

CASE REPORT

Spinal Anesthesia With Staged Airway Contingency for Emergency Cesarean Delivery in a Parturient With Myasthenia Gravis, Ankylosing Spondylitis, and Multiple Sclerosis: An Observational Analytic Study

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ABSTRACT

Background. Emergency preterm cesarean delivery in a parturient with myasthenia gravis, ankylosing spondylitis, and multiple sclerosis combines neuromuscular weakness, a rigid spine with a possibly difficult airway, and demyelinating disease. Magnesium sulfate for fetal neuroprotection can worsen myasthenic weakness.

Objective. To describe the anesthetic plan and the outcome in one such patient.

Methods. A 34-year-old multiparous woman at 30 weeks + 2 days presented about 8 hours after premature rupture of membranes, with active ocular and bulbar symptoms. After a multidisciplinary consensus meeting she received single-shot spinal anesthesia (0.5% hyperbaric bupivacaine 10 mg with fentanyl 25 mcg, L4-L5, 27-gauge Quincke needle). A staged airway contingency plan with rocuronium and sugammadex was prepared and not used. Magnesium sulfate was given under neuromuscular surveillance.

Results. The sensory block reached T4. Mean arterial pressure stayed between 82.0 and 90.0 mmHg (largest fall, -8.9%), and no vasopressor was required. One transient rise in respiratory rate to 26 breaths/min occurred at delivery and resolved with face-mask oxygen. A preterm girl (1,415 g) had Apgar scores of 5, 7, and 8, and her Downes score fell from 5 to 1 within 24 hours on nasal continuous positive airway pressure. The mother had no myasthenic crisis during 24 hours of intensive care and moved to the ward on postoperative day 1.

Conclusion. In this patient, low-dose spinal anesthesia with a prepared airway contingency and monitored magnesium sulfate was followed by an uneventful maternal course. A single case cannot show efficacy, and follow-up was short.

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1. Introduction

Pregnancy in women with autoimmune disease forces clinicians to weigh the physiological changes of gestation against the activity of the underlying disease. In myasthenia gravis (MG), a pooled analysis of pregnancies found worsening of myasthenic symptoms in about one third. A systematic review reported myasthenic crisis in 6.4% of pregnancies and 8.2% of postpartum periods.^{1,2} Ankylosing spondylitis (AS) and multiple sclerosis (MS) each bring their own constraints to obstetric anesthesia. MG and MS are known to co-occur more often than expected by chance, but we found no published report that combines MG, AS, and MS in one parturient apart from the authors' own.³

MG is an antibody-mediated disease. Autoantibodies, mainly against the postsynaptic acetylcholine receptor or muscle-specific tyrosine kinase, reduce the safety margin of neuromuscular transmission and cause fatigable weakness.^{4,5} Pregnancy brings plasma volume expansion, altered drug pharmacokinetics, emotional stress, and

hormonal shifts, any of which may provoke exacerbations. Labor and operative delivery add a further threat, because fatigue of the diaphragm and intercostal muscles endangers spontaneous breathing. Response to neuromuscular blocking agents is unpredictable. The risk of drug-induced exacerbation also narrows the choice of anesthetic agents.⁶

AS adds mechanical restrictions. Chronic inflammation, driven by interleukin-23 and interleukin-17 signaling, is followed by new bone formation and, eventually, syndesmophytic bridging that stiffens the spine.^{7,8} Syndesmophyte formation and progression have been linked to sustained disease activity.⁹ For anesthesia the consequences are twofold. First, narrowing of the interspinous spaces and calcification of the ligamentum flavum can obstruct a midline subarachnoid needle, and second, restricted cervical mobility and thoracic rigidity make a direct view of the glottis difficult. Conversion from regional to general anesthesia may then lead to a cannot-intubate, cannot-oxygenate situation.^{10,11}

MS is an autoimmune demyelinating disease of the central nervous system. It is characterized by autoreactive lymphocytic infiltration, microglial activation, and neuroaxonal loss. Anesthesiologists have long been wary of neuraxial techniques in MS. They feared that local anesthetic acting on demyelinated axons could cause neurotoxic injury or neurological deterioration. A systematic review of observational evidence suggests that neuraxial anesthesia can be given safely in women with MS, and a cohort of 118 deliveries found no association between neuraxial procedures and postpartum relapse.^{12,13} Other risks remain. Autonomic vasomotor dysfunction and temperature-dependent conduction block are recognized in MS, and the relapse rate is higher in the first 3 months after delivery.¹⁴ Rapid withdrawal of steroids and estrogen has been proposed as one trigger.

The three conditions meet at their sharpest in an obstetric emergency such as preterm premature rupture of membranes, where delivery may not be deferred.¹⁵ Drug interactions then matter. Magnesium sulfate for fetal neuroprotection is the clearest example, since given before 32 weeks of gestation it reduces cerebral palsy in the infant.¹⁶ Yet it also blocks presynaptic calcium entry and inhibits acetylcholine release at the neuromuscular junction. In MG it has been described as contraindicated and has been followed by acute respiratory failure.^{17,18} A chart review also reported exacerbation of MG after intravenous magnesium.^{19,20}

We found no published description of the anesthetic management of this triple combination during emergency cesarean delivery. The objective of the present case report is to describe the anesthetic plan used in this patient. This covers the choice of spinal anesthesia, the staged airway contingency, and the handling of magnesium sulfate. We also report the maternal and neonatal outcome.

2. Methods

2.1. Report design and reporting guideline

This is a report of a single patient and follows the CAsE REport (CARE) guideline.²¹ No comparison group was

studied. No hypothesis testing was planned. The report describes the anesthetic plan used in this patient and what followed, including maternal hemodynamic stability, ventilation, neuromuscular status, and neonatal condition. Observation began at presentation (hour 0) at 30 weeks of gestation. It continued through emergency operative delivery and intensive care monitoring until transfer to the ward on postoperative day 1.

2.2. Setting

The patient was managed in the Emergency Obstetric Surgical Suite and the Intensive Care Unit (ICU) of Dr. Moewardi Regional General Hospital, Surakarta, Indonesia. The hospital is a tertiary academic referral center affiliated with Universitas Sebelas Maret.

2.3. Patient information and case selection

The patient is a 34-year-old multiparous woman (gravida 3, para 2, abortus 0). She presented at 30 weeks and 2 days of gestation with spontaneous premature rupture of membranes (PROM) and active neurological symptoms. Specialist care had followed her 5-year history of MG, AS, and MS. Her maintenance medication was pyridostigmine 60 mg five times daily, methylprednisolone 8 mg twice daily, and adalimumab 40 mg subcutaneously every 2 weeks. We selected her for reporting because she had all three confirmed autoimmune diseases together with a high-risk preterm pregnancy that required emergency operative delivery.

Weight-based doses such as rocuronium and sugammadex can only be interpreted with the patient weight and height.

2.4. Case timeline (Table 1)

Table 1 gives the clinical timeline in relative time. Presentation to the hospital is hour 0 of the clinical course. For the intraoperative record, T0 denotes the pre-spinal baseline. Later epochs are labeled by the minutes elapsed since T0 (T+15 to T+120). The report keeps the two clocks apart throughout.

Table 1. Clinical timeline of the patient in relative time (hour 0 = presentation; T0 = pre-spinal baseline).

Time point	Clinical event	Investigation and result	Intervention
About 8 h before presentation	Spontaneous premature rupture of membranes at 30 weeks + 2 days of gestation	Not reported	None reported
Hour 0 (presentation)	Emergency obstetric triage with irregular painful contractions, reduced fetal movements, ptosis with diplopia, bulbar weakness, and limb weakness (MRC 4/5)	BP 128/76 mmHg (MAP 93.3), HR 88/min, RR 20/min, temperature 36.0 °C, SpO ₂ 98% on ambient air; Hb 12.4 g/dL, platelets 245,000/ μ L, INR 1.02, creatinine 0.68 mg/dL; ASA IV-E	Admission to emergency obstetric care
Hour 0 onward (stabilization)	Maternal optimization, tocolysis, and fetal lung maturation during threatened preterm birth	Capillary glucose checks for corticosteroid effect	Balanced crystalloid 100 mL/h; dexamethasone 5 mg IM every 12 h (4 doses); ampicillin 1 g IV every 8 h; nifedipine 20 mg then 10 mg three times daily; pyridostigmine and methylprednisolone continued; hydrocortisone 100 mg IV prepared; adalimumab withheld
Before delivery	Fetal neuroprotection started	Neuromuscular function monitored at the bedside	IV magnesium sulfate 4 g over 20 min, then 1 g/h for 24 h
Consensus meeting	Cervical dilation progressing, irregular contractions, persistent fetal heart rate decelerations on cardiotocography	Multidisciplinary decision: emergency cesarean delivery	Plan A (spinal anesthesia) chosen; Plan B (airway rescue with rocuronium and sugammadex) prepared
T0 (pre-spinal baseline)	Patient sitting for spinal puncture	BP 138/66 mmHg (MAP 90.0), HR 76/min, RR 20/min, SpO ₂ 98%	L4-L5 puncture, 27-gauge Quincke needle, single pass, no ultrasound; 0.5% hyperbaric bupivacaine 10 mg + fentanyl 25 mcg
T+15 min	Block onset with warm sensation in both legs	Sensory level T6, Bromage 2; BP 123/68 mmHg (MAP 86.3), HR 71/min	None

Time point	Clinical event	Investigation and result	Intervention
T+30 min	Incision and delivery of a preterm girl (1,415 g); transient tachypnea	Sensory level T4, Bromage 3; RR 26/min, SpO ₂ 98%, MAP 82.0 mmHg; infant Apgar 5 at 1 min	Face-mask oxygen 4 L/min; infant dried, warmed, and started on nasal CPAP (FiO ₂ 0.30); oxytocin infusion after extraction
T+45 to T+75 min	Placental delivery, myometrial closure, and abdominal closure	RR 24/min at T+45 and 20/min from T+60; sensory level T4, Bromage 3; blood loss about 450 mL; infant Apgar 7 at 5 min and 8 at 10 min	Oxytocin 10 IU in 500 mL at 125 mL/h; no vasopressor given
T+90 to T+105 min	Skin closure after 48 min of surgery; sensory regression	T+90: T6, Bromage 2; T+105: T8, Bromage 1; temperature 36.2 °C	Transfer to post-anesthesia care unit
T+120 min	ICU admission	Sensory level T10, Bromage 1; SBP 115 mmHg, RR 20/min, SpO ₂ 97%	24 h of ICU surveillance (forced vital capacity, negative inspiratory force, neurological examination); pyridostigmine and methylprednisolone resumed; magnesium sulfate infusion completed at 24 h
Postoperative day 1	Transfer to the obstetric-neurology step-down ward	Muscle strength at baseline, bulbar function intact, no MS relapse, no surgical site infection; infant Downes score 5 to 1 within 24 h	Nasal CPAP in the neonatal ICU (PEEP 6 cmH ₂ O, FiO ₂ 0.30)

Notes: Hour 0 is the time of presentation; T0 is the baseline immediately before spinal anesthesia, and T+n is n minutes after T0. ASA, American Society of Anesthesiologists physical status; BP, blood pressure; CPAP, continuous positive airway pressure; FiO₂, fraction of inspired oxygen; Hb, hemoglobin; HR, heart rate; ICU, intensive care unit; IM, intramuscular; INR, international normalized ratio; IV, intravenous; MAP, mean arterial pressure; MRC, Medical Research Council muscle strength scale; MS, multiple sclerosis; PEEP, positive end-expiratory pressure; RR, respiratory rate; SpO₂, peripheral oxygen saturation.

