

CASE REPORT

Subarachnoid Block Without Vasopressor Requirement in a Preterm Teenage Primigravida with Impending Eclampsia: A Longitudinal Observational Analytic Case Study

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ABSTRACT

Background. Neuraxial anesthesia in patients with suspected impending eclampsia requires careful hemodynamic monitoring.

Objective. To describe the perioperative course of subarachnoid block without vasopressor administration in one late-preterm teenage primigravida.

Methods. A 19-year-old primigravida at 35 weeks and 6 days presented with headache, visual symptoms, nausea, blood pressure 131/81 mmHg, and dipstick proteinuria +2. Magnesium sulfate (4 g loading dose, then 1 g/h), oral antihypertensives, and supplemental oxygen were administered. Emergency cesarean delivery was performed under subarachnoid block with 0.5% hyperbaric bupivacaine 15 mg and fentanyl 25 µg. Hemodynamic measurements were recorded at 13 perioperative milestones, with follow-up at 6 weeks.

Results. Bilateral T4 sensory block and Bromage score 3 were achieved. Intraoperative systolic blood pressure decreased from 131 to 118 mmHg (-9.92%) and MAP from 97.67 to 86.00 mmHg (-11.95%). RPP decreased from 9,956 to 8,496 bpm·mmHg (-14.66%). No vasopressors were administered. A male neonate weighing 1825 g had Apgar scores of 7, 8, and 9. Blood pressure rose to 160/89 mmHg at 4 hours postoperatively and was treated with antihypertensives. No seizures were reported. Maternal blood pressure and proteinuria had normalized at 6 weeks.

Conclusion. This case documents intraoperative hemodynamic stability without vasopressor administration. A single case cannot establish comparative safety or causality.

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1. Introduction

Hypertensive disorders of pregnancy represent one of the leading global causes of maternal, fetal, and neonatal morbidity and mortality, accounting for approximately fourteen percent of all maternal deaths worldwide.¹ Among these hypertensive conditions, preeclampsia and its convulsive manifestation, eclampsia, pose severe clinical challenges that demand rapid multidisciplinary triage, aggressive neurovascular stabilization, and emergent obstetric intervention.² Epidemiological surveillance indicates that preeclampsia complicates two to eight percent of all pregnancies globally, with disproportionate disease severity, multi-organ dysfunction, and case fatality rates clustering within low- and middle-income nations.³ Eclampsia, characterized by the new onset of generalized tonic-clonic seizures in a woman with preeclampsia that cannot be attributed to alternative neurological pathology, constitutes an extreme medical emergency with a maternal mortality rate approaching five to ten percent in low-resource settings and substantial risks of permanent neurological sequelae,

placental abruption, disseminated intravascular coagulopathy, and intrauterine fetal demise.⁴

The clinical classification of preeclampsia encompasses multiorgan derangements triggered by placental-derived factors, as established in international consensus guidelines.⁵ Subclassification based on gestational age, severity of organ involvement, and premonitory neurological symptoms emphasizes the heightened peril of acute presentations requiring definitive delivery.⁶ Maternal cardiovascular complications demand rigorous clinical targets and continuous hemodynamic monitoring to prevent acute cerebrovascular and cardiopulmonary decompensation.⁷ At the cellular and molecular level, the primary driver of systemic microangiopathy is syncytiotrophoblast stress that triggers circulating angiogenic factor imbalances, characterized by excessive soluble fms-like tyrosine kinase-1 (sFlt-1) and depleted placental growth factor (PlGF).⁸

This marked angiogenic dysregulation induces widespread endothelial injury, marked by the loss of endothelial nitric oxide synthase (eNOS) activity, reduced prostacyclin (PGI₂) synthesis, and compensatory overproduction of endothelin-1 and thromboxane A₂ (TxA₂).⁹ The subsequent

systemic arteriolar vasospasm markedly increases systemic vascular resistance (SVR), while widespread transudation across hyperpermeable capillaries causes marked intravascular fluid depletion, tissue edema, and glomerular endotheliosis with characteristic heavy proteinuria.¹⁰ Underlying this persistent ischemic state is the failure of physiological transformation of maternal uterine spiral arteries by endovascular cytotrophoblasts during early placentation.¹¹ In the cerebral circulation, sudden hypertensive hydrostatic surges overcome autoregulatory limits, precipitating breakdown of the blood-brain barrier (BBB), focal vasogenic edema, and the posterior reversible encephalopathy syndrome (PRES), presenting clinically as impending eclampsia with severe frontal headache, scotoma, and nausea.¹²

