Peripheral Neuropathy In Systemic Lupus Erythematosus

Thesis Submitted for Partial Fulfillment of Master Degree of Internal Medicine

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2014

التهاب الأعصاب الطرفي في مرضى الذئبة الحمراء

رسالة مقدمة توطئة للحصول على درجة الماجستير في الأمراض الباطنة

من

الطبيب / محمود محمد فتح الله

بكالوريوس الطب والجراحة العامة - كلية الطب - جامعة عين شمس

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كلية الطب

جامعه عين شمس

2014

قالوا سبحانك لا علم لنا

إلا ما علمتنا إنك أنت

العليم الحكيم

صدق الله العظيم

۞سورة البقرة – الآية 32 ۞

Acknowledgment

First, thanks are all due to **God** for blessing this work until it has reached its end, as a part of his generous help throughout my life.

I would like to direct special thanks to **Prof. Dr. Mohamed Eltayeb Nasser**, Professor of Internal Medicine, Faculty of Medicine, Ain Shams University, for the great support and encouragement he gave me throughout the whole work. It is a great honor to work under his guidance and supervision.

I would like to express my sincere gratitude to **Dr. Walid Anwar Abd Elmohsen**, Assisstant Professor of Internal Medicine, Faculty of Medicine, Ain Shams University, for his close supervision, continuous help, and the tremendous effort he has done in the meticulous revision of the whole work.

I am greatly honored to express my utmost thanks to **Dr. Abd Elrahman Khedr**, Lecturer of Internal Medicine, Faculty of Medicine, Ain Shams University, from whom I received faithful supervision, valuable suggestions, and continuous guidance throughout this work.

I would also like to express my deep gratitude to **Dr. Sherif Hamdy**, Lecturer of Internal Medicine, Faculty of Medicine, Ain Shams University, for his precious advices.

Mahmoud Mohamed Fathallah

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DML: distal motor latency
EMG: electro-myo-graphy
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 ribosimal 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.