Pregnancy and myasthenia gravis

A case report

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Summary: Myasthenia gravis in pregnancy is seen very rarely. We present such a case who was diagnosed and treated in our department.

Key words: Myasthenia gravis; Autoimmune diseases.

INTRODUCTION

Myasthenia Gravis (MG), is a rare autoimmune disorder with symptoms such as early muscular fatigue, dyplopia and dysphagia, due to neuromuscular junction disorder ([1]). It is most frequent in women, in their 3rd decade ([2]) and the incidence in pregnancy is 1/20.000 ([1]). There are antibodies 90% against acetylcholine receptors and 50% against striated muscles ([3]).

The differentiation from other myopathies depends on muscular response to anticholinesterase drugs. IgG type antibodies against acetylcholine receptors are important in the diagnosis. Although the myometrium is not involved and the contractions are not effected, the second stage of labor can prolong because the abdominal muscles are attacked.

Anticholinesterase drugs are used in the treatment and the most preferred ones are neostigmine and pridostigmine ([1]). Curare, hydantoins, tranquilizers, ether, halothane, MgSO4, neuroleptics, antibiotics such as aminoglycosides should be avoided ([4-6]), since they cause myasthenic crises. Myasthenic symptoms can arise in newborn.

CLINICAL CASE

A twenty-nine year old, gravida 1 pregnant patient was followed-up by the 24th week of gestation.

Fatigue, eyelid drop, dyplopia and dysphagia during the past 7 years, were present when she was admitted to the neurology clinic in her 2nd month of pregnancy, and she was diagnosed as MG by using the edrophonium test. When admitted to our clinic, she was taking pridostigmine 240 mg/day. Her neurologic examination was normal and the obstetrical findings were appropriate for her gestational period. Complete blood count, liver and renal function tests were normal. Anti DNA, ANA, AMA, ASMA, ARA, antiheterophile, antiparietal and antithyroid antibodies were negative. RF was (−). She was

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followed up weekly and admitted to our clinic on labor at 38th week of gestation. She delivered a 2800 gram female infant with 1st and 5th minute apgar scores of 9 and 10, respectively, with vacuum extraction due to prolonged 2nd stage of labor. The newborn was treated in the pediatric clinic because of her myasthenic symptoms. The patient and the infant were discharged respectively on the 3rd and 15th days of their hospitalization.

DISCUSSION

Pregnancy with MG is very rare. It is due to a defect in postsynaptic nicotinic acetylcholine receptors at the motor end-plate, which leads to a reduction in the end-potential amplitude of striated muscle, caused by the destruction of the postsynaptic membrane by antibody bound complements ([7]). In these cases, acetylcholine receptors are found to be 20-30% less than normal ([8, 9]).

The thymus can also play an important role in the etiology of the disease ([3–5]).

MG can be concomitant with Hashimato thyroiditis 13%, rheumatoid artritis 5-6%, polymyositis-dermatomyositis 1%, sarcoidosis 1%, and SLE 0.5%. In these cases antibodies against acetylcholine receptors and striated muscle are seen in 90% and 50%, respectively. Twenty to forty percent antinuclear and 30% antithyroid antibodies are detected. In our case all autoantibodies were negative.

Clinically dyplopia, dysphagia and early fatigue are seen in MG. Although the muscles are normal in the resting state, early fatigue develops as the day progresses. Skeletal muscle weakness is at the proximal, rather than the distal site of the extremities. In our case the same complaints were present, typically, besides normal neurological examination.

Diagnosis of MG is made by the edrophonium test. Symptoms recover immediately after 2 - 10 mg edrophonium. In our case the diagnosis was made by the same method. IgG type antibody detection against acetylcholine receptors is important in the diagnosis. Immuncomplexes can be seen at neuromusclar junctions in skeletal muscle biopsies ([7]).

There is no prospective controlled study indicating whether the MG attacks increase in pregnancy or not. Although there can be myasthenic attacks during pregnancy, there are some patients who are symptom free during pregnancy.

The myasthenic picture worsens especially during the first trimester and early postpartum period ([4]). In our case myasthenic symptoms became evident during the first trimester and was diagnosed later on.

Myasthenic symptoms can worsen due to surgical intervention and medication. Therefore, curare and its derivatives, narcotic analgesics, $MgSO_4$, tranquilizers, anesthetics such as ether and halothane, adrenergic blockers, antiarythmics, antibiotics like aminoglycoside, Ca-antagonists, steroids, lithium and choloroquine should be avoided. Local anesthesia can be used with care during delivery. The second stage of labor may be prolonged in time, due to insufficient bearing down ([10]), and may necessitate vacuum extraction, as recurred in our case.

Although the effect of myasthenia on fetal movements and other fetal tests is not known exactly, there was no obstetrical abnormality in our case.

MG can be seen transiently in the newborn and as the antibody titer in the newborn circulation decreases, the clinical symptoms cease ([4]). When muscular weakness is determined, anticholinesterases are used for the treatment. Plasmapheresis is advised in severely attacked newborns.

Maternal antibodies can cross to the newborn through breast milk and then cause a myasthenic picture, like anticholinesterase drugs. Therefore if the mater-

nal antibody titer is high, or high dosage of cholinergic drug has been given to the mother or the mother has MG attack, she should not breast feed her infant.

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