

ASSOCIATION OF MULTIPLE NEUROLOGICAL DISEASES IN YOUNG FEMALE: CASE REPORT

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Abstract

A 20-year-old female was diagnosed as a case of epilepsy since 1995. The patient was diagnosed as a case of myasthenia gravis in April 2002. Since 1999, her complaint became more announcing over two years. Until April 2002, when the patient consulted a neurologist, she was diagnosed as case of Myasthenia gravis.

The patient diagnosed as a case of multiple sclerosis then after. The key to the clinical criteria for the diagnosis was lesions disseminated in space and in time.

This case may represent an association of multiple neurological diseases of dysimmune reaction.

Key words: Multiple Sclerosis, Myasthenia and epilepsy

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Introduction

Myasthenia gravis (MG) is not rare, has a prevalence of at least one in 7500. It affects individuals in all age groups, but peaks of incidence occur in women in their twenties and thirties and in men in their fifties and sixties. Overall, women are affected more frequently than men, in a ratio of approximately 3:2^[1]. The neuromuscular abnormalities in MG are brought about by an autoimmune response mediated by specific anti-acetylcholine receptor (anti-AChR) antibodies. The anti-AChR antibodies reduce the number of available AChRs at the neuromuscular junctions^[2].

Multiple sclerosis affects approximately 350,000 Americans and 1.1 million individuals worldwide. The prevalence of MG is probably not affected by geographic variation on the other hand the prevalence of multiple sclerosis has been shown to vary with geographic latitudes^[1].

Using the definition of epilepsy as two or more unprovoked seizures, the incidence of epilepsy is approximately 0.3 to 0.5% in different populations throughout the world, and the prevalence of epilepsy has been estimated at 5 to 10 persons per 1000. Because seizures are common, this clinical problem is encountered frequently during medical practice in a variety of settings^[3]. Although epileptic seizures are uncommon in multiple sclerosis, they are more prevalent than in the general population, which may support an etiological relationship. Gurtubay reported two patients where the epileptic seizures formed part of the first episode of their illness^[4].

The Case:

A 20-year-old female was diagnosed as a case of epilepsy since 1995. The seizures were uncontrolled. The seizures were generalized tonic clonic in nature without focal symptomatology. Tongue biting and incontinence accompanied the seizures, which were followed by confusion for as long as thirty minutes. She was maintained on 300mg of Carbamazepine twice daily

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with poor compliance and variable response.

The patient diagnosed as a case of myasthenia gravis in April 2002. Since 1999, increased in severity over two years, the patient started to have diplopia, drooping of upper eyelids, difficulty in chewing and swallowing, nasal regurgitation, inability of combing her hair and difficulty in climbing stairs. Facial weakness produces a "snarling" expression when the patient attempts to smile. Speech has a nasal timbre. The examination reveals limb weakness is often proximal and asymmetric. This weakness, by exertion with diurnal variation. Deep tendon reflexes were preserved. Until April 2002, when the patient consulted a neurologist, and was diagnosed as case of MG. The diagnosis was confirmed by neostigmine test and electroneurophysiological study. She was kept on pyridostigmine, with good improvement. She was advised for thymectomy but her family refused.

The patient was admitted to our hospital on June 2002 because of impairment of her balance, clumsiness of the hands, and a feeling of weakness and tiredness in the legs.

She was admitted to the hospital on October 2002 because of an acute onset left sided weakness. Over the course of a few days, her left arm and leg became weak and her left leg become numb. She then noticed that her walking went unsteady. Her symptoms progressively worsened up to the time of her admission to hospital. The examination revealed normal fundi, bilateral facial palsy, of an upper motor neuron type more on the right. All the tendon reflexes were hyperactive and were unequal, being more active on the left side than the right and Babinski sign bilaterally. The superficial abdominal reflexes could not be elicited. Finger nose test revealed intention tremor. The patient was unable to walk steadily

along a straight line. Vibration sense was impaired bilaterally over the malleoli and joint sense was impaired in the toes. The remainder of the physical examination disclosed no abnormal findings.

The patient's birth and development were normal. She did not have a history of encephalitis or febrile convulsion. She is unmarried and because of her condition is unemployed. There is family history of MG. Her sister was diagnosed as a case of MG.

Visual evoked potential showed prolonged P₁₀₀ latencies in both eyes, but well preserved waveform. Connective tissue screen normal. Thyroid function test was normal. Brain MRI was normal.

The patient diagnosed as a case of Multiple sclerosis. The key to the clinical criteria for the diagnosis was lesions disseminated in space and in time. She received pulses therapy of methyl prednisolon with Good improvement.

Discussion

This case may represent an association of multiple neurological diseases of dysimmune reaction. The present case may be an example of multiple medical disorders characterized by immune dysregulation and represents the association of MG, multiple sclerosis and epilepsy.

Current evidence indicates that multiple sclerosis is an autoimmune disease that develops in a genetically susceptible individuals who have resided in certain permissive environments^[5]. Multiple sclerosis and MG occasionally are found in association with other diseases but rarely in association with each other. Both diseases are immunogenic in origin, and their association is probably not coincidental^[6].

There have been numerous case reports of the concurrence of MG and multiple sclerosis^[7]. At least 28 cases of MG in combination with multiple

sclerosis have been described in the literature. Margolis and Graves in 1945 described a 43-year-old white female with an 18-year history of transient neurologic signs and symptoms felt to be manifestations of multiple sclerosis. Fleeting ocular palsies develop that never completely cleared, and she noted worsening of her ocular symptoms with fatigue. She responded to neostigmine with clearing of her ptosis and of much of the ocular palsy^[6].

Multiple sclerosis may be present in the MG patient as described by Kean and Hoyt. Patten and associates have described three patients, with an overlap syndrome or both multiple sclerosis and myasthenia gravis. Aita and co-workers described 4 cases of unusual combination of MG and multiple sclerosis^[6].

Certainly, these interesting patients with their unusual combination of diseases raise more questions than they answers.

There have been numerous case reports of the concurrence of MG and multiple sclerosis, again suggesting a common autoimmune basis, but the statistical association is not certain^[1].

The finding of autoimmune system activation in patients with a seizure disorder has led to the suggestion that immune mechanisms may play a role in the pathogenesis of some forms of epilepsy^[8]. Although epileptic seizures are uncommon in multiple sclerosis they are more prevalent than in the general population, which supports an etiological relationship. Similarly, in a considerable proportion of patients with multiple sclerosis and epileptic seizures, alterations in magnetic resonance and electroencephalographic studies that can be correlated with the clinical features of epilepsy were observed. There is great variability with regard to the type of seizure, point at which this occurs during the course of the disease,

degree of recurrence and other aspects^[4].

In 1977, Pechadre et al reported that children with epilepsy who were treated with intramuscular injections of immunoglobulin for recurrent upper respiratory tract infections had a decrease in the frequency and severity of their seizures. This has been supported by several reports^[9].

The suggestion of Masson^[10], may explain how the immune dysregulation affects the nervous system to develop these conditions, by that acquired ionic channel dysfunction resulting from autoimmune aggression. Channelopathies are responsible for muscular diseases certain forms of Mendel's law hereditary epilepsy^[10]. It probably plays a part in the clinical, and particularly the sensitive expression (paresthesia and pain) of certain central nervous system affections, such as multiple sclerosis^[10].

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