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Case Report

Overlap of myasthenia gravis and graves' disease

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Abstract. Both myasthenia gravis and graves' disease are auto-immune diseases. Patients with myasthenia gravis may have evidence of coexisting auto-immune thyroid diseases like graves' disease. The coexistence of two diseases is rarely observed but easily recognized if the association comes to mind. In this report, we present a case of 17-year-old female patient having myasthenia gravis with concomitant graves' disease and is treated successfully with both pyridostigmine and propylthiouracil options.

In conclusion, our case is a good example that the clinical features of autoimmune diseases can overlap and the presence of one auto-immune disease in a patient should require detailed investigations for other autoimmune diseases.

Key words: Myasthenia gravis, thyroid disease, graves' disease

1. Introduction

Myasthenia gravis (MG) is an auto-immune disease characterized by impaired neuromuscular junction transmission of the neural stimuli to the muscles due to circulating anti-acetylcholine receptor antibodies. It causes skeletal muscle fatigue and weakness. The clinical expression of MG varies ranging from a mild localized disease such as ocular myasthenia gravis to a severe generalized disease (1).

Patients with MG may have evidence of coexisting autoimmune thyroid diseases (AITD) as well as other autoimmune disorders like type 1 diabetes, primary hypogonadism, pernicious anemia, vitiligo and adrenal insufficiency (2).

Epidemiological studies proved that AITD occur in approximately 5%-10% of MG patients, whereas a fairly low incidence of MG (approximately 0.2%) has been reported patients with AITD. Grave's disease (GD) is the most frequent AITD associated with MG (3).

Both MG and GD are auto-immune diseases and the coexistence of these two diseases is relatively rare but well recognized if the association comes to mind.

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2. Case report

A 17-year-old female patient was presented to the our outpatient clinic with a history of limitation on outward gaze on the left eye, diplopia, discomfort like pressure or pain sensation in the eyes and drooping of eyelid on the left side which is gradually increasing during 2 months. She had no such complaints in the past, no such family history and also no significant diurnal variations. Neurological examination revealed unilateral proptosis, left lateral rectus muscle weakness and mild tremor in both hands. In the physical examination, we found painless, symmetric, diffuse enlargement of the thyroid gland (goiter).

Magnetic resonance imaging of brain and orbital showed no abnormality. All the investigations including blood, routine urine and biochemical results were normal except thyroid functions. (TSH: 0.0003 µIU/mL, Free T4: 3.53 ng/dL, Free T3: >30.00 pg/mL). High radioactive iodine uptake helped us in ruling of possible other causes of overactive thyroid.

After the implementation of neostigmine test, her weakness was recovered nearly complete so the test was considered as positive. However, repetitive nerve stimulation showed no decremental response. Single fiber electromyography was found to be consistent with an increased jitter. The level of anti-acetylcholine reseptor antibody was measured as 1,53 nmol/L (normal range: up to 0,6 nmol/L). Thoracic computerised tomography scan was consistent with thymus hyperplasia.

With both clinical tests and morphological aspects, MG was diagnosed. Also, diagnosis of GD was made and confirmed by high levels of thyrotrophin, thyroxin and low thyroid stimulating hormone.

She was initially treated with pyridostigmine 60 mg 3 times daily. Treatment of GD was with propylthiouracil 100 mg 3 times daily. After both pyridostigmine and propylthiouracil treatments ptosis, tremor and limited outward eye movement showed marked improvement and has remained well at follow up.

3. Discussion

Myasthenia gravis is an autoimmune neuromuscular disorder characterized by impaired neuromuscular transmission. The coexistence of other AITD including GD in MG is rarely observed but easily recognized. Epidemiological studies show that AITD occur in approximately 5-10% of patients with MG, whereas MG is reported in a fairly low frequency (%0.2) of patients with AITD. The clinical presentation of MG associated with AITD is often restricted to eye muscles. However, the reason for the association of AITD with ocular MG as in our case is unknown. To date, several hypotheses have been proposed. First, ocular and generalized MG might actually represent seperate diseases with different conditions. Second, an immunological cross- reactivity against epitopes or auto-antigens shared by the thyroid and eye muscles might be the basis of this association. The third explanation for the higher frequency of ocular MG in AITD could be that these disorders have a common genetic background (4). Garlepp et al. (5) reported increased association between ocular MG and thyroid immunity and greater frequency of thyroid antibodies in ocular MG than generalized MG. Additionally, Marino et al. (4) showed that GD with clinical evidence of ophthalmopathy had a higher frequency of ocular MG (51.8%) than GD patients without clinical ophthalmopathy (16.6%).

In conclusion, our case is a good reminder that the clinical features of autoimmune diseases can overlap. The presence of one autoimmune disease should require a detailed investigations for other autoimmune diseases. Also, it should be remembered that ptosis is not only expected symptom in MG. If ptosis or paresis of the orbicularis oculi muscle develops in a patient with MG, coincidence of AITD should be considered.

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