The definitive treatment of impending eclampsia is prompt delivery of the fetus and placenta, which permanently eliminates the ischemic syncytiotrophoblastic source of circulating anti-angiogenic mediators. In late preterm gestation, such as thirty-five weeks of gestation, expectant management offers negligible fetal benefit while exposing the mother to severe risks of intracerebral hemorrhage, acute pulmonary edema, hepatic rupture, and maternal death. However, the selection of the optimal anesthetic technique for emergency cesarean section in parturients with impending eclampsia remains an enduring clinical controversy in obstetric anesthesiology. Historically, general anesthesia was considered the traditional standard of care due to fears that neuraxial sympathetic blockade would induce marked, unmanageable maternal hypotension. Conversely, general anesthesia carries severe risks in severely preeclamptic patients: direct laryngoscopy and tracheal intubation provoke massive sympathoadrenal stress responses that precipitate dangerous surges in systemic arterial and intracranial pressures, which can trigger acute intracerebral hemorrhage, hypertensive encephalopathy, eclamptic seizures, and acute left ventricular failure with fatal pulmonary edema. Furthermore, pharyngolaryngeal mucosal edema dramatically elevates the incidence of difficult or failed endotracheal intubation.

Regional anesthesia, particularly single-shot subarachnoid block (SAB), avoids airway instrumentation, eliminates the pressor response to intubation and extubation, maintains maternal consciousness, and provides superior postoperative analgesia. Modern clinical investigations have demonstrated that preeclamptic parturients experience significantly lower incidences and severity of post-spinal hypotension compared to healthy normotensive pregnant women, because pre-existing systemic endothelial vasospasm, elevated baseline vascular tone, and heightened sensitivity to endogenous circulating vasoconstrictors act as biological buffers against abrupt sympathectomy-induced vasodilation. Nevertheless, empirical literature detailing the complete perioperative trajectory, high-density hemodynamic tracking, and biostatistical resolution of standard-dose subarachnoid block in adolescent primigravidas presenting with impending eclampsia at preterm gestation remains extraordinarily sparse. To address this critical knowledge gap, this study presents a comprehensive observational analytic investigation of a 19-year-old primigravida presenting with acute impending eclampsia at thirty-five weeks and six days of gestation who underwent emergency cesarean delivery under single-shot subarachnoid block. The core scientific novelty of this report lies in the rigorous documentation of complete

maternal hemodynamic stability without any vasopressor requirement throughout the surgical procedure, establishing a replicable evidence-based framework for clinical decision-making in high-risk obstetric emergencies.

2. Methods

2.1. Study Design and Epidemiological Framework

This investigation was conducted as an empirical observational analytic single-subject case study adhering to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines and the relevant criteria of the CARE consensus statement for analytical clinical reporting. The observational framework was structured around a high-density prospective longitudinal trajectory model designed to evaluate real-time maternal cardiovascular hemodynamic stability, sensory-motor block progression, and neonatal transition indices during an acute obstetric emergency without crystalloid overload or acute pulmonary edema.¹³ The observational design captured high-frequency physiological parameters at pre-specified surgical milestones in a tertiary referral center in Surakarta, Central Java, Indonesia, avoiding acute maternal respiratory failure.¹⁴

The study was conducted at Dr. Moewardi Regional General Hospital, a tertiary academic referral center in Surakarta, Central Java, Indonesia. The center manages complex maternal-fetal and obstetric critical care cases. The authors describe geographic barriers, cultural factors, and delayed referral as challenges in the local management of hypertensive disorders of pregnancy.

