

# **Peripheral Neuropathy In Systemic Lupus Erythematosus**

Thesis Submitted for Partial Fulfillment of Master Degree of  
Internal Medicine

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# التهاب الأعصاب الطرفي في مرضى الذئبة الحمراء

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا سبحانك لا علم لنا

إلا ما علمتنا إنك أنت

العليم الحكيم

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# **List of Abbreviations**

DML: distal motor latency

EMG: electro-myography

NCS: nerve conduction study

NCV: nerve conduction velocity

# Introduction

Systemic lupus erythematosus (SLE) is an inflammatory, autoimmune, multiorgan disease often involving the central and peripheral nervous systems. The prevalence of PN is relatively high in SLE and occurs more frequently in patients with central nervous system involvement and high SLE-disease activity index (*Goransson et al., 2006 and Florica et al., 2011*).

The *American College of Rheumatology* (ACR) (1999) established case definitions for 19 central and peripheral nervous system syndromes. Central neuropsychiatric lupus (NPSLE) include headache, seizures, cerebrovascular disease, psychosis and movement disorder (chorea). Peripheral NPSLE include cranial cranial, peripheral and autonomic neuropathy in addition to myasthenia gravis.

The nervous system is commonly affected in both children and adults with SLE, where neuropsychiatric lupus (NPSLE) is associated with a worse prognosis and more cumulative damage. Neuropsychiatric lupus (NPSLE) manifestations can occur in the absence of either serologic activity or other systemic disease manifestations (*Hanly et al., 2007*).

A large number of etiopathophysiologic processes are involved: antineuronal antibodies, antibodies against ribosomal P-protein, and cytokines have been implicated in the pathogenesis of diffuse neuropsychiatric symptoms. Focal neurologic symptoms are the consequence of vascular injury induced by circulating immune complex, occlusive vasculopathy as a result of endothelial cell activation induced by cytokines and complement activation, or macro- and microvascular thrombosis induced by antiphospholipid antibodies. In the later stages of disease, cerebrovascular manifestations are often related to accelerated atherosclerosis, which is entertained by increased intravascular complement turnover and antiphospholipid antibodies (*Arinuma et al., 2008*).

As neuropsychiatric lupus (NPSLE) manifestations can occur in the absence of either serologic activity or other systemic disease manifestations (*Hanly et al., 2007*), this encourage using simple non invasive studies as neurophysiological studies in assessing peripheral nerve function in patients with SLE.



# **Aim of the Work**

To study the association of peripheral neuropathy with systemic lupus erythematosus using neurophysiological study and to correlate clinical and laboratory findings with neurophysiological parameters.



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# Subjects and Methods

This study was conducted on 56 SLE patients diagnosed according to the ARA 1982 revised criteria for the classification of SLE (*Tan et al., 1982*) in addition to 30 healthy individuals who were matched in age and sex with the patients to serve as a control group.

All patients were randomly taken from the inpatients of Internal Medicine department and outpatient clinic of rheumatology department in Ain Shams University Hospital.

## **Patients Group:**

This group comprised 56 patients with SLE, all these patients fulfilled the ARA 1982 revised criteria for the classification of SLE (*Tan et al., 1982*).

Exclusion criteria: patients with peripheral neuropathy due to other causes were excluded.

1. Endocrine: diabetes mellitus, hypothyroidism.