2.5. Clinical findings and diagnostic assessment

On presentation, emergency obstetric triage assessed the patient with vital signs, a neurological examination, and an obstetric assessment. Her physical status was classified as American Society of Anesthesiologists (ASA) class IV-E.

Table 2. Demographic, obstetric, and baseline findings, with diagnostic ascertainment of the three autoimmune conditions.

Domain / parameter	Finding	Diagnostic ascertainment and clinical relevance
Maternal demographics		
Age	34 years	Reproductive age; MG is more frequent in women (ratio 2:1) and MS in women (ratio 3:1)
Parity and obstetric history	Multiparous (G3P2A0)	Previous uneventful pregnancies before onset of the three autoimmune conditions
Gestational age	30 weeks + 2 days	Preterm gestation; threatened preterm delivery, fetal neuroprotection indicated
Presenting obstetric event	PROM about 8 h before presentation	Membrane rupture with irregular uterine contractions and reduced fetal movements
ASA physical status	ASA class IV-E (emergency)	High-risk parturient with three coexisting autoimmune disorders
Diagnostic ascertainment of the three conditions		
Myasthenia gravis (MG; 5-year history)	Anti-AChR antibody positive (reported as >8.5 nmol/L); EMG decremental response (reported as >15%)	Postsynaptic AChR-mediated neuromuscular transmission failure; Osserman class IIb; active ocular, bulbar, and respiratory signs
Ankylosing spondylitis (AS; 5-year history)	HLA-B27 positive; bilateral sacroiliitis (grade III); spinal syndesmophytes	Axial inflammatory osteitis; ligamentum flavum calcification; restricted cervical extension (35°)
Multiple sclerosis (MS; 5-year history)	Relapsing-remitting course; CSF oligoclonal bands present; periventricular plaques on MRI	Central demyelinating disease; relevant to neuraxial local anesthetic exposure and to postpartum relapse risk
Baseline physiological parameters at admission		
Blood pressure (BP)	128/76 mmHg (MAP 93.3 mmHg)	Normotensive at admission
Heart rate (HR)	88 beats/min, regular	Regular rhythm
Respiratory rate (RR)	20 breaths/min, eupneic	Normal resting rate; bulbar dysphonia and mild dyspnea reported
Core body temperature	36.0 °C	Afebrile; heat-related worsening of demyelinating symptoms (Uhthoff phenomenon) not expected
Peripheral oxygen saturation (SpO ₂)	98% on ambient air	Adequate resting gas exchange before neuraxial block
Laboratory panel		
Hematology (Hb, WBC, platelets)	Hb 12.4 g/dL; WBC 9,800/μL; platelets 245,000/μL	No anemia, leukocytosis, or thrombocytopenia
Coagulation (PT, aPTT, INR)	PT 11.8 s (ref 10-14); aPTT 29.4 s (ref 25-35); INR 1.02	Normal hemostatic profile; no laboratory contraindication to spinal puncture
Renal and electrolytes	BUN 12 mg/dL; Cr 0.68 mg/dL; Na 138 mEq/L; K 4.1 mEq/L; Cl 102 mEq/L	Normal renal function and electrolytes (serum potassium relevant to neuromuscular function)
Hepatic and metabolic	AST 22 U/L; ALT 19 U/L; random blood glucose 104 mg/dL	Normal liver enzymes; normoglycemia during corticosteroid therapy

Notes: Values are specific clinical measurements and qualitative findings. AChR, acetylcholine receptor; ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time; ASA, American Society of Anesthesiologists; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CSF, cerebrospinal fluid; Cr, creatinine; EMG, electromyography; G3P2A0, gravida 3, para 2, abortus 0; Hb, hemoglobin; HLA-B27, human leukocyte antigen B27; INR, international normalized ratio; MAP, mean arterial pressure; MRI, magnetic resonance imaging; PROM, premature rupture of membranes; PT, prothrombin time; WBC, white blood cells.

MG had been diagnosed with elevated serum anti-acetylcholine receptor antibodies and a decremental response on repetitive nerve stimulation electromyography. It was classified as Osserman class IIb. The reported thresholds were an antibody titer above 8.5 nmol/L and a decrement above 15%. AS had been confirmed by human leukocyte antigen B27 positivity, radiographic bilateral grade III sacroiliitis, and syndesmophytes. MS had been diagnosed according to the 2017 McDonald criteria. The course was relapsing-remitting, oligoclonal bands in the cerebrospinal fluid were positive, and magnetic resonance imaging of the brain showed periventricular and spinal demyelinating lesions. Before the procedure the airway was assessed, and cervical extension and sternal distance were the recorded measurements.

2.6. Therapeutic intervention

A multidisciplinary consensus meeting of maternal-fetal medicine obstetrics, anesthesiology, neurology, internal medicine, and neonatology determined the plan.²² Two

Table 3. Chronic maintenance immunotherapy and peripartum drug regimen, with disease-specific considerations.

Agent	Dose and route	Target condition and mechanism	Peripartum considerations
Pyridostigmine bromide	60 mg orally 5 times daily (300 mg/day)	MG: reversible inhibition of acetylcholinesterase, prolonging acetylcholine action at the neuromuscular junction	Continued without interruption up to the immediate preoperative period; oral route; vigilance for cholinergic crisis versus myasthenic weakness
Methylprednisolone	8 mg orally twice daily (16 mg/day)	MG, AS, and MS: broad suppression of T-cell activation, cytokine transcription, and antibody synthesis	Continued; stress-dose hydrocortisone 100 mg IV prepared; serial glucose monitoring; infection risk considered with ruptured membranes
Adalimumab	40 mg subcutaneously every 2 weeks	AS: recombinant human IgG1 anti-TNF- α monoclonal antibody that reduces axial inflammation	Withheld in the setting of ruptured membranes (about 8 h at presentation) and threatened preterm delivery, to limit the risk of maternal-neonatal infection
Magnesium sulfate (MgSO ₄)	4 g IV loading over 20 min, then 1 g/h IV, planned for 24 h	Fetal neuroprotection before preterm birth (proposed mechanism: NMDA receptor blockade)	Clinical consideration in MG: presynaptic Ca ²⁺ antagonism may reduce acetylcholine release and has been reported to precipitate myasthenic exacerbation (see Table 6); given with bedside airway and reversal readiness
Dexamethasone sodium phosphate	5 mg IM every 12 h for 4 doses (48 h)	Fetal lung maturation: stimulation of type II pneumocyte surfactant synthesis and secretion	Maternal blood glucose followed closely because of the additive hyperglycemic effect with background methylprednisolone
Nifedipine	20 mg orally as loading dose, then 10 mg orally 3 times daily	Tocolysis: L-type calcium channel blocker reducing myometrial contractility	Given to suppress contractions while antenatal steroids took effect; additive vasodilation and hypotension considered during subsequent sympathetic blockade
Ampicillin sodium	1 g IV every 8 h	Infection prophylaxis: penicillin-class bactericidal cell-wall synthesis inhibitor active against GBS and ascending flora	Prophylaxis after PROM; chosen instead of aminoglycosides, macrolides, and fluoroquinolones, which can impair neuromuscular transmission
Intravenous hydration	Ringer's lactate : 5% dextrose : 0.9% NaCl = 1:2:1	Maintenance of intravascular volume, normoglycemia, and electrolyte balance	Balanced crystalloid-dextrose hydration with attention to fluid volume to limit the risk of pulmonary edema during corticosteroid and tocolytic therapy

Notes: Ca²⁺, calcium ion; GBS, group B streptococcus; IgG1, immunoglobulin G1; IM, intramuscular; IV, intravenous; MG, myasthenia gravis; MS, multiple sclerosis; AS, ankylosing spondylitis; NaCl, sodium chloride; NMDA, N-methyl-D-aspartate; PROM, premature rupture of membranes; TNF- α , tumor necrosis factor alpha.

2.7. Outcome measures and follow-up

Monitoring was continuous. It used five-lead electrocardiography, non-invasive oscillometric arterial blood pressure, pulse oximetry (SpO₂), and infrared capnography. Sensory block height was mapped bilaterally by loss of pinprick sensation. Motor block was graded with the modified Bromage scale. Values were recorded at nine intraoperative epochs: baseline before spinal anesthesia (T0), block onset (T+15 min), incision and delivery of the infant (T+30), placental delivery with uterine exteriorization and oxytocin infusion (T+45), myometrial closure (T+60), peritoneal and fascial closure (T+75), skin closure with transfer to the post-anesthesia care unit (T+90), sensory regression (T+105), and transfer to the ICU (T+120).

pathways were prepared. Plan A was single-shot spinal anesthesia at the L4-L5 interspace with 0.5% hyperbaric bupivacaine 10 mg and fentanyl 25 mcg, using a 27-gauge Quincke needle. The aim was to keep the mass of local anesthetic small.²³ Neuromuscular blocking agents were avoided in Plan A. Plan B was a difficult-airway rescue set, prepared and not used. It comprised video laryngoscopy, flexible fiberoptic bronchoscopy, gum elastic bougies, and second-generation supraglottic airway devices,²⁴ together with rocuronium at a reduced dose of 0.3 mg/kg and sugammadex 4 to 16 mg/kg at the bedside. Sugammadex reverses rocuronium by encapsulating it. Observational data in MG have not shown excess respiratory complications after rocuronium reversed with sugammadex.^{25,26}

Fetal neuroprotection was given as intravenous magnesium sulfate, 4 g as a loading dose followed by 1 g/h, under close neuromuscular surveillance. Table 3 lists the maintenance drugs and the peripartum drugs. It also gives their doses and the precautions applied.

