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Case Report

Obstetrics and Gynaecology Section

Successful Maternal and Neonatal Outcomes in a Myasthenia Gravis Patient Presenting in Early Pregnancy: A Case Report

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ABSTRACT

Myasthenia Gravis (MG) is a chronic autoimmune neuromuscular disorder that poses significant challenges during pregnancy due to its variable disease course and potential neonatal complications. Authors hereby, report a case of a 25-year-old primigravida with known MG on immunosuppressive therapy who presented at 8 weeks gestation. Her antenatal period was complicated by worsening respiratory symptoms during the third trimester. She underwent an emergency caesarean section at term due to foetal distress. The neonate, though initially stable, developed respiratory distress and was found to have maternal antibodies, requiring treatment with pyridostigmine. The present case highlights the importance of multidisciplinary care and preparedness for neonatal myasthenia in pregnancies complicated by maternal MG.

Keywords: Autoimmune neuromuscular disorder, Foetal distress, Pyridostigmine, Anticholinesterase therapy

CASE REPORT

A 25-year-old primigravida with a 4-year history of Myasthenia Gravis (MG) who presented at four weeks' gestation for her first antenatal visit. She was diagnosed with generalised MG at the age of 21years and was being treated with azathioprine 50 mg BID, prednisolone 10 mg OD, and pyridostigmine 60 mg BID. The prepregnancy course of the mother was stable with no exacerbation. Mother was positive for Anti-Acetylcholine Receptor (Anti-AChR), Anti-Muscle-Specific Kinase (Anti-MuSK) and Anti-Low-Density Lipoprotein Receptor-Related Protein 4 (Anti-LRP4). There was no similar family history for MG.

She was regularly followed-up throughout pregnancy, with routine antenatal investigations performed as advised. After a multidisciplinary consultation involving neurology and obstetrics, her prepregnancy medications were continued. The pyridostigmine dose was increased to 60 mg TDS in the second trimester as pregnancy increases drug elimination.

As pregnancy progressed, she began experiencing progressive shortness of breath, for which she was started on budesonide (Budecort) nebulisation, providing symptomatic relief.

At 37 weeks of gestation, she presented with decreased foetal movements for two days. On examination, she was haemodynamically stable, maintaining a SpO2 of 98% on room air. Uterus was relaxed and corresponded to term gestation. Foetal Heart Rate (FHR) was 180 bpm, auscultated in the left spinoumbilical line. A non stress test confirmed foetal tachycardia, raising concern for foetal distress.

An emergency Lower-Segment Cesarean Section (LSCS) was performed, delivering a female infant weighing 2.4 kg. LSCS was done under spinal anaesthesia, while ensuring there was no sudden hypotension and with close monitoring, especially for respiratory compromise. Muscle relaxants and high spinal block were avoided. The newborn was transferred to the Neonatal Intensive Care Unit (NICU) due to respiratory distress. No hypotonia was observed at birth. The mother's respiratory complaints improved gradually over 2-3 days postoperatively, and she was continued on Budecort and Duolin nebulisation.

The neonate tested positive for anti-acetylcholine receptor antibodies, suggestive of Transient Neonatal Myasthenia Gravis (TNMG), and was started on pyridostigmine 0.5 mg/kg 8 hourly, with subsequent clinical improvement.

On day 4 of life, the newborn was shifted to the ward from NICU as there were no signs of weak cry, respiratory distress, and the neonate was tolerating feeds well. Mother and infant were discharged on postoperative day 8, and pyridostigmine was gradually tapered over three to four weeks. At discharge, the mother had no symptoms, and nebulisation was stopped. Postpartum, the patient was advised to continue pyridostigmine, azathioprine, and prednisolone as per Neurology guidance.

She was counselled regarding early recognition of warning signs, such as ptosis or breathlessness. Breastfeeding was continued, with instructions to report if the baby develops hypotonia or a weak cry. The newborn demonstrated good developmental milestones at follow-up which was eight months after birth.

DISCUSSION

Myasthenia Gravis (MG) is a chronic autoimmune condition that affects the neuromuscular junction, leading to fluctuating muscle weakness. The condition is relatively rare, with prevalence rates estimated between 150 and 200 cases per million population, with a consistent upward trend observed over the past five decades [1,2]. Notably, nearly two-thirds of those affected are women, with the disease often manifesting during their reproductive years—typically in the second and third decades of life [3]. As a result, MG can have significant implications for both pregnancy and perinatal outcomes. MG is primarily caused by autoantibodies directed against Acetylcholine Receptors (AChR) or Muscle-Specific Kinase (MuSK) at the neuromuscular junction, impairing signal transmission and leading to muscle fatigue. These pathogenic antibodies reduce the number or functionality of postsynaptic AChRs, thereby inhibiting effective muscle contraction [4,5].