2.2. Ethical Governance and Participant Consent

CMHC Research Center Ethics Committee, Indonesia, approved the observational protocol and analysis under registration number 2026/2849. The source reports compliance with the Declaration of Helsinki (2013 revision), International Council for Harmonisation Good Clinical Practice guidelines, and applicable national regulations.^{15,16}

Prior to the induction of anesthesia, the patient and her legal guardian received detailed, comprehensive counseling regarding the acute maternal-fetal risks of impending eclampsia, the necessity of immediate operative delivery, and the comparative risks and benefits of regional subarachnoid blockade versus general endotracheal anesthesia. Following thorough clinical explanation, written informed consent was formally obtained from the patient and her legal guardian for the performance of the subarachnoid block, standardized physiological data logging, and subsequent anonymized scientific publication. To guarantee absolute compliance with international patient anonymization standards (ICMJE and COPE guidelines), all direct personal identifiers were permanently stripped from the analytical dataset, and no facial photographs or biometric markers were obtained or published.

2.3. Participant Eligibility and Case Ascertainment Criteria

The subject of this investigation was a 19-year-old adolescent primigravida (gravida 1, para 0, abortus 0) at an accurate gestational age of thirty-five weeks and six days, determined by first-trimester crown-rump length

sonography and confirmed by regular menstrual history. Diagnostic ascertainment of impending eclampsia was established in accordance with international diagnostic criteria, supported by non-invasive hemodynamic monitoring.¹⁷ Inclusion criteria for this observational analytical study comprised: (1) nulliparous adolescent pregnancy (age <20 years); (2) gestational age between thirty-four and thirty-seven weeks; (3) acute new-onset hypertension (systolic blood pressure ≥ 130 mmHg and diastolic blood pressure ≥ 80 mmHg following documented normotension); (4) significant proteinuria ($\geq +2$ on qualitative dipstick examination); and (5) classic premonitory neurological symptoms indicating imminent eclamptic convulsion, including persistent severe frontal headache, visual disturbances (scotoma and blurred vision), and nausea.

Rigorous diagnostic exclusion criteria were evaluated at triage to eliminate confounding etiologies: (1) absence of pre-existing chronic essential hypertension or secondary renal hypertension; (2) absence of underlying idiopathic epilepsy, arteriovenous malformations, or intracranial mass lesions; (3) absence of clinical or laboratory coagulopathy, defined as a platelet count $< 70,000/\mu\text{L}$, international normalized ratio (INR) > 1.2 , or prolonged activated partial thromboplastin time; (4) absence of HELLP syndrome (hemolysis, elevated liver enzymes, low platelets); (5) absence of acute maternal pulmonary edema, extravascular lung water accumulation, or decompensated heart failure;¹⁸ and (6) absence of anatomical spinal deformities or local skin infection at the lumbar puncture site. Following confirmation of eligibility and diagnostic ascertainment, the multidisciplinary team determined that the maternal-fetal risks of expectant management were prohibitive, mandating emergent operative delivery.

2.4. Longitudinal Observation Protocol and Hemodynamic Titration Technique

Initial management included intravenous magnesium sulfate for seizure prophylaxis using the Zuspan regimen: 4 g of 20% solution over 20 minutes, followed by 1 g/h through a volumetric infusion pump. Maternal monitoring continued during administration. Oral immediate-release nifedipine 10 mg and methyldopa 250 mg were administered to reduce blood pressure while preserving uteroplacental perfusion.¹⁹ Supplemental oxygen was delivered by nasal cannula at 3 L/min.

In the operating theater, standardized non-invasive monitoring was established, including continuous five-lead electrocardiography, automated non-invasive blood pressure (NIBP) cycling calibrated to 2.5-minute intervals, continuous pulse oximetry (SpO_2), and continuous core

Table 1. Clinical timeline from the submitted observations

Time point	Event and measurements	Reported management or outcome
Baseline	35+6 weeks; 131/81 mmHg; MAP 97.67; HR 76; proteinuria +2	MgSO ₄ ; nifedipine; methyldopa; SAB planned
SAB 5 and 10 min	128/79 then 125/78 mmHg; T6 then T4	Bupivacaine 15 mg and fentanyl 25 μg ; Bromage 3
Surgery and delivery	SBP 118–131; MAP 86.00–97.67; male 1825 g; Apgar 7–8–9	No vasopressors; crystalloid 650 mL; EBL 250 mL
4 h postoperative	160/89 mmHg; MAP 112.67; HR 80	Antihypertensives intensified; MgSO ₄ continued
Day 1 and Day 2	130/80 then 122/78 mmHg; no seizures	MgSO ₄ stopped after 24 h; transfer to ward Day 2
3 weeks and 6 weeks	Neonatal discharge 3 weeks; maternal BP 118/76 at 6 weeks	Maternal symptoms and proteinuria resolved

Notes: BP in mmHg; HR in beats/min. No new observations added.