The maternal outcomes were mean arterial pressure, systolic and diastolic pressure, heart rate, respiratory rate, SpO₂, block height, motor block, and any adverse event. After surgery the mother stayed in the ICU for 24 hours. Serial forced vital capacity, negative inspiratory force, and neurological examinations looked for delayed myasthenic decompensation. The neonatal outcomes were birth weight, length, head circumference, Apgar score at 1, 5, and 10 minutes, and the Downes respiratory distress score.²⁷

With one patient, no inferential testing (p values, t tests, analysis of variance, or chi-square tests) was applied. The data are described only. Percentage change from baseline was calculated at each epoch as [(Tn - T0) / T0] × 100%. Mean arterial pressure was calculated as (systolic + 2 × diastolic) / 3.

2.8. Ethics and consent

This study was reviewed and approved by CMHC Research Center, Indonesia (Reference number 2026/0971). Care followed the principles of the Declaration of Helsinki and its later amendments. The report follows the Committee on Publication Ethics (COPE) standards. The patient gave written informed consent for treatment, and she and her legal guardian gave consent for publication. The approval number and the consent statements are given in the Declarations.

3. Results

3.1. Presentation and baseline findings

A 34-year-old multiparous woman (gravida 3, para 2, abortus 0) presented to emergency obstetric triage at 30 weeks and 2 days of gestation. She reported leakage of amniotic fluid about 8 hours earlier, irregular painful uterine contractions, and a subjective decrease in fetal movements. Her physical status was classified as ASA class IV-E. She had a 5-year history of MG, AS, and MS, and immunological, neurophysiological, and radiological testing had confirmed each one (Table 2).

On admission her hemodynamics were stable: arterial blood pressure was 128/76 mmHg (mean arterial pressure [MAP] 93.3 mmHg), heart rate (HR) was a regular 88

beats/min, respiratory rate (RR) was 20 breaths/min, and core temperature was 36.0 °C. Hematological, coagulation, renal, hepatic, and electrolyte results were within normal limits for gestation (hemoglobin 12.4 g/dL, white blood cells 9,800/ μ L, platelets 245,000/ μ L, prothrombin time 11.8 s, activated partial thromboplastin time 29.4 s, international normalized ratio 1.02, serum creatinine 0.68 mg/dL, and sodium 138 mEq/L). The full panel is given in Table 2. These results showed no coagulopathy, no leukocytosis, and no laboratory contraindication to neuraxial puncture.

Besides the obstetric presentation, she had active neurological deficits attributable to her three autoimmune conditions. Examination showed bilateral asymmetric ptosis and horizontal diplopia that worsened on sustained upward gaze. Bulbar involvement consisted of moderate liquid dysphagia, dysarthric speech, and nasal dysphonia. Proximal limb weakness was fatigable and graded 4/5 on the Medical Research Council scale in all four limbs. Advanced AS accounted for axial stiffness and marked restriction of cervical mobility (extension limited to 35°, sternomental distance 11.5 cm). Mild bilateral lower-limb paresthesias were attributed to earlier MS demyelination. Figure 1 shows the peripartum clinical timeline, from emergency triage through stabilization, operative delivery, and postoperative intensive care.

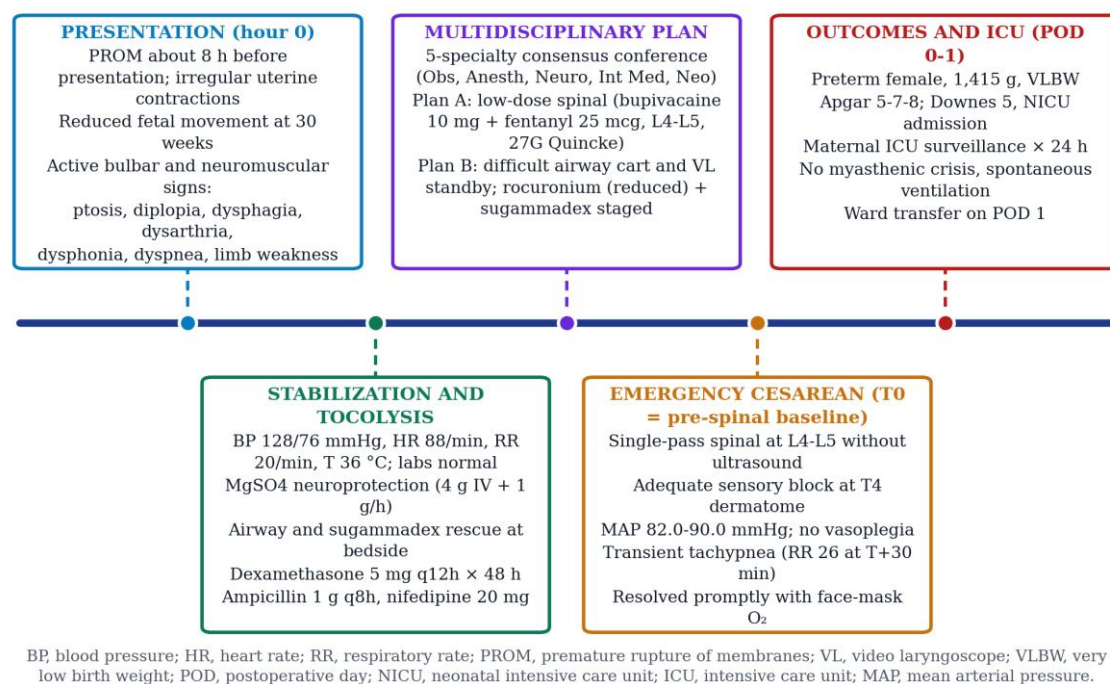


Figure 1. Peripartum clinical timeline. Sequence from emergency presentation with PROM and bulbar symptoms (hour 0), through pharmacological stabilization and tocolysis and the multidisciplinary decision, to single-pass spinal anesthesia and cesarean delivery and recovery in intensive care. Intraoperative times follow Table 4 (T0 = pre-spinal baseline; delivery at T+30 min); the interval between presentation and T0 is not reported. PROM, premature rupture of membranes.

Table 3 summarizes her chronic drug regimen and the peripartum considerations for each agent. Maintenance therapy had three parts: oral pyridostigmine bromide 60 mg five times daily (300 mg/24 h) to support neuromuscular transmission; oral methylprednisolone 8 mg twice daily (16 mg/24 h) as background immunosuppression for all three conditions; and subcutaneous adalimumab 40 mg every 2 weeks for axial spondyloarthritis. Pyridostigmine was continued up to the immediate preoperative period under neurological

supervision, to reduce the risk of both cholinergic crisis and myasthenic relapse. Methylprednisolone was also continued, and intravenous stress-dose hydrocortisone (100 mg) was prepared at the bedside in case of acute adrenal insufficiency during surgical stress. Adalimumab was withheld because the membranes had ruptured about 8 hours before presentation and preterm labor was threatened, since continuing it could add to the risk of maternal-fetal infection. The intraoperative course that followed is plotted in Figure 2 and described in Section 3.3.

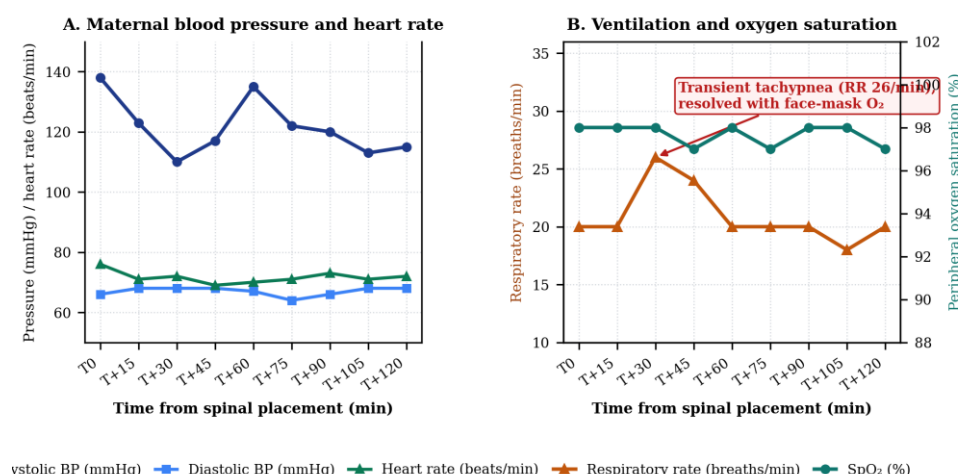


Figure 2. Serial intraoperative measurements during spinal anesthesia for emergency cesarean delivery. (A) Systolic and diastolic blood pressure and heart rate at nine epochs (T0 = pre-spinal baseline, then T+15 to T+120 min). (B) Respiratory rate and peripheral oxygen saturation (SpO₂) at the same epochs; the rise in respiratory rate at T+30 min resolved after face-mask oxygen. Values are those listed in Table 4.

3.2. Diagnostic findings

The diagnostic basis for each autoimmune condition is listed in Table 2. For MG, anti-acetylcholine receptor (AChR) antibodies were positive (reported as >8.5 nmol/L) and electromyography (EMG) showed a decremental response (reported as >15%); the disease was classified as Osserman class IIb with ocular and bulbar signs. For AS, human leukocyte antigen B27 (HLA-B27) was positive, with bilateral grade III sacroiliitis, spinal syndesmophytes, and ligamentum flavum calcification, together with the restricted cervical extension described above. For MS, the course was relapsing-remitting, cerebrospinal fluid showed oligoclonal bands, and magnetic resonance imaging showed periventricular plaques.