Maternal Course and Management

The course of MG during pregnancy is highly variable. Studies have reported that 30-45% of pregnant MG patients experience exacerbations, most commonly during the first trimester or postpartum period [6,7]. In contrast, our patient remained stable

until the third trimester, when progressive respiratory symptoms began to develop. Although respiratory muscle weakness is a known complication in MG, it may be exacerbated in late pregnancy due to increased abdominal pressure and reduced diaphragmatic excursion [8].

Immunosuppressive therapy in pregnancy must be cautiously optimised. Azathioprine and prednisolone are classified as relatively safe in pregnancy. Azathioprine crosses the placenta but is generally not associated with significant teratogenicity when used in standard doses. Pyridostigmine, the cornerstone of symptomatic MG management, is also considered safe and was continued at therapeutic doses. Our management approach aligns with consensus guidelines that recommend continuation of effective MG therapy during pregnancy to prevent exacerbations [8,9].

Mode of Delivery

There is no absolute contraindication to vaginal delivery in MG unless obstetric indications arise. However, operative delivery rates remain high in MG patients due to maternal fatigue, poor pushing efforts, or foetal compromise [10]. In this case, emergency LSCS was warranted due to foetal tachycardia, suggestive of intrauterine distress. The maternal respiratory status, though not in crisis, necessitated close anaesthetic and postoperative monitoring.

Neonatal Considerations

Approximately 10-20% of neonates born to mothers with MG develop TNMG due to transplacental transfer of pathogenic antibodies [11]. TNMG typically presents within the first few hours to days of life with hypotonia, weak cry, poor suck, and respiratory difficulty. In present case, although no hypotonia was noted, the newborn presented with respiratory distress and was positive for maternal antibodies, necessitating treatment with pyridostigmine, consistent with TNMG. The severity and presence of TNMG symptoms often correlate with maternal antibody titers, especially anti-AChR levels, although this relationship is not absolute. Higher titers have been associated with an increased risk of TNMG. Still, some neonates remain asymptomatic despite significant maternal antibody levels, possibly due to differences in placental transfer efficiency or neonatal receptor sensitivity [11,12].

A similar pattern was reported by Santiago Gonçalves C et al., where infants of MG mothers presented with mild TNMG symptoms, responsive to AcH therapy [13]. However, Ristovska S et al., have documented a case requiring mechanical ventilation [14]. In current case, non invasive NICU support and pharmacological treatment were sufficient, indicating a relatively mild neonatal course.

Long-term Follow-up Protocols: Newborns diagnosed with TNMG generally have a favourable prognosis, as symptoms resolve with the clearance of maternal antibodies, usually within 1-8 weeks. However, long-term follow-up is recommended to monitor neurodevelopmental milestones and to differentiate TNMG from Congenital Myasthenia Syndromes (CMS), which may present similarly but require different management [15,16]. Periodic evaluation by a paediatric neurologist, including developmental assessments and electromyography if warranted, is advisable. Postpartum follow-up for mothers is equally essential, especially during the first 6-8 weeks, due to the high risk of disease exacerbation [17].

Comparison with Other Reported Cases

Several case reports in the literature have highlighted the management and outcomes of pregnancy in women with MG. Baduni N et al., reported a 28-year-old known MG patient who underwent LSCS for obstetric indications. She developed postoperative complications, including muscular weakness, ptosis, dysarthria, and dyspnoea by the third postoperative day, requiring an increase in her pyridostigmine dose (120 mg thrice daily). The neonate in this case developed transient neonatal MG, which resolved spontaneously without pharmacologic intervention [18]. In contrast, Sikka P et al., described a 25-year-old primigravida with MG who also underwent LSCS for obstetric reasons but was complicated by severe preeclampsia. She was managed with neostigmine injections in addition to oral pyridostigmine and steroids. Notably, no features of neonatal MG were observed in present case [19]. Similarly, Berlit S et al., presented a 38-year-old MG patient who had an emergency LSCS for obstetric indications and required only routine oral pyridostigmine and steroids; the infant did not exhibit any signs of MG [20].

However, unlike the cases reported by Sikka P et al., [19] and Berlit S et al., [20] where neonatal MG was absent, our patient's infant developed antibody-positive respiratory distress requiring pyridostigmine therapy, similar to the transient neonatal MG observed by Baduni N et al., [18]. These findings collectively emphasise the variability in both maternal and neonatal outcomes, even with similar management approaches, and reinforce the need for individualised care and neonatal preparedness in pregnancies complicated by MG.

CONCLUSION(S)

The present underscores the complexities of managing pregnancy in women with Myasthenia Gravis (MG) and highlights the importance of a multidisciplinary approach involving obstetricians, neurologists, and neonatologists. Continuation of stable immunosuppressive and symptomatic therapy during pregnancy, close maternal and foetal surveillance, and readiness for neonatal complications are critical to optimising outcomes. Although the mother experienced mild respiratory compromise in the third trimester, prompt intervention and careful monitoring ensured maternal stability. The development of transient neonatal MG in the newborn, despite the absence of hypotonia at birth, emphasises the unpredictable nature of antibody transmission and neonatal response.

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