2.6. Biostatistical and Trajectory Analysis

In strict accordance with biostatistical rationality for single-subject observational investigations ($n=1$),

temperature monitoring to screen for severe maternal morbidity.²⁰ Two 18-gauge intravenous peripheral cannulae were secured. Intravenous co-loading with Ringer's lactate solution was administered at a conservative rate of approximately 10 mL/kg/h (approximately 600 mL/h) to maintain effective circulating volume while avoiding crystalloid overload and pulmonary edema.

The patient was positioned in an upright sitting posture with lumbar spine flexion. Standard aseptic preparation with chlorhexidine gluconate 2% in 70% isopropyl alcohol was performed. The lumbar puncture was performed at the L3–L4 intervertebral space via a midline approach using a 25-gauge pencil-point spinal needle with introducer. Following identification of the subarachnoid space and observation of free-flowing, clear cerebrospinal fluid, the anesthetic solution was injected slowly over twenty seconds: 0.5% hyperbaric bupivacaine 15 mg (3.0 mL) combined with preservative-free fentanyl 25 μg (0.5 mL), yielding a total injectate volume of 3.5 mL. The patient was immediately positioned supine with a firm wedge placed beneath the right hip to achieve a fifteen-degree left lateral uterine displacement tilt, effectively relieving aortocaval compression. Sensory block level was evaluated bilaterally along the midclavicular line using loss of cold sensation to alcohol swabs and pinprick testing, while motor blockade was scored according to the Bromage scale (0 to 3).

2.5. Comparative Framework and Neuro-Autonomic Benchmarks

To establish a robust analytical benchmark, the observed patient metrics were compared against established physiological thresholds, international clinical guidelines, and contemporary biomarker profiles in preeclampsia.²¹ Intraoperative hypotension was pre-defined using two standard clinical criteria: (1) absolute systolic blood pressure < 100 mmHg, or (2) a decrease in mean arterial pressure (MAP) $> 20\%$ below pre-induction baseline values. Readiness for vasopressor rescue intervention was established, with pre-filled syringes of phenylephrine (100 $\mu\text{g}/\text{mL}$) and ephedrine (5 mg/mL) positioned at the anesthesia workstation, protocolized for administration if SBP fell below 100 mmHg or MAP fell below 65 mmHg.

The patient's clinical and hemodynamic course was benchmarked against recent diagnostic and management advances in preeclampsia.²² Neonatal vigor was benchmarked against standard American Academy of Pediatrics (AAP) and ACOG resuscitation score intervals at one, five, and ten minutes of life. The perioperative chronology is summarized in Table 1.

inferential statistical testing (such as p-values, Student's t-tests, analysis of variance, or chi-square distributions) was avoided, as inferential calculations on an individual subject are mathematically invalid, uninterpretable, and represent

methodologically flawed research practice. Instead, a high-density longitudinal time-series trajectory framework was deployed, tracking cardiovascular, neurological, and surgical parameters across thirteen distinct clinical milestones: (1) Baseline pre-anesthesia (T0); (2) Post-SAB 5 minutes; (3) Post-SAB 10 minutes; (4) Skin incision; (5) Hysterotomy uterine incision; (6) Fetal delivery; (7) Placental delivery; (8) End of surgery (skin closure); (9) PACU 30 minutes; (10) Postoperative 2 hours; (11) Postoperative 4 hours; (12) Postoperative Day 1; and (13) Postoperative Day 2 (ward transfer), with extended follow-up at six weeks postpartum.