The obstetric findings were progressing cervical dilation, irregular contractions, and persistent fetal heart rate decelerations on cardiotocography. The team applied the structured decision algorithm shown in Figure 3 and agreed that emergency cesarean delivery was needed. Neuraxial spinal anesthesia (Plan A) was selected as the primary technique on two conditions: a low-dose local anesthetic and an immediately available airway-rescue plan (Plan B). Plan A avoids systemic neuromuscular blocking agents, whose effect in MG can be unpredictable and prolonged,⁶ and preserves spontaneous diaphragmatic ventilation. Airway management can also be difficult in AS because of cervical rigidity,^{10,11} which added to the reasons for avoiding general anesthesia.

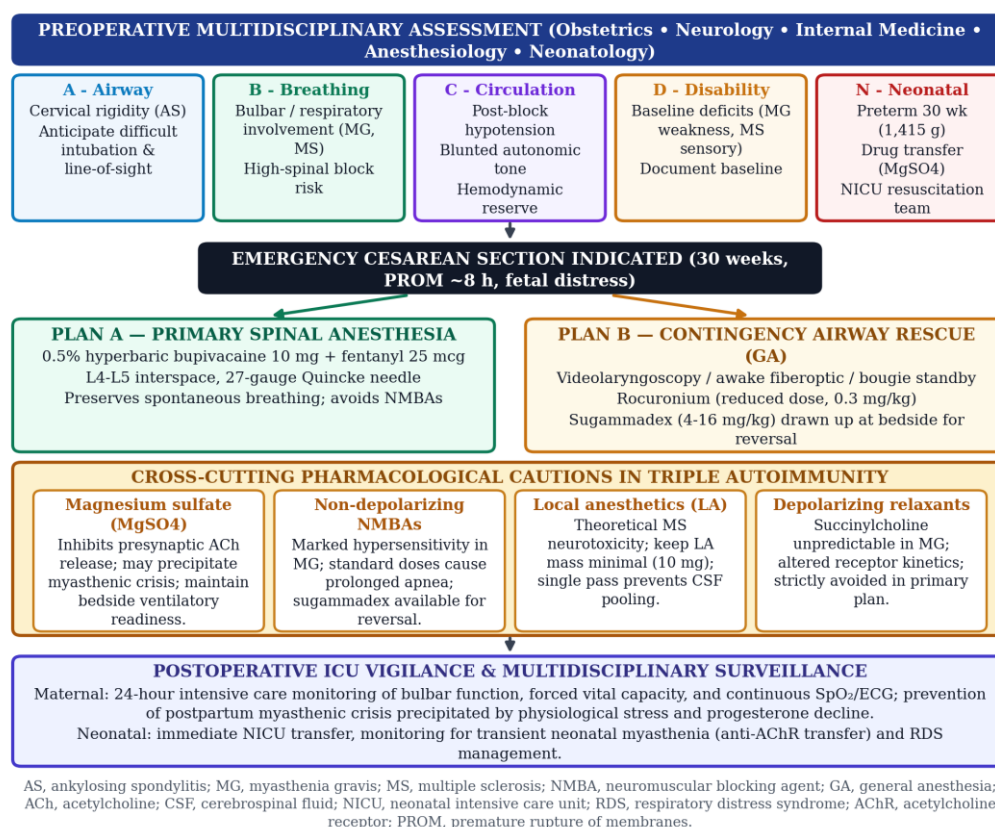


Figure 3. Decision algorithm and drug considerations for the parturient with MG, AS, and MS. The figure shows the domain-based assessment, the primary low-dose spinal technique (Plan A, performed), the staged airway rescue (Plan B, prepared and not used), and the drug considerations for magnesium sulfate, rocuronium with sugammadex, and local anesthetic dose. AS, ankylosing spondylitis; MG, myasthenia gravis; MS, multiple sclerosis.

Plan B was prepared in advance and followed a pre-formulated airway strategy.²⁴ A dedicated difficult-airway cart held videolaryngoscopes, flexible fiberoptic scopes, and supraglottic airway devices. For muscle relaxation, rocuronium 0.3 mg/kg was prepared with sugammadex (4 to 16 mg/kg) at the bedside for immediate reversal.²⁸ Plan A was carried out as planned. Plan B was not used.

3.3. Treatment course and outcome

Stabilization addressed maternal optimization, tocolysis, and fetal neuroprotection together. Hydration was given intravenously as a balanced crystalloid mixture (Ringer's lactate, 5% dextrose, and 0.9% saline in a 1:2:1 volumetric ratio) at 100 mL/h. Intramuscular dexamethasone sodium phosphate (5 mg every 12 h for four doses) was given for fetal lung maturation,²⁹ and capillary blood glucose was checked serially for steroid-induced hyperglycemia. Because the membranes had been ruptured since before presentation, intravenous ampicillin (1 g every 8 h) was started as prophylaxis, and aminoglycosides and fluoroquinolones were avoided. Tocolysis used oral nifedipine (20 mg loading dose, then 10 mg three times daily) while the steroids took effect.³⁰ Intravenous magnesium sulfate was started for fetal neuroprotection before birth at 30 weeks.¹⁶ It was given as a 4 g loading infusion over 20 minutes followed by 1 g/h, planned for 24 hours, with bedside neuromuscular monitoring, airway readiness, and sugammadex available.

After the decision described in Section 3.2, spinal anesthesia was performed and serial measurements were recorded at nine epochs, with T0 defined as the pre-spinal baseline (Table 4, Figure 2). Systolic blood pressure (SBP) was 138 mmHg at T0 (diastolic blood pressure [DBP] 66 mmHg, admission values 128/76 mmHg). SBP was 123 mmHg at block onset (T+15 min, -10.9%) and lowest at 110 mmHg at delivery (T+30 min, -20.3%). It then measured 117 mmHg at T+45 (-15.2%), 135 mmHg at T+60 (-2.2%), 122 mmHg at T+75 (-11.6%), 120 mmHg at T+90 (-13.0%), 113 mmHg at T+105 (-18.1%), and 115 mmHg at T+120 (-16.7%). DBP ranged from 64 to 68 mmHg at all epochs (-3.0% to +3.0%). From a baseline of 90.0 mmHg, MAP ranged from 82.0 to 89.7 mmHg, with its largest fall (-8.9%) at T+30, above the conventional perfusion threshold of 65 mmHg. After the baseline value of 76 beats/min, HR ranged from 69 to 73 beats/min (-3.9% to -9.2%). No bradycardia or other sign of autonomic instability was recorded. Definitions of hypotension after spinal anesthesia vary widely between studies,³¹ so the SBP fall at T+30 is reported factually: 110 versus 138 mmHg, a decrease of 20.3% from baseline. No rescue vasopressor was required. The sensory block was at the T6 dermatome at T+15 (Bromage 2), reached T4 at T+30 (Bromage 3), and remained at T4 through T+75.

Table 4. Serial hemodynamic, respiratory, and block-level measurements at nine perioperative epochs (T0 = pre-spinal baseline, T+15 to T+120 min).

Epoch	SBP, mmHg [%Δ]	DBP, mmHg [%Δ]	MAP, mmHg [%Δ]	HR, beats/min [%Δ]	RR, breaths/min [%Δ]	SpO ₂ , % [%Δ]	Sensory level	Bromage score	Clinical events
T0 (pre-spinal baseline)	138 [0.0%]	66 [0.0%]	90.0 [0.0%]	76 [0.0%]	20 [0.0%]	98 [0.0%]	None	0	Sitting position; L4-L5 lumbar puncture with a 27G Quincke needle; single pass without ultrasound
T+15 min (block onset)	123 [-10.9%]	68 [+3.0%]	86.3 [-4.1%]	71 [-6.6%]	20 [0.0%]	98 [0.0%]	T6	2	Warm sensation in both lower extremities; sensory block ascending smoothly; hemodynamically stable
T+30 min (cesarean delivery)	110 [-20.3%]	68 [+3.0%]	82.0 [-8.9%]	72 [-5.3%]	26 [+30.0%]	98 [0.0%]	T4	3	Uterine incision and delivery of the 1,415 g infant; transient tachypnea (RR 26 breaths/min); face-mask O ₂ 4 L/min started
T+45 min (placental delivery, uterine exteriorization)	117 [-15.2%]	68 [+3.0%]	84.3 [-6.3%]	69 [-9.2%]	24 [+20.0%]	97 [-1.0%]	T4	3	Placental delivery and uterine exteriorization; tachypnea resolving; no clinical sign of high block; oxytocin 10 IU infusion
T+60 min (uterine closure)	135 [-2.2%]	67 [+1.5%]	89.7 [-0.3%]	70 [-7.9%]	20 [0.0%]	98 [0.0%]	T4	3	Myometrial closure; RR back to 20 breaths/min; patient comfortable; no shivering
T+75 min (abdominal closure)	122 [-11.6%]	64 [-3.0%]	83.3 [-7.4%]	71 [-6.6%]	20 [0.0%]	97 [-1.0%]	T4	3	Peritoneal and fascial closure; no vasopressor was required
T+90 min (skin closure, PACU)	120 [-13.0%]	66 [0.0%]	84.0 [-6.7%]	73 [-3.9%]	20 [0.0%]	98 [0.0%]	T6	2	Surgery completed; subcuticular skin closure; transfer to PACU
T+105 min (block regression)	113 [-18.1%]	68 [+3.0%]	83.0 [-7.8%]	71 [-6.6%]	18 [-10.0%]	98 [0.0%]	T8	1	Regression of sensory and motor block; spontaneous breathing, stable; temperature 36.2 °C

Epoch	SBP, mmHg [%Δ]	DBP, mmHg [%Δ]	MAP, mmHg [%Δ]	HR, beats/min [%Δ]	RR, breaths/min [%Δ]	SpO ₂ , % [%Δ]	Sensory level	Bromage score	Clinical events
T+120 min (ICU admission)	115 [−16.7%]	68 [+3.0%]	83.7 [−7.0%]	72 [−5.3%]	20 [0.0%]	97 [−1.0%]	T10	1	Admission to the ICU for 24 h of neuro-respiratory surveillance

Notes: Values are raw measurements, with the percentage change (%Δ) relative to the pre-spinal baseline in square brackets: $\% \Delta = [(T_n - T_0) / T_0] \times 100$. Time is given in minutes relative to T₀, the pre-spinal baseline; the interval between hospital presentation and T₀ is not reported. Admission BP was 128/76 mmHg (see Table 2) and the T₀ value was 138/66 mmHg. Sensory level is the highest dermatome with loss of cold or pinprick sensation. Bromage scale: 0, no motor block (full flexion of knees and feet); 1, partial block (just able to move knees); 2, almost complete block (able to move feet only); 3, complete block (unable to move feet or knees). DBP, diastolic blood pressure; HR, heart rate; ICU, intensive care unit; MAP, mean arterial pressure, calculated as $(SBP + 2 \times DBP) / 3$; PACU, post-anesthesia care unit; RR, respiratory rate; SBP, systolic blood pressure; SpO₂, peripheral oxygen saturation.

