories. Although PNP and combined neuropathic abnormalities appeared more frequently in patients with moderate psoriasis than in those with mild disease, these differences did not reach statistical significance. This finding suggests that, within the studied population, neurological involvement was not clearly associated with current cutaneous disease severity.

A higher frequency of neuropathic abnormalities was observed among patients with moderate psoriasis. Isolated PNP was detected in 10% of patients with moderate disease compared with 5% of those with mild psoriasis. Likewise, the coexistence of PNP and carpal tunnel syndrome was more common in the moderate psoriasis group (20%) than in the mild disease group (6%). Although these findings suggest a possible increase in neurological involvement with greater disease severity, the overall association was not statistically significant. Therefore, a definitive relationship between psoriasis severity and peripheral nerve involvement could not be established in the present study.

Several factors may explain the absence of a statistically significant association between disease severity and PNP. The study population consisted predominantly of patients with mild psoriasis (83.3%), with relatively few patients with moderate disease and none with severe disease. This uneven distribution may have limited the statistical power of the analysis, thereby reducing the ability to detect significant differences between groups. Moreover, the PASI score reflects disease activity at a single point in time and may not adequately represent the cumulative inflammatory burden experienced throughout the disease course. The absence of a significant association between disease severity and neuropathy suggests that current cutaneous manifestations may not fully reflect peripheral nerve involvement.

The numerically higher frequency of neuropathic abnormalities detected in patients with moderate psoriasis may still suggest a potential link between disease activity and peripheral nerve involvement. This association may become clearer in larger studies that include patients with severe disease. Further research encompassing a broader range of psoriasis severity is therefore needed.

The results of the current study are consistent with previous reports suggesting that the relationship between psoriasis severity and peripheral nerve involvement is complex and remains insufficiently established. Previous research has also reported a higher prevalence of peripheral neuropathy among patients with psoriasis than among healthy controls, without demonstrating a consistent correlation between electrophysiological abnormalities and disease severity [19].

Treatment characteristics may affect both psoriasis severity and electrophysiological findings. A substantial proportion of patients were receiving psoriasis-directed therapies, including biologic agents, methotrexate, phototherapy, and topical treatment. These treatments may have contributed to the relatively low PASI scores and predominance of mild disease observed in this study. Therefore, disease severity assessed at the time of examination may not fully represent the cumulative inflammatory burden experienced over the course of the disease. Because the

PASI score measures current disease activity, patients with long-standing psoriasis may have low scores following successful treatment despite previous periods of substantial inflammation.

In addition, some evidence suggests that peripheral neuropathy may occur as an adverse event associated with biologic therapy, particularly tumor necrosis factor (TNF) inhibitors. However, clinical evidence regarding biologic agent-associated peripheral neuropathy (BAPN) remains limited [20].

Previous research has shown that biologic agents targeting key inflammatory pathways, including TNF-α, IL-17, and IL-23, are highly effective in reducing disease activity and systemic inflammation. Their anti-inflammatory effects may also influence peripheral nerve involvement by reducing inflammation-related neural injury [21].

Limitations

1. One important limitation of this study is the absence of a psoriasis-free control group. Without such a comparison, the electrophysiological abnormalities observed cannot be directly compared with those occurring in individuals without psoriasis. Consequently, it is not possible to determine whether the frequency of peripheral neuropathy, carpal tunnel syndrome, radiculopathy, or other abnormalities was higher than in the general population. The lack of a control group also limits the ability to establish a specific association between psoriasis and peripheral nerve dysfunction. Future case-control studies including appropriately matched psoriasis-free participants are needed to clarify whether these findings occur more frequently among patients with psoriasis.
2. The study population consisted predominantly of patients with mild psoriasis, with only a small number having moderate disease and no patients with severe psoriasis. This may limit the ability to detect a significant association between disease severity and electrophysiological abnormalities.
3. The cross-sectional design does not allow causal relationships between psoriasis duration and peripheral nerve involvement to be established. Thus, it remains unclear whether the identified neurological abnormalities developed during the course of psoriasis or were already present beforehand. In addition, the absence of a healthy control group limits the ability to establish whether the observed abnormalities are specifically related to psoriasis.
4. Most patients were receiving treatment, including biologic therapies, which may have contributed to lower disease activity and potentially influenced electrophysiological findings through their anti-inflammatory effects. Moreover, psoriasis severity was assessed using a single PASI score, which reflects disease activity only at the time of examination and may not adequately capture the cumulative inflammatory burden throughout the disease course.
5. Neurological assessment in this study was limited to nerve conduction studies and needle EMG. Additional diagnostic procedures for detecting small-fiber neuropathy, such as skin biopsy, were not performed. Therefore, early or small-fiber peripheral nerve involvement may have gone undetected.
6. Several potential confounders may have influenced the electrophysiological findings. Age and BMI are known to affect nerve conduction parameters and may also be associated with psoriasis characteristics, yet no multivariable analysis was performed to adjust for these factors. The absence of psoriatic arthritis in the study population prevented evaluation of its possible impact on electrophysiological abnormalities.

CONCLUSIONS

Electrophysiological abnormalities were identified in patients with psoriasis vulgaris. Carpal tunnel syndrome was the most frequently observed isolated abnormality, followed by radiculopathy and peripheral neuropathy.

Peripheral neuropathy was identified as an isolated electrophysiological finding in 5.8% of patients, with an overall prevalence of 14.2% when cases combined with carpal tunnel syndrome (CTS) were included. Neurological complaints were reported more frequently than electrophysiologically confirmed generalized peripheral neuropathy, suggesting that some symptoms may be attributable to other neurological conditions or abnormalities not detected by routine electrophysiological testing.

A significant difference in disease duration was observed across electrophysiological diagnostic groups, with the highest mean rank among patients with isolated peripheral neuropathy. In contrast, no significant relationship was observed between psoriasis severity and NCS/EMG findings, suggesting that greater disease severity was not associated with consistent, generalized changes in electrophysiological parameters within the studied population.

These findings indicate that peripheral neuropathy, carpal tunnel syndrome, and radiculopathy can be detected in patients with psoriasis vulgaris; however, they should be regarded as distinct electrophysiological diagnoses rather than manifestations of a single neuropathic process. Because the study employed a cross-sectional design and did not include a psoriasis-free control group, the results cannot establish that these abnormalities are specifically attributable to psoriasis or that psoriasis plays a causal role in their development.

Conflicts of interest:

The authors declare that they have no conflicts of interest.

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