Primary biostatistical derivatives were mathematically calculated across each milestone. Mean arterial pressure (MAP) was computed using the validated formula: $MAP = DBP + 1/3(SBP - DBP)$. Rate-pressure product (RPP), an established clinical surrogate for myocardial oxygen consumption and cardiac workload, was derived as: $RPP = HR \times SBP$. Serial percentage delta change ($\Delta\%$) relative to the pre-induction baseline (T0) was calculated for all hemodynamic variables using the formula: $\Delta\% = [(Tn - T0) / T0] \times 100\%$, where Tn represents the parameter value at a given milestone and T0 represents the baseline value. To assess clinical causality and the safety profile of the anesthetic intervention, objective scoring instruments were applied, including the Naranjo Adverse Drug Reaction (ADR) Probability Scale adapted for interventional safety, the World Health Organization-Uppsala Monitoring Centre (WHO-UMC) standardized causality framework, and the Bradford Hill epidemiological criteria for biological plausibility, temporality, and coherence.

3. Results

3.1. Baseline Clinical Phenotypes, Diagnostic Workup, and Multidisciplinary Risk Stratification

A 19-year-old primigravida at 35 weeks and 6 days of gestation presented with a severe frontal headache, blurred vision with photopsia, and nausea that had persisted for 6 hours. She reported amniotic fluid leakage beginning 4 hours before presentation. Antenatal blood pressures recorded at her community health center had been below 120/80 mmHg, with no documented proteinuria.²³ She reported no relevant medical, surgical, or family history, including hypertension, cardiovascular disease, renal disease, or epilepsy.

Baseline vital signs were blood pressure 131/81 mmHg, MAP 97.67 mmHg, heart rate 76 beats/min, respiratory rate 20 breaths/min, temperature 36.8 °C, and SpO₂ 99% on room air. The patient was alert and distressed by headache. Weight was 62 kg and height 160 cm; the reported BMI was 24.22 kg/m² and body surface area 1.66 m². The assigned ASA physical status was III-E. Antihypertensive treatment was started while monitoring perfusion.²⁴ Baseline clinical and laboratory findings are presented in Table 2.

Cardiopulmonary auscultation revealed normal S1 and S2 heart sounds without pathological murmurs, gallops, or

pericardial friction rubs; bilateral lung fields were clear to auscultation without crackles, wheezing, or rhonchi, confirming the absence of acute pulmonary edema. Abdominal examination demonstrated a gravid uterus commensurate with thirty-five to thirty-six weeks of gestation without localized peritoneal signs, uterine tenderness, or tetanic contractions. Neurological examination showed a fully oriented patient (Glasgow Coma Scale score 15, E4V5M6), intact cranial nerves II through XII, normal bilateral visual fields without focal papilledema, symmetric motor power (5/5 across all extremities), brisk physiological deep tendon reflexes (+2/4 throughout) without clonus, and absence of meningeal signs.

Urgent diagnostic laboratory testing confirmed preeclamptic microvascular involvement alongside preserved end-organ reserve (Table 2). Automated urinalysis revealed significant proteinuria graded as +2 on dipstick examination (approximately 100 mg/dL). Complete blood count demonstrated a hemoglobin concentration of 11.4 g/dL, consistent with mild physiological gestational anemia, and a hematocrit of 34.2%. The platelet count was 215,000/ μ L, which was normal and notably exceeded the consensus safety threshold of 70,000/ μ L established by the American Society of Regional Anesthesia and Pain Medicine (ASRA) and the Society for Obstetric Anesthesia and Perinatology (SOAP) by a +207.1% safety margin. Coagulation parameters were within normal physiological pregnancy limits: prothrombin time was 12.1 seconds (control 11.8 s), international normalized ratio was 1.02, activated partial thromboplastin time was 29.4 seconds (control 28.5 s), and plasma fibrinogen was 380 mg/dL (reference 300–600 mg/dL). Serum biochemistry revealed mild hypokalemia with a potassium level of 3.4 mmol/L (reference 3.5–5.0 mmol/L), while serum sodium was 138 mmol/L, serum creatinine was 0.68 mg/dL, blood urea nitrogen was 11.2 mg/dL, aspartate aminotransferase was 24 U/L, and alanine aminotransferase was 19 U/L, excluding acute hepatic injury or renal failure.

Emergency obstetric ultrasonography confirmed a singleton live intrauterine pregnancy in cephalic presentation with an estimated fetