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Case Report

Myasthenia gravis in pregnancy: a case report

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ABSTRACT

Myasthenia gravis is an autoimmune neuromuscular disorder characterized by weakness and fatigue of skeletal muscles of the face and extremities. It affects the people of both sexes and all ages, but twice as many female patients are affected as male patients. Myasthenia gravis usually strikes women in their third decade of life. Although disease course is variable, pregnant patients face risks of exacerbation, respiratory failure, adverse drug response, crisis and death. Because the severity of symptoms, as well as maternal mortality, is highest in the first 2 years following onset of myasthenia gravis, it is advisable for women to delay pregnancy for at least 2 years following diagnosis. Severity of symptoms and risk of maternal mortality is lowest 7 years after onset of the disease. We report a case of 26-year-old patient with G2P1IUFD1 at 37 weeks of gestation with myasthenia gravis.

Keywords: Myasthenia gravis, Chronic hypertension

INTRODUCTION

Owing to high prevalence of myasthenia gravis (MG) in women of child bearing age, and because it does not affect fertility in women.[1] it is not uncommon to see pregnant women with myasthenia gravis. The effect of pregnancy on myasthenia gravis varies considerably among women and even between pregnancies in the same women.[2] Improvement of symptoms during second and third trimesters has been attributed to normal immunosuppressive changes in late pregnancy.[3] Exacerbations of symptoms are most likely to oc-cur in the first trimester or following delivery.[4]

In general, MG does not have any severe adverse effects on pregnancy.[2] Reports do not suggest an in-creased risk of spontaneous abortions or premature births for women with MG.[4] It is possible for infants to develop transient neonatal MG. this happens in 10% to 20% of cases owing to placental transfer of immu-noglobulin G antibodies in second and third trimesters.[5] The neonate typically develops sucking and pto-sis.[1,3,5] This condition usually reveses itself after 3 weeks owing to dergradation of antibodies.[3]

Mode of delivery

As the uterus is made up of smooth muscle, it is not affected by presence of Ach receptor antibodies and vaginal delivery is recommended for women with MG.[3,5] Cesarean sections should be performed only for obstretric indications, as surgery can be stressful for women with MG.

CASE REPORT

A 26-year-old G2P1IUFD1 married since 5 years, with 2 months of amenorrheoa and with chief complaints of difficulty in swallowing, low volume of speech and dropping of eyelid. Patient was a known case chronic hypertension tablet amlodipine 5 mg OD came to OPD with UPT positive status. Tablet amlodipine was stopped

immediately and tab lobet 100 mg BD and dose increased to 100 mg TDS as BP was not controlled. On further probing the history she gave history of previous term IUFD (in view of pre-eclampsia). After her delivery she was started on antihypertensive. Patient was admitted for further evaluation of hypertension and on subsequent investigations 2D echo, acetylcholine receptor antibodies EMG were done, she was diagnosed as a case of myasthenia gravis for which she was started on tab. Azathrioprine 500 mg OD and tab. Prednisolone 5 mg OD. Patient was maintaining well on the above drugs and was following regularly in OPD. She was admitted again at 36 weeks of gestation for safe confinement. Induction of labour was done at 37 weeks of gestation as now the USG OBS doppler started showing notching of uterine arteries. She delivered vaginally a 2.8 kg male, the delivery was uneventful. Baby was also evaluated for transient neonatal myasthenia gravis, but all investigations were negative. Mother and baby were discharged on day 4 after delivery. Patient was adviced to continue with tab. AZA and tab. Prednisolone in same doses.

DISCUSSION

Myasthenia gravis is not rare among women of reproductive age, the reported incidence ranges from 1:10,000 to 1:50,000.[6] There is evidence, as yet unexplained, that polymorphonuclear leukocyte chemotaxis and adherence functions are depressed beginning in the second trimester and continuing during rest of the pregnancy. It is possible that these depressed leukocyte functions of pregnant women account in part of improvement observed in some autoimmune diseases. It may also explain the increased susceptibility to certain infections.[7] On the other hand it is well known that some diseases could exacerbate during the pregnancy. This has been reported for example in patients with systemic lupus erythematous and myasthenia gravis.[7,8] The clinical course of myasthenia gravis in pregnancy is considered to be unpredictable. It has been reported that: (a) approximately one third of patients remain the same, one third improve, and the remaining one third worsens, (b) the course in one pregnancy does not predict the course in subsequent pregnancies, (c) exacerbations usually occur equally in all three trimesters, (d) therapeutic termination does not demonstrate a consistent benefit in cases of first trimester exacerbation.[9-12]

Figure 1 shows the diagnostic algorithm for myasthenia gravis. We did acetylcholine receptor antibody test for our patient based on her history which turned out to be positive and hence she was diagnosed with myasthenia gravis.