Thirty minutes after spinal injection, a preterm female infant was delivered in cephalic presentation. Table 5 gives her anthropometric data, Apgar scores, and Downes respiratory distress scores.²⁷ She weighed 1,415 g, which falls in the very low birth weight (VLBW, <1,500 g) category. Crown-heel length was 39 cm and head circumference was 28 cm, both reported at the 50th percentile for 30 weeks. The 1-minute Apgar score was 5 (heart rate 2, respiratory effort 1, muscle tone 1, reflex irritability 1, color 0). The neonatal team provided thermal

support under a radiant warmer, dried the infant, cleared the airway, and started nasal continuous positive airway pressure (CPAP) with a fraction of inspired oxygen (FiO₂) of 0.30 through a T-piece resuscitator. The infant improved with positive-pressure inflation. The 5-minute Apgar score was 7 (heart rate 2, respiratory effort 1, muscle tone 1, reflex irritability 2, color 1), and the 10-minute score was 8 (heart rate 2, respiratory effort 2, muscle tone 1, reflex irritability 2, color 1).

Table 5. Neonatal anthropometric data, Apgar scores, and Downes respiratory distress score progression.

Parameter	Observed value	Reference or category	Interpretation and response
Neonatal anthropometric data			
Sex and gestational age	Female, 30 weeks + 2 days	Preterm (<37 weeks)	Preterm newborn at risk of surfactant deficiency and hypothermia
Birth weight	1,415 g	VLBW category (<1,500 g)	Very low birth weight infant
Crown-heel length	39 cm	50th percentile for 30 weeks, as reported	Appropriate for gestational age
Head circumference	28 cm	50th percentile for 30 weeks, as reported	Head circumference appropriate for gestational age
Apgar scores			
1-minute Apgar score	5/10 (HR 2, respiratory effort 1, tone 1, reflex 1, color 0)	Low (<7)	Initial depression may reflect prematurity; magnesium exposure was not quantified; drying, warmth, and airway clearing given immediately
5-minute Apgar score	7/10 (HR 2, respiratory effort 1, tone 1, reflex 2, color 1)	Normal (≥7)	Improvement after positive-pressure stabilization; peripheral cyanosis resolving; heart rate >140 beats/min
10-minute Apgar score	8/10 (HR 2, respiratory effort 2, tone 1, reflex 2, color 1)	Normal (≥7)	Sustained spontaneous respiratory effort and grimace; stable on non-invasive support
Downes score progression			
Delivery (0 h)	5/10 (RR 68 breaths/min, moderate retractions, audible grunt)	Moderate distress (4-6)	Respiratory distress consistent with RDS; nasal CPAP started (PEEP 6 cmH ₂ O, FiO ₂ 0.30)
After CPAP stabilization (2 h)	4/10 (RR 62 breaths/min, mild retractions, intermittent grunt)	Moderate distress (4-6)	Gradual improvement on CPAP; no cyanosis
NICU (6 h)	3/10 (RR 56 breaths/min, minimal retractions, no grunting)	Mild distress (1-3)	Clinical improvement; FiO ₂ weaned to 0.25 on CPAP
NICU (12 h)	2/10 (RR 52 breaths/min, minimal subcostal retraction)	Mild distress (1-3)	Blood gas: pH 7.36, PaCO ₂ 42 mmHg, PaO ₂ 78 mmHg
NICU (24 h)	1/10 (RR 48 breaths/min, no distress, clear bilateral air entry)	Mild distress (1-3)	Weaned toward room air on CPAP; enteral trophic feeding started by nasogastric tube
Neuro-immunological surveillance			
Transplacental passage of anti-AChR antibodies (clinical neonatal MG)	No clinical features of neonatal MG during 24 h of observation	Transient neonatal MG is reported in about 10-20% of newborns of mothers with MG (see Table 6)	Normal spontaneous limb tone, strong cry, and intact suckling reflex; no ptosis or respiratory weakness

Notes: Downes score²⁷ has five criteria (cyanosis, retractions, grunting, air entry, respiratory rate), each scored 0 (none), 1 (mild), or 2 (severe), for a total of 0-10. Bands used in this table: 1-3, mild distress; 4-6, moderate distress; ≥7, impending respiratory failure. Apgar scores: 7-10 is conventionally regarded as normal and <7 as low. AChR, acetylcholine receptor; CPAP, continuous positive airway pressure; FiO₂, fraction of inspired oxygen; HR, heart rate; MG, myasthenia gravis; NICU, neonatal intensive care unit; PaCO₂ and PaO₂, partial pressure of arterial carbon dioxide and oxygen; PEEP, positive end-expiratory pressure; RDS, respiratory distress syndrome; RR, respiratory rate; VLBW, very low birth weight.

Figure 4 shows the maternal and neonatal courses side by side: maternal MAP and HR (panel A), maternal RR and peripheral oxygen saturation (SpO₂; panel B), neonatal Apgar scores (panel C), and the Downes score (panel D). In the neonatal intensive care unit (NICU), nasal CPAP continued at a positive end-expiratory pressure (PEEP) of 6 cmH₂O and FiO₂ 0.30, and the Downes score fell from 5

at delivery to 1 at 24 hours (Table 5). FiO₂ was weaned to 0.25 by 6 hours. The infant was observed for clinical features of transient neonatal MG: spontaneous limb flexion was vigorous, the cry was strong, and the suckling reflex was normal. No clinical feature of neonatal MG was seen during the 24-hour observation.

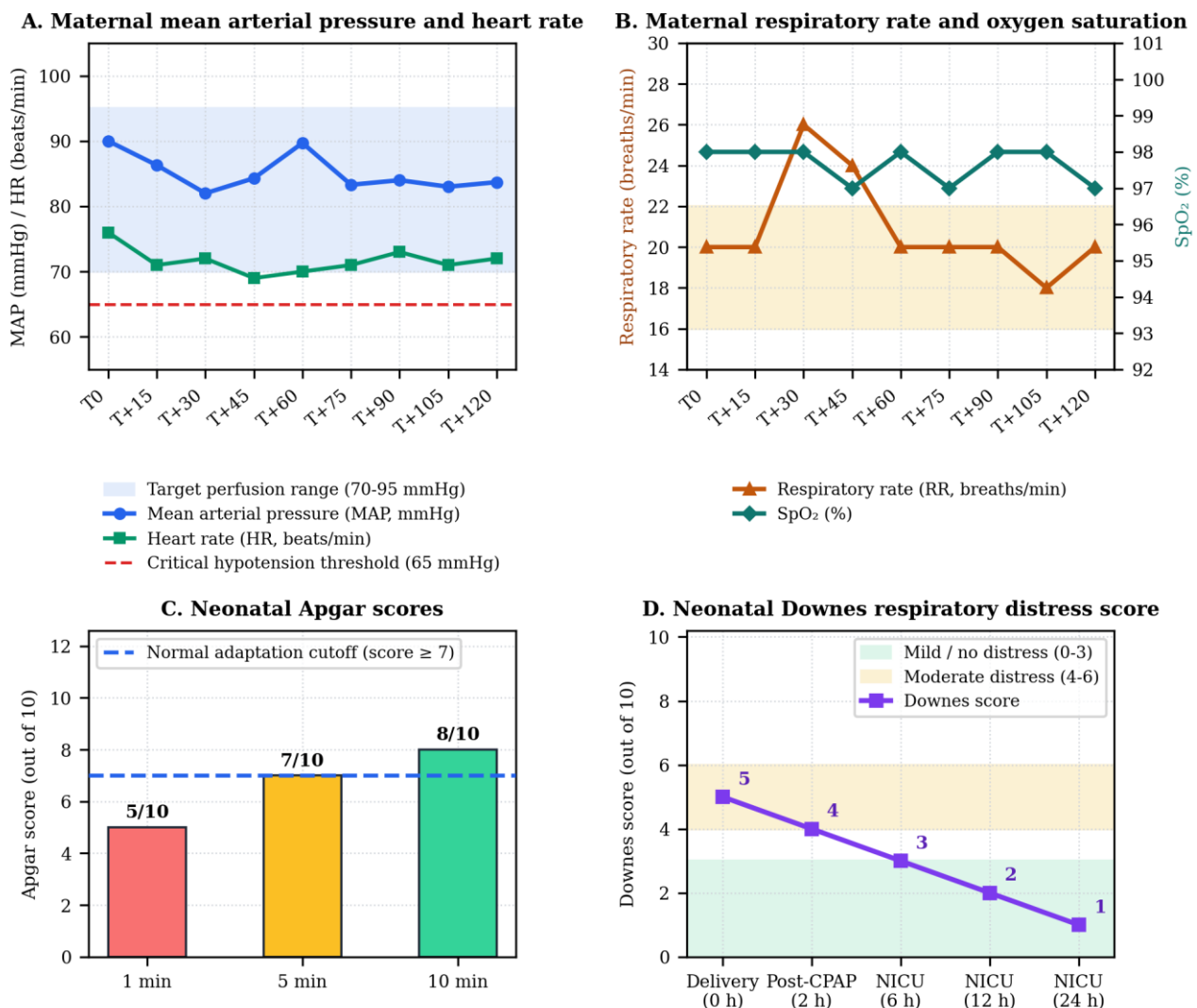


Figure 4. Maternal and neonatal courses. (A) Maternal mean arterial pressure (MAP) and heart rate. (B) Maternal respiratory rate and SpO₂. (C) Neonatal Apgar scores (5 at 1 min, 7 at 5 min, 8 at 10 min). (D) Neonatal Downes respiratory distress score over 24 h (5 at delivery, 1 at 24 h). Panels A and B repeat the maternal series of Figure 2 and Table 4. SpO₂, peripheral oxygen saturation.

One transient intraoperative change in respiratory pattern was recorded: an isolated rise in RR to 26 breaths/min (+30.0%) at T+30 min, which coincided with uterine exteriorization and extraction of the infant (Table 4). SpO₂ remained 98%. Examination showed a sensory block confined to the T4 dermatome, with no motor block of the upper extremities, no Horner syndrome, no difficulty with phonation, and no dyspnea; there was no clinical sign of a high or total spinal block. Supplemental oxygen was started at once by simple face mask at 4 L/min. RR was 24 breaths/min at T+45 (+20.0%) and returned to the baseline of 20 breaths/min at T+60 (0.0%).

The abdomen was opened through a Pfannenstiel skin incision, and the uterus through a lower uterine segment hysterotomy. Estimated blood loss was about 450 mL. After extraction of the infant, an intravenous oxytocin infusion (10 IU in 500 mL of normal saline at 125 mL/h) was given, and uterine contraction was prompt and sustained. No second-line uterotonic such as methylergonovine or carboprost was needed; both can cause hypertension or bronchospasm. Uterine repair, peritoneal closure, and abdominal wall closure were completed without surgical complication. The operating time from skin incision to closure was recorded as 48

minutes. At the end of surgery (T+90) the sensory block was still at T6 with Bromage 2 motor block. It regressed to T8 and Bromage 1 by T+105, and to T10 by T+120 (Bromage 1).

After surgery the mother was admitted to the ICU for 24 hours of surveillance, with serial forced vital capacity (FVC) and negative inspiratory force (NIF) measurements and neurological examinations aimed at detecting delayed myasthenic decompensation. Oral pyridostigmine (60 mg five times daily) and methylprednisolone (8 mg twice daily) were resumed without interruption, and the magnesium sulfate infusion was completed after 24 hours without adverse effect. Vital signs remained stable throughout the ICU stay, and no episode of respiratory distress, bulbar deterioration, or autonomic instability was recorded.

On postoperative day 1 the mother was transferred to the obstetric-neurology step-down ward. Muscle strength was stable at baseline, and bulbar function was reported as intact. There was no MS relapse and no surgical site infection. Both mother and infant had an uncomplicated early course. Published findings relevant to these observations are summarized in Table 6, together with their relation to this patient.

Table 6. Published evidence relevant to the clinical issues in this case, and its relation to the observations in this patient.

Topic	Source	Verified finding	Relation to this case (observation only)
MG in pregnancy: maternal and obstetric outcomes	(a) Meta-analysis ¹ ; (b) systematic review with case series ² ; (c) single-center series ³² ; (d) national registry ³⁴ ; (e) national register cohort ³³	(a) 15 studies, 734 pregnancies: pooled proportion with MG worsening 0.36 (95% CI 0.25-0.40). (b) 824 pregnancies: exacerbation 33.8%; myasthenic crisis 6.4% in pregnancy and 8.2% postpartum. (c) 66 pregnancies: worsening in 40 (63%), IVIg or plasma exchange in 10 (15.7%), no myasthenic crisis, cesarean 38%. (d) 134 MG births: elective cesarean aOR 1.8 (1.1-3.1), neuraxial anesthesia aOR 1.5 (1.1-2.1), preterm PROM aOR 2.7; emergency cesarean not increased. (e) 443 MG births versus 4,249 matched: no increased risk of pregnancy complications; spontaneous labor onset less often (69.8% vs 79.5%).	This patient presented with PROM at 30 weeks and was delivered by emergency cesarean. No myasthenic crisis was recorded in 24 h of ICU observation. One patient cannot be compared with these rates.
Multidisciplinary planning of delivery in MG	(a) Review ²² ; (b) review ⁴⁴	(a) Counseling, planning, and monitoring by a multidisciplinary team (obstetrician, anesthesiologist, pediatrician, neurologist); outcome favorable with treatment. (b) Pyridostigmine, prednisolone, and azathioprine regarded as safe in pregnancy; maternal MG stable in most; delivery at hospitals with neonatal intensive care advised.	A multidisciplinary decision was made (Figure 3), pyridostigmine and methylprednisolone were continued, and the infant was admitted to the NICU.
AS in pregnancy	Cohort study ³⁵	332 axial spondyloarthritis pregnancies: live birth 98.8%, preterm birth 4.9%, low birth weight 3.1%, small for gestational age 9.5%, cesarean 27.7% of pregnancies (47.4% of these emergency). No anesthesia or airway data.	Delivery at 30 weeks (preterm) by emergency cesarean; birth weight 1,415 g.
AS and airway management	(a) Case report ¹⁰ ; (b) case report ¹¹	(a) 65-year-old man with AS (Mallampati III, sternal distance 10 cm, severe restriction of cervical extension): awake videolaryngoscopy under remifentanyl sedation succeeded; AS complicates airway management. (b) AS with cervical deformity: awake fiberoptic nasotracheal intubation under remifentanyl and topical anesthesia succeeded. Neither report is obstetric.	Cervical extension 35° and sternal distance 11.5 cm. Airway equipment and a rescue plan (Plan B) were prepared; the airway was not instrumented.
MS: postpartum relapse and neuraxial anesthesia	(a) Meta-analysis ¹⁴ ; (b) cohort ¹³ ; (c) systematic review ¹² ; (d) cohort ³⁶ ; (e) MSBase cohort ³⁷	(a) 11 cohorts, 2,739 pregnancies: relapse rate higher in the first 6 months postpartum; incidence rate ratio 1.87 (1.40-2.50) in the first 3 months. (b) 118 deliveries in 104 women: postpartum relapse in 31 women (26%); neuraxial procedures not associated with increased postpartum relapse. (c) 34 studies, 2,326 patients: neuraxial anesthesia can be administered safely; postpartum relapse in 364 of 1,139 cohort patients, not directly associated with technique. (d) 111 women: annualized relapse rate 0.14 before pregnancy, 0.13 during pregnancy, and 0.33 at 3 months postpartum (rate ratio 2.42, 1.03-5.67). (e) 2,009 live births: postpartum relapse 4.1% on ocrelizumab versus 15.2-30.5% in other disease-modifying therapy groups.	No MS relapse was recorded by postoperative day 1. This follow-up does not cover the postpartum period of highest relapse risk.
Magnesium sulfate in MG	(a) Case report ¹⁷ ; (b) case report ¹⁸ ; (c) retrospective review ¹⁹	(a) MG with severe preeclampsia managed without magnesium; magnesium sulfate described as strongly contraindicated in MG. (b) MG patient developed acute respiratory failure needing intubation after IV magnesium sulfate, and again on re-exposure. (c) Exacerbation in 7 of 55 encounters (12.7%) after IV magnesium or labetalol.	Magnesium sulfate (4 g, then 1 g/h) was given with bedside airway and reversal readiness. No respiratory deterioration was recorded.
Magnesium sulfate for fetal neuroprotection	Cochrane review ¹⁶	6 randomized trials, 5,917 women (<34 weeks): cerebral palsy RR 0.71 (0.57-0.89); death or cerebral palsy RR 0.87 (0.77-0.98); adverse effects requiring cessation RR 3.21 (1.88-5.48). No data on MG.	Given at 30 weeks. Infant follow-up was 24 h, so neurological outcome cannot be assessed.
Rocuronium and sugammadex in MG	(a) Cohort ²⁵ ; (b) cohort ²⁶ ; (c) systematic review of case reports ²⁸	(a) 1,143 MG thymectomy patients: rocuronium reversed with sugammadex versus no relaxant, no excess respiratory complications. (b) 2,304 MG surgical patients: treatment for myasthenic crisis 6.2% (rocuronium-sugammadex) versus 5.3% (no neuromuscular blocker), HR 1.14 (0.70-1.85). (c) 43 case reports (15 in MG): most report successful reversal; response unpredictable; quantitative monitoring advised.	Rocuronium 0.3 mg/kg and sugammadex were prepared as part of Plan B and were not administered.
Transient neonatal MG	(a) Systematic review ² ; (b) systematic review ⁴²	(a) Transient neonatal MG in 13.0% of 824 MG pregnancies. (b) Transient neonatal MG in 10-20% of newborns of myasthenic mothers; maternal antibodies cross the placenta.	No clinical feature of neonatal MG during 24 h of observation; infant antibody testing is not reported.
Hypotension after spinal anesthesia for cesarean delivery	Systematic review ³¹	38 studies (n = 3,086): hypotension after spinal anesthesia for elective cesarean ranged from 29% to 80% depending on the definition.	SBP fell 20.3% at T+30 (110 versus 138 mmHg); lowest MAP 82.0 mmHg; no rescue vasopressor was required.

Notes: Findings are reproduced as reported in the cited abstracts. The right-hand column records observations in this patient only; it does not imply comparison, effect, or causality. aOR, adjusted odds ratio; AS, ankylosing spondylitis; CI, confidence interval; HR, hazard ratio; ICU, intensive care unit; IV, intravenous; IVIg, intravenous immunoglobulin; MAP, mean arterial pressure; MG, myasthenia gravis; MS, multiple sclerosis; NICU, neonatal intensive care unit; PROM, premature rupture of membranes; RR, relative risk; SBP, systolic blood pressure.

4. Discussion

4.1. Principal lesson of the case

This report describes the anesthetic management of an emergency cesarean delivery in a 34-year-old woman at 30 weeks and 2 days of gestation (American Society of Anesthesiologists [ASA] physical status IV-E). She had three coexisting autoimmune diseases: MG, AS, and MS. The case combined four problems. One was the risk of neuromuscular ventilatory failure. Another was a rigid axial spine that could obstruct neuraxial needle placement. A third was a poor prospect of laryngeal exposure if general anesthesia became necessary, and the fourth was the theoretical vulnerability of demyelinated central axons to local anesthetic. We did not measure whether the three diseases interact. What the case shows is that their problems overlapped at the neuromuscular junction, the axial skeleton, the airway, and the central nervous system, and that each had to be addressed within a single plan.

Delivery was completed under single-shot spinal anesthesia (Plan A). No myasthenic crisis, airway intervention, or vasopressor requirement occurred. The infant survived with improving respiratory status. Plan B, the airway-rescue strategy, was prepared but not used. One uncontrolled case cannot show that this plan caused the outcome, nor that it is better than another approach. The lesson is narrower. When each risk was assigned a prepared response, a neuraxial technique could be used in this patient without any of the feared complications being observed.

4.2. Comparison with the published literature

Pregnancy in MG carries a risk of worsening that varies between series. A meta-analysis of 15 studies (734 pregnancies) gave a pooled proportion with worsening of 0.36 (95% confidence interval [CI] 0.25–0.40), and about two thirds of the events occurred antepartum.¹ A systematic review and case series of 824 pregnancies reported exacerbation in 33.8%, myasthenic crisis in 6.4% during pregnancy and 8.2% postpartum, and transient neonatal MG in 13.0%.² In a single German center, 40 of 66 pregnancies (63%) were complicated by worsening of symptoms. No myasthenic crisis was recorded.³² Population registries paint a more reassuring picture overall. A Swedish cohort found no increased risk of pregnancy complications in MG,³³ whereas a Norwegian registry found higher odds of elective cesarean (adjusted odds ratio [aOR] 1.8), neuraxial anesthesia (aOR 1.5), and preterm prelabor rupture of membranes (PPROM; aOR 2.7), with no increase in emergency cesarean.³⁴ Our patient had PPRM and was delivered by emergency cesarean under neuraxial anesthesia. Registry associations apply to groups and say nothing about the course of one woman. Even so, the pattern in this patient is not unusual for MG.

For AS, a cohort of 332 pregnancies in axial spondyloarthritis reported a live birth rate of 98.8%, preterm birth in 4.9%, and cesarean delivery in 27.7% of pregnancies, almost half of them emergency procedures.³⁵ That source contains no anesthetic or airway data. Airway reports come from non-obstetric patients. Awake videolaryngoscopy under remifentanyl sedation succeeded in a man with AS and severe cervical extension restriction, and the authors noted that direct laryngoscopy has a low success rate in this condition.¹⁰ Awake fiberoptic nasotracheal intubation was likewise successful in a patient with AS and cervical deformity.¹¹ These reports

support the decision to prepare videolaryngoscopic and fiberoptic options for our patient, whose cervical extension was limited to 35° with a sternomental distance of 11.5 cm. Obstetric patients also undergo difficult intubation more often than the general surgical population, which adds to the risk of conversion to general anesthesia.

For MS, the main concern after delivery is relapse. A meta-analysis of 11 cohorts (2,739 pregnancies) found an increased relapse rate in the first 6 months postpartum, with an incidence rate ratio of 1.87 (95% CI 1.40–2.50) in the first 3 months.¹⁴ In women with relapsing-remitting MS conceiving after assisted reproduction, the annualized relapse rate rose from 0.13 during pregnancy to 0.33 at 3 months postpartum.³⁶ Relapse risk also differs by disease-modifying therapy.³⁷ As for neuraxial technique, a French series of 118 deliveries (46 under neuraxial anesthesia and 50 under neuraxial analgesia) found postpartum relapse in 26% of the women, with no association with the neuraxial procedure.¹³ A systematic review of 34 studies including 2,326 patients, mostly case reports and small series, concluded that neuraxial anesthesia can be given safely, although some adverse neurological events were documented.¹² A recent review also considers neuraxial and general anesthesia safe in MS and MG. It notes that the response to neuromuscular blocking drugs is unpredictable.⁶ The evidence is observational. Our patient was followed only to postoperative day 1, so we cannot comment on relapse in her.

Magnesium sulfate is the point of greatest conflict in the literature. One case report of MG with severe preeclampsia, managed without magnesium, describes the drug as strongly contraindicated because it impairs already slowed nerve-muscle transmission.¹⁷ Another report described acute respiratory failure requiring intubation after intravenous magnesium in a woman with MG, and the failure recurred on re-exposure.¹⁸ In a retrospective review, exacerbation followed intravenous magnesium or labetalol in 7 of 55 encounters (12.7%).¹⁹ Against this stands the neonatal benefit. In an updated Cochrane review of 6 randomized trials (5,917 women before 34 weeks), magnesium sulfate reduced cerebral palsy (relative risk [RR] 0.71, 95% CI 0.57–0.89), although adverse effects requiring cessation of treatment were more frequent (RR 3.21, 95% CI 1.88–5.48).¹⁶ A meta-analysis of 7 randomized trials gave a similar reduction in neurological impairment without an effect on mortality.³⁸ In pregnancies with PPRM at 23 to 31 weeks, magnesium sulfate was associated with longer latency and less cerebral palsy.³⁹ Our patient received a 4 g loading dose followed by 1 g/h, under continuous respiratory monitoring. No respiratory deterioration was recorded. This differs from the reports of respiratory failure, but the comparison is indirect. The cited cases differ in dose, duration, and disease severity, and no serum magnesium level or quantitative neuromuscular monitoring is available for our patient.

Evidence on rocuronium with sugammadex in MG is larger than for the other topics, though it comes from surgical patients rather than parturients. In 1,143 patients with MG undergoing thymectomy, rocuronium reversed with sugammadex was not associated with excess respiratory complications compared with no muscle relaxant.²⁵ Among 2,304 patients with MG, treatment for myasthenic crisis was given in 6.2% after rocuronium and sugammadex and in 5.3% after no neuromuscular blocker (hazard ratio 1.14, 95% CI 0.70–1.85).²⁶ A systematic review of 43 case reports, 15 of them in MG, reported successful reversal in most patients but an unpredictable response, and it

advised quantitative monitoring.²⁸ Sugammadex has also been described as a safer alternative to neostigmine in neuromuscular disorders.⁴⁰ Plan B was not executed in our patient. None of this evidence was tested here.

For hemodynamics, a systematic review of 38 studies found hypotension after spinal anesthesia for elective cesarean in 29% to 80% of patients, depending on the definition.³¹ Prophylactic norepinephrine or phenylephrine infusion lowers this risk in cesarean delivery.⁴¹ Our patient's mean arterial pressure stayed between 82.0 and 90.0 mmHg without a vasopressor. For the neonate, transient neonatal MG is reported in 10% to 20% of newborns of myasthenic mothers because maternal antibodies cross the placenta,⁴² and maternal acetylcholine receptor antibodies can also harm the fetus.⁴³ Finally, the spinal dose in our patient (bupivacaine 10 mg with fentanyl 25 mcg) lies within the range of a randomized trial in cesarean delivery. In that trial, 25 mcg was the highest fentanyl dose studied, and the groups given more fentanyl needed more phenylephrine.²³

4.3. Pathophysiological or mechanistic interpretation

This section separates what was observed from what is inferred. The observations are those reported in the Results: there was no myasthenic crisis, the spinal was a single pass at L4-L5, arterial pressure and heart rate stayed stable without a vasopressor, and respiratory rate rose once, transiently. The preterm infant improved on nasal CPAP. Everything below that explains why these things happened is inference, and none of it was tested in this patient.

In MG, antibodies to the postsynaptic acetylcholine receptor reduce receptor numbers and increase their turnover, and this narrows the safety margin of neuromuscular transmission.⁴ Fatigable weakness follows.⁵ During labor or operative delivery, a parturient could develop fatigue of the diaphragmatic and intercostal muscles, and that fatigue could lead to ventilatory decompensation. Pregnancy may also destabilize anticholinesterase dosing through changes in plasma volume, protein binding, and renal clearance. Regional anesthesia avoids neuromuscular blocking agents and leaves spontaneous ventilation intact, and for that reason we considered it the logical first choice. No crisis occurred in this patient. That fits the reasoning. It does not demonstrate it.

Magnesium reduces calcium-dependent release of acetylcholine at the presynaptic terminal. This is the usual explanation for why it adds to the postsynaptic deficit in MG. Reported consequences include respiratory failure.^{17,18} We gave magnesium sulfate for fetal neuroprotection because delivery before 32 weeks was threatened, with an advanced airway cart, ventilatory support, and sugammadex were available and with continuous respiratory monitoring at the bedside. No respiratory deterioration was observed. Possible explanations include the monitoring and early recognition, the dose and duration of exposure before delivery, and individual susceptibility. Magnesium levels were not measured, so we cannot say how much magnesium reached the neuromuscular junction, and neuroprotection was not assessed in the infant.

In AS, chronic inflammation involving the interleukin-23 and interleukin-17 axis is linked to new bone formation,^{7,8} and disease activity has been associated with syndesmophyte progression.⁹ Ossification of the

supraspinous and interspinous ligaments and the ligamentum flavum in the lumbar spine may make midline needle insertion difficult. Repeated blind attempts in rigid anatomy risk periosteal trauma, hematoma, dural injury, and delay. In our patient, palpation found an accessible interspace at L4-L5, and dural puncture then succeeded in a single pass with a 27-gauge Quincke needle, without ultrasound. Ultrasound is generally advised to define distorted spinal anatomy, and one successful puncture does not show that palpation is adequate in established ankylosis. How far this result can be generalized also depends on the level of experience of the operator.

The need for Plan B arose from the same skeletal pathology. Had spinal anesthesia failed or been unattainable, conversion to general anesthesia would have exposed the patient to a difficult airway. Laryngoscopy requires alignment of the oral, pharyngeal, and laryngeal axes, and cervical syndesmophytes and joint rigidity restrict neck extension and that alignment. Videolaryngoscopy uses indirect camera visualization, so it does not need the alignment, and fiberoptic bronchoscopy allows intubation with spontaneous breathing preserved. The cart assembled before the procedure held videolaryngoscopes, flexible fiberoptic bronchoscopes, gum elastic bougies, and second-generation supraglottic devices. A pre-formulated strategy with video laryngoscopy and alternatives matches the difficult-airway guideline.²⁴ The airway was never instrumented. The adequacy of the plan remains untested.

For MS, the theoretical concern is that demyelinated central pathways may be more sensitive to local anesthetic neurotoxicity. The risk would be conduction block, neurological worsening, or postpartum relapse, and the observational evidence discussed above has not shown an association between neuraxial technique and relapse.^{12,13} and published reviews suggest that neuraxial anesthesia can be given safely in MS.^{6,12} Safety is thought to depend on the total mass of local anesthetic delivered, which supports the choice of a low-dose regimen (bupivacaine 10 mg with fentanyl 25 mcg). Body temperature was also kept at 36.0 °C intraoperatively, because a small rise in core temperature can transiently worsen conduction in demyelinated axons (the Uhthoff phenomenon). The block gave surgical anesthesia sufficient for cesarean delivery. We cannot say whether a different dose would have been adequate.

