



Myasthenia Gravis in Pregnancy

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Myasthenia gravis (MG) is estimated to affect about 700,000 people worldwide, and has a higher incidence in women of child-bearing age. The effect of pregnancy on the disease activity of MG is rather unpredictable: in a previous study on women with MG who became pregnant, 41% deteriorated, 29% had no change and the other 31% improved.⁽¹⁾ Deterioration is more likely to occur in the first trimester or after the delivery.

A successful pregnancy outcome is expected when MG symptoms are under control prior to the onset of pregnancy. It is therefore advised for the pregnancy to be planned with the optimal management of MG. A management plan to deal with potential MG complications should be in place; it should involve a multidisciplinary team that consists of specialists in neurology, obstetrics, anesthesiology and neonatology. The treatment of MG has to be modified during the pregnancy to minimize the risk to the fetus. Pyridostigmine is safe in pregnancy and is often the only medication used, usually in milder cases. Steroids (usually prednisone or prednisolone) are the preferred immunosuppressant if MG symptoms are more severe and cannot be controlled by pyridostigmine alone; however, the use of steroids increases the risk of premature rupture of the membranes and may cause deterioration of gestational diabetes and hypertension, in addition to the numerous adverse effects in the non-pregnant population. Therefore the lowest steroid dose which controls the disease activity is to be used. As non-steroidal immunosuppressants may result in abortion or fetal malformations, their use is generally avoided during pregnancy; however, cyclosporine and azathioprine have been used successfully when the use of prednisone is not possible because of such medical conditions as diabetes. The safety of azathioprine in pregnancy is controversial, however.⁽²⁾ Methotrexate and mycophenolate should never be used during pregnancy. Intravenous immunoglobulin (IVIG) appears to be well tolerated

and can safely be used during pregnancy for MG exacerbations. Plasmapheresis is also rather safe, but given the more invasive nature, it is generally reserved for patients with severe symptoms including respiratory insufficiency. As the therapeutic effect of thymectomy is delayed, it should be deferred in the pregnant women.

Vaginal delivery with regional anesthesia is preferred in pregnant women with MG, as surgery and anesthesia may predispose to subsequent MG exacerbation. If cesarean section is selected, the use of neuromuscular blockers better be avoided during anesthesia, and the mother should be closely followed in the recovery room and the oncoming few days for symptoms of MG exacerbation. As magnesium is a neuromuscular blocker, its use in the treatment of pre-eclampsia and eclampsia is to be avoided.



All infants of myasthenic mothers should be observed for symptoms of neonatal MG in the first 48–72 hours of life. Neonatal MG is a transient form of myasthenia caused by transfer of mother's autoantibodies to the fetus, and can occur even when the symptoms are mild in the mother. Treatment of neonatal MG consists of supportive care, pyridostigmine and, when needed, plasmapheresis. The condition resolves spontaneously.

References

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