

MYASTHENIA GRAVIS AND PREGNANCY

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ABSTRACT

Myasthenia Gravis with pregnancy is a rare concomitance. This case report describes successful outcome of all pregnancies in a lady with this problem. The management is described.

KEY WORDS: *Myasthenia Gravis. Caesarean section (C. section). Neonatal.*

INTRODUCTION

Myasthenia Gravis (MG) is due to the dysfunction of neuromuscular transmission. IgG antibodies to nicotinic acetylcholine receptors (n-ACh-R) are the cause of the disturbant nerve impulse transmission to muscle fibers. They may cross placenta and cause transitory neonatal MG.¹

MG is relatively frequent among women of reproductive age (1:10,000 down to 1:50,000). Pregnancy has a variable effect on MG i.e. a third remain stable, a third improve and one-third get aggravated.² MG signs and symptoms in pregnant women tends to improve throughout second and third trimester coinciding with immunosuppression which normally takes place in that period. Body's immunological response reactivates again at time of delivery and in puerperium as well, which may be the cause of an exacerbation and deterioration of MG.

MG results in increased maternal mortality, morbidity, pregnancy wastage and premature labour. Anti-cholinesterase medicines and corticosteroids are the mainstays of medical therapy of maternal MG and require frequent adjustment during pregnancy due to changing requirements and physiologic changes in absorption and excretion. Thymectomy, plasmapheresis, immunosuppressant drugs, gammaglobulin and ACTH are adjuvants of varying usefulness.³

We describe a case of MG in a lady who was managed successfully in all her pregnancies with good outcome.

CASE REPORT

A 33 years old multigravida lady presented at 8 weeks of gestation for booking, with a history of previous three caesarean sections. In 1995, her first pregnancy ended up in spontaneous abortion. In 1996, she delivered a female baby through C. section performed due to prolonged second stage. The development of classical symptoms of MG during her second pregnancy and delayed recovery from non-depolarizing muscle relaxant (used for general anaesthesia) led to the suspicion of MG. The neonate also suffered from transient neonatal MG.

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Later on, she was investigated and found to have raised anti-cholinesterase receptor antibodies (117 nmol/L), cholinesterase level (4355 μ /L) and serum anti-DNA (3.9 I.V/ml). Other antibodies including ANA, AMSA and AMA were negative. Thyroid function tests and scan was normal. MRI showed enlarged thymus (thymoma) with no lymphadenopathy.

The treatment for MG started with an anti-cholinesterase agent in a dose of 60 mg daily. Thymectomy was carried out in 1998 for enlarged thymus gland and her symptoms further improved. Thereafter, the dose of anti-cholinesterase drug was reduced to half i.e., 30 mg daily during subsequent three pregnancies. Every antenatal and postnatal period was uneventful. She breast-fed her babies and there was no evidence of neonatal MG.

The present pregnancy was supervised intensively by an obstetrician and neurologist. On examination, she had good palatal movement, good grip, abducted her shoulders 40 times without fatigue and could stare at the ceiling for almost 2 minutes without blinking. No alteration in dosage of medicine was advised by neurologist. The acetylcholine receptor antibodies level was 30 nmol/l. No evidence of pre-eclampsia was found and ultrasound showed fetal abnormalities. An elective C. section with bilateral tubal ligation was carried out on term under spinal anaesthesia. A male baby weighing 4 kg was delivered with an APGAR scores of eight at one minute and ten at five minutes. The patient continued her normal oral therapy before and after the operation. Her puerperium was uneventful. After delivery, her acetylcholine receptor antibodies level was 33.6 nmol/l, thyroid antibodies, ANA, ASMA and AMA all were negative.

DISCUSSION

Myasthenia Gravis is an acquired relapsing and remitting autoimmune disorder associated with acetylcholine receptor (AChR) deficiency at the motor end plates, caused by complement fixing antibodies.⁴ The disease was only recognized because of severe weakness and was fatal within a short course of time.⁵ The patient under discussion initially had symptoms of generalized weakness and easy fatigability after moderate work like climbing the stairs or getting up from squatting position, which exacerbated during pregnancy. However, symptoms of anaemia which is so common in our country may be mistaken with symptoms of MG. Ptosis

should rouse the suspicion of MG in the first place.

This patient was diagnosed of having MG on the basis of her symptoms/signs, the level of anti-cholinesterase receptor antibodies and enlarged thymus gland. Further confirmation was made by giving injection endorphin which resulted in improvement of her symptoms.

The combination of MG and pregnancy has two important aspects, reflection of pregnancy on the neurological illness and the influence of MG on the course of pregnancy and delivery.⁶ In this patient, prior to establishment of diagnosis of MG, her first pregnancy ended up in abortion, may be due to developing antibodies. In one study, 69 cases of MG with pregnancy were studied and results showed 63 term deliveries, 1 abortion and 5 premature births.⁷ Another study showed 64 pregnancies with MG, which resulted in 10 abortions.⁸

The factors involved in the initiation or induction of auto-immune MG are unknown. The possibility of the overlapping MG and some other auto-immune disorders is very common and well-established. Particularly prominent are abnormalities in the thymus. Approximately 15% of patients have a thymoma, and another 60% have thymic hyperplasia.⁷ Thymectomy is a standard procedure for enlarged thymus gland. Removing a major source of antibody production clinically improves MG.⁹

The baby also faces significant risks due to antibodies level of IgG group. The transient NMG signs are lethargy, slow respiration, faint cry, generalized muscular weakness, absence of Moro's reflex and difficulties in sucking and breathing. Newborn of thymectomized mothers showed lower rate of NMG compared to non-thymectomized women.⁷ NMG occurring in 12 – 20% of infants born to myasthenic mothers.¹⁰ In our case, the first baby suffered from TNMG, but after the initiation of medical therapy and thymectomy, all the subsequent newborns remained well.

The first 3 weeks following parturition are especially dangerous for such patients since in one-third of such patients sudden exacerbations may occur. In postpartal period, the patients have to continue with the acetylcholinesterase inhibitor dose, equal to the one taken before pregnancy, if no deterioration is noticed.³ Puerperal infections (endometritis,

mastitis, urinary tract infections) require immediate treatment, otherwise they may provoke severe deterioration of MG.

Anti-cholinesterase medicines as well as nicotinic acetylcholine receptor antibodies are found in mother's milk only in negligible amount. However, nursing is sometimes disrupted to avoid exhaustion of mother which can deteriorate MG.

Close co-operation is required between obstetrician, neurologist, anesthesiologist and neonatologist managing the pregnant women with MG and their newborns. Care should be provided in appropriately equipped institutions with well-trained staff.

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