Schlezinger 9 described the course of MG during pregnancy in 22 myasthenic women with a total of 33 pregnancies. He showed that in one third of pregnant woman an exacerbation occurred, whereas two third remained same or remission occurred.

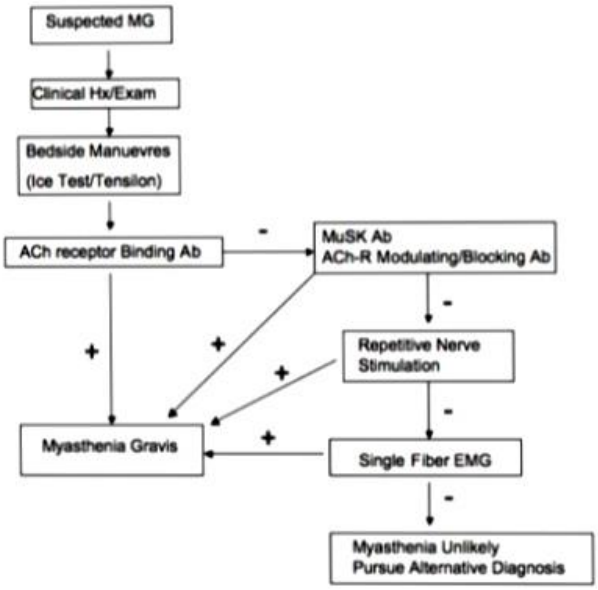

Figure 1: Algorithm for MG.

In a study by, Djelmis et al reported the deterioration was observed in the last 4 weeks of pregnancy and in the puerperium.[11] In another study by Mitchell et al, reported that 27.2% had improvement and 72.7% deteriorated during pregnancy.[14] The deterioration was observed in the third trimester in all patients. They concluded that there were no predictive factors to identify the mother at risk of exacerbation during pregnancy and the risk of neonatal myasthenia gravis.

Batochi et al, reported that 42% had no change, 39% improved and 19% got worse.[15] In the experience of batochi et al the clinical worsening was more frequent in the second trimester and two patients developed

respiratory failure. He concluded that the course of the MG in pregnancy is highly variable and unpredictable and can change in subsequent pregnancies.

Management

Optimal management of MG for pregnant women should involve obstretricians and neurologists. Almost 15% of patients have thymomas and 80-90% present with hyperplastic thymus.[2,16] Thymectomy has become a standard treatment protocol for patients with MG and thymomas or hyperplasia of the thymus.[2,17] During pregnancy, women who have not undergone thymectomy present with higher incidences of exacerbations when compared with those who have undergone thymectomy.[18]

Pharmacologic treatment for MG is usually centred on increasing the levels of Ach and decreasing the production of auto-antibodies.

Acetylcholine esterase inhibitors, such as pyridoxamine and neostigmine, are frequently used in treatment of MG.[2,3,19] There was one case report of microcephaly related to use of pyridoxamine.[19] Some authors suggest that the placental transfer of maternal antibodies, not the pyridoxamine, might have caused the foetal anamolies.[20] Their claim is supported by the studies showing safe use of pyridoxamine in pregnancies.[1,21,23]

Women with MG who are prescribed corticosteroids must be told before that there is increased association with cleft palate or they can be started on steroids after 12 weeks when the formation of palate is complete.[22,24]

Azathrioprine has not been associated with any congenital anamolies but may be related with low birth weight and intrauterine growth retardation.[21,22] Although cyclosporine has been shown to cross the placenta readily, there has been no evidence that it is associated with increased risk of severe complications or malformations.[2]

Rituximab readily crosses the placenta at about 16 weeks of gestation.[22] To date, there have not been any reports of major malformations attributed to rituximab exposure.[23] There were some cases of decreased B-cell counts in infants born to women using rituximab, but this condition resolved within 6 months.[23]

Mycophenolate mofetil(MFM) is a second line drug for treatment of mild forms of MG.[14] It is associated with first trimester miscarriages and structural malformations of the ears and jaw, cleft lip and palate, as well as hypoplastic fingers and toe-nails.[24]

The drugs which are contraindicate in MG and exacerbate the symptoms neuromuscular blocking agent(magnesium sulfate), antiarrhythmic drugs (quinidine), and local anesthetics (esters) as well as antibiotics from aminoglycoside, quinolone and macrolide classes.[25]

CONCLUSION

There is no evidence that MG can adversely affect pregnancy outcomes, and most of the medications used for symptom control appear to be relatively safe (except for MFM used as second line therapy). Pre-pregnancy thymectomy might decrease the need for medications in pregnancy.

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