In the name of God

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Is there any Association Between Type 2 Diabetes Mellitus and Myasthenia Gravis?

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Abstract:

Myasthenia gravis (MG) is associated with diabetes mellitus (DM) type 1 and other autoimmune disorders. Some cases of MG may progress to DM type 2 following corticosteroid therapies. We reported a 53-year-old woman with DM type 2 who subsequently developed MG. She did not take any corticosteroids prior to MG occurrence. MG may be associated with DM type 2 disease even in the absence of corticosteroid therapy.

Keywords: Myasthenia gravis, Diabetes mellitus type 2, Diabetes mellitus type 1, Autoimmune disorder

Introduction:

The association between myasthenia gravis (MG) and diabetes mellitus (DM) type 1 has been described in the literature. $^{(1, 2)}$ Myasthenia gravis associated DM may result from an autoimmune destruction of pancreatic β cells and may be accompanied by other autoimmune disorders such as autoimmune thyroid dis-

orders or pernicious anemia. (1, 3) Some myasthenia gravis patients may develop hyperglycemia and DM type 2 following corticosteroid initiation. (1) We did not find any case report of MG occurrence in DM type 2 subjects without previous history of corticosteroid therapy in the literature. Here we reported a 53-year-old woman with complicated DM type 2 who

subsequently developed to MG. She had never received corticosteroid drugs before MG occurrence.

Case Report:

A 53-year-old woman with an one and a half year history of type 2 Diabetes Mellitus and under treatment with Glibenclamid (15 mg/day) and Metformin (1500 mg/day) was presented to endocrine clinic of Emam-Khomaini hospital of Urmia city with blurred vision, polyuria, polydipsia, fatigue and 10 kilogram weight loss. Past history was positive for cesarean delivery. There was not a history of the similar disorders or other autoimmune diseases in her relatives. She had normal vital signs. She is an overweight woman with body mass index (BMI) 28.2 kg/m2. Physical examination revealed nasal speech, bilateral ptosis (with greater severity in left side), positive Simpson test, non proliferative diabetic retinopathy in ophtalmoscopic exam, cesarean section scar in her abdomen and mild proximal muscle weakness. Consciousness, sensation, vibration, and cerebellar tests were all intact. The findings of her heart and lung exam were unremarkable.

Laboratory results showed normal CBC, Urine analysis, serum Calcium, phosphorus, renal and liver function tests. Fasting blood sugar (FBS): 425 mg/dl, 2 hour post prandial BS(BS2hpp): 578 mg/dl, Potassium: 4.4 mEq/L, Sodium: 136 mEq/L, Serum freeT4:1.4 ng/dl (0.7-1.8), serum TSH: 1.3 mIU/L (0.17-4.05), ESR: 20 mm/hour, CRP: Negative, CPK: 46 U/L (20-190) and LDH: 559 U/L (NI <480). CXR and ECG results were normal.

On EMG-NCV there was not any evidence of peripheral neuropathy or myogenic changes but mild irritation of L4 and L5 roots were present. Jolly test showed decrescendo response strongly in favor of MG diagnosis.

She received insulin therapy during admission period. Neurologic consult was request because of her myasthenic symptoms and she transferred to neurology ward for management of MG. MG diagnosis and treatment was considered by neurologist based on clinical and laboratory findings. She was discharged to home with Mestinon tablet (60 mg every 8 hours) and Insulin therapy after improving of her diabetes mellitus control and myastenic symtoms.

Discussion:

The occurrence of MG in autoimmune DM type 1 subjects has been described in the literature. (1-4) MG is also associated with other autoimmune disorders including autoimmune thyroid diseases, systemic lupus erythematosus, idiopathic thrombocytopenia and pernicious anemia. (3) DM associated MG is classified as autoimmune DM type 1 or DM type 2 which develops following corticosteroid therapy .We described a 53-year old woman with a previous history of DM type 2 complicated with non proliferative diabetic retinopathy (NPPDR) who subsequently developed with signs and symptoms of MG. We did not check anti GAD and antiislet cell autoantibodies in our patient because they were not available in our local laboratories but the presence of NPPDR and also absence of ketonoria and acidosis in our case strongly was in favor of DM type 2 diagnoses.

In addition to well recognized association of MG and autoimmune DM type I, occurrence of MG in our DM type 2 patient who had never received corticosteroid drugs previously indicates that MG may be associated with DM type 2 disease even in the absence of corticosteroid therapy. Further than autoimmune destruction of pancreatic β cells and corticosteroid therapy other factors may also play a role in the development of myasthenia gravis in diabetic patients.

Conflict of interests:

The authors declare that they do not have any conflict of interest.

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