The rationale for rocuronium at 0.3 mg/kg with sugammadex available (up to 16 mg/kg) is as follows. In MG, standard doses of non-depolarizing relaxants can cause deep and unpredictable paralysis,^{6,28} and the response to depolarizing relaxants is variable. Reversal with neostigmine is problematic in patients already taking pyridostigmine. Sugammadex forms a 1:1 complex with rocuronium and acts independently of acetylcholinesterase activity, so it is considered a safer alternative.⁴⁰ A reduced rocuronium dose with immediate reversal gave the team a prepared means of controlling neuromuscular blockade should general anesthesia have been required. This remained a contingency. No relaxant or reversal agent was given.

Intraoperative mean arterial pressure stayed between 82.0 and 90.0 mmHg, with a largest decrease of 8.9% at T+30 min (T0 = pre-spinal baseline). Heart rate was between 69 and 76 beats/min. Spinal anesthesia produces sympathetic blockade with vasodilation, venous pooling, and reduced venous return, and hypotension is common after spinal

anesthesia for cesarean delivery.³¹ In our patient, possible autonomic dysfunction from MS and long-standing autoimmune disease raised the concern of an exaggerated hypotensive response. Three measures were used that may have limited it: a low-dose bupivacaine regimen, which may limit cephalad spread of the block; crystalloid co-loading of 1,500 mL of balanced crystalloids; and a 15-degree left lateral uterine tilt, which reduces aortocaval compression. A single case cannot determine which of these measures, if any, accounted for the stability.

The one perturbation was a transient rise in respiratory rate at T+30 min, coinciding with uterine exteriorization and delivery of the infant (described in the Results). It has several possible explanations, and the data cannot separate them. In a parturient without neuromuscular disease, a brief rise in respiratory rate during cesarean delivery is often attributed to anxiety, peritoneal traction, or ascent of the block. In this patient the differential was wider, and it included cephalad extension of motor block affecting the phrenic or intercostal roots, diaphragmatic fatigue from myasthenic weakness, and weakness related to magnesium. The bedside findings reported in the Results (sensory level at T4, normal phonation, preserved grip strength, and no dyspnea or Horner syndrome) argued against a high block. The prompt response to oxygen was reassuring, but a response to oxygen does not establish the cause, and the rate fell gradually to 24 and 20 breaths/min over the next 30 minutes. No quantitative neuromuscular or respiratory function testing is available. We regard the event as unexplained but self-limited.

The neonatal findings need cautious interpretation. The infant was born at 30 weeks and weighed 1,415 g (very low birth weight). The 1-minute Apgar score was 5, and the Downes score was 5.²⁷ These findings may reflect prematurity, in which surfactant deficiency is the underlying disorder of respiratory distress syndrome.²⁹ Transplacental magnesium is a further possibility, but it was not measured. The Apgar scores improved to 7 at 5 minutes and 8 at 10 minutes, and the Downes score fell to 1 at 24 hours on nasal CPAP. Antenatal dexamethasone was given and resuscitation was prompt, but their individual contributions cannot be separated in one infant, particularly as the steroid course was incomplete before delivery. In NICU observation the infant showed vigorous motor activity and normal suckling, without ptosis or respiratory weakness, so transient neonatal MG was not observed. Maternal antibodies cross the placenta.⁴² Even so, a single unaffected infant does not show that antibody positivity seldom leads to neonatal disease.

Figure 5 is a conceptual schematic that contrasts two management pathways. It is a hypothetical contrast and not a result. The left-hand pathway was not applied to any patient in this report. Its elements are risks that have been described: unplanned general anesthesia with difficult airway access in AS,²⁴ unadjusted neuromuscular blocker dosing,^{28,40} and unmonitored magnesium infusion with respiratory failure in MG.^{17,18} The remaining elements of the conventional pathway, including a high local anesthetic

mass and postoperative ventilator dependence, reflect our own reasoning and not cited data. The right-hand pathway outlines the plan that was applied here, which consisted of low-dose spinal anesthesia, a prepared airway and sugammadex rescue, magnesium under continuous respiratory monitoring, and attention to temperature and hemodynamics. The figure expresses how the team organized its reasoning. It does not show that one pathway leads to better outcomes than the other.

4.4. Implications for practice

Several practical points arise from this case, though it establishes none of them. First, assigning each coexisting disease a specific anesthetic question (ventilation, access, airway, neurotoxicity) made it possible to select the neuraxial technique deliberately and to prepare an airway alternative. Second, when magnesium sulfate is considered in MG, the possible respiratory effect can be managed by continuous respiratory monitoring and by having ventilatory support and rescue equipment present before the infusion starts. Quantitative neuromuscular monitoring would add objective information.⁴⁴ Third, the data on rocuronium and sugammadex in MG come from non-obstetric patients,^{25,26,28} so the response should be treated as unpredictable.

Peripartum immunomodulation required a balance between disease control and infection risk. Oral pyridostigmine (60 mg five times daily) was continued until the immediate preoperative period to avoid myasthenic relapse, and pyridostigmine is regarded as safe in pregnancy. Methylprednisolone (8 mg twice daily, 16 mg/day) was also continued. Intravenous stress-dose hydrocortisone was kept available because of the risk of relative adrenal insufficiency during emergency laparotomy. Adalimumab, which is used for axial inflammation in AS, was withheld because of prolonged rupture of membranes (more than 8 h). The aim was to avoid additive immunosuppression and limit the risk of maternal and neonatal sepsis. No ketoacidosis was recorded in the mother, and no neonatal hypoglycemia was documented in this report. Still, the combined effect of chronic methylprednisolone and antenatal dexamethasone on glucose remains a reason for monitoring.

Multidisciplinary collaboration was central to this case. Obstetrics, anesthesiology, neurology, internal medicine, and neonatology each had a defined task. The obstetric team set the gestational limits and the urgency of delivery. Neurology assessed neuromuscular reserve and the threshold for myasthenic crisis, and internal medicine supervised immunosuppression and infection risk. The anesthetic team designed the primary technique and the rescue pathways, while the neonatal team carried out resuscitation of the preterm infant. Planning, counseling, and monitoring by such a team is recommended in MG in pregnancy,²² and delivery at a center with neonatal intensive care is advised.⁴⁴ Every intervention was checked against all coexisting diseases before it was given.



Figure 5. Conceptual schematic of the pathophysiological pathways considered by the authors in a parturient with MG, AS, and MS; it is an interpretive illustration and not a study result. Tier 1 shows the three coexisting autoimmune conditions; Tier 2 contrasts a hypothetical conventional approach (general anesthesia with neuromuscular blockade and unplanned airway events), which is not based on patient data, with the staged plan used in this patient; Tier 3 lists the neuromuscular, axial, and neurological considerations; Tier 4 shows the outcomes observed in this patient (lowest arterial pressure 110/68 mmHg, lowest MAP 82 mmHg; no myasthenic crisis during 24 h of ICU observation). AS, ankylosing spondylitis; ICU, intensive care unit; MAP, mean arterial pressure; MG, myasthenia gravis; MS, multiple sclerosis.

4.5. Strengths

The strengths of this report are its documentation and its reasoning. The intraoperative record covers nine epochs from the pre-spinal baseline (T₀) to T+120 min, so the hemodynamic and respiratory course can be followed. The reasoning behind Plan A and Plan B is stated explicitly. It includes what was prepared and what was not used. Neonatal data are reported in the same detail as the maternal data. The magnesium safety measures are described. Written informed consent and ethics approval were obtained for the report.

4.6. Limitations

This is a report of a single patient at a single center, without a comparator, so no conclusion about efficacy, safety, or causation can be drawn. The earlier report of what appears to be the same patient (see the Introduction) means that this is not an independent observation. No quantitative neuromuscular monitoring and no serum magnesium levels were reported. Exposure to magnesium sulfate and to antenatal dexamethasone before delivery was brief. Neuroprotection of the infant was not assessed. Maternal follow-up ended on postoperative day 1, which is too early to detect an MS relapse or a delayed neuromuscular event,

and neonatal follow-up was also short. A resident performed the spinal, and one successful puncture in AS does not allow conclusions about the technique. The intraoperative values were recorded at 15-minute intervals. The transient tachypnea could not be explained. Several inferences in Section 4.3 rest on mechanisms that were not measured. For these reasons, the report may inform practice but cannot establish it.

4.7. Recommendations for future work

Further case reports and small series of anesthetic management in patients with MG combined with AS or MS would help to show how often the problems seen here occur and how they were handled. Reports should state magnesium dose and duration, serum magnesium level when available, quantitative neuromuscular monitoring, operator experience, and follow-up beyond the first postoperative day, including relapse and neonatal outcome. Comparative data would be more informative than extrapolation from non-obstetric cohorts. They are needed on neuraxial technique, magnesium use, and rocuronium with sugammadex in pregnant women with MG.

5. Conclusion

In this 34-year-old parturient with myasthenia gravis, ankylosing spondylitis, and multiple sclerosis at 30 weeks and 2 days of gestation, emergency cesarean delivery was performed under single-shot spinal anesthesia with 0.5% hyperbaric bupivacaine (10 mg) and fentanyl (25 mcg) at L4-L5. The prepared airway-rescue plan (rocuronium, sugammadex, and videolaryngoscopy) was not needed. Magnesium sulfate was given under continuous respiratory monitoring. Intraoperative blood pressure and heart rate remained stable without a vasopressor, and one transient rise in respiratory rate resolved with oxygen. No myasthenic crisis occurred. The 1,415 g infant recovered from an initial Apgar score of 5 and a Downes score of 5 on nasal CPAP, and the mother was transferred to the ward on postoperative day 1. These observations come from one patient. Follow-up was short and there was no comparator, so they describe this plan and its outcome and do not show that the approach is better than others.

6. Declarations

Ethical approval and consent to participate. This study was reviewed and approved by CMHC Research Center, Indonesia (Reference number 2026/0971). Patient care followed the principles of the Declaration of Helsinki and its later amendments, and the report follows the Committee on Publication Ethics (COPE) standards. Written informed consent for treatment was obtained from the patient.

Consent for publication. Written informed consent for publication of this report and its clinical data and figures was obtained from the patient and her legal guardian. A copy is on file and is available to the journal editor on request.

Availability of data and materials. The clinical, laboratory, and physiological data that support this report are included in the article and its tables and figures. Additional de-identified data files are available from the corresponding author on reasonable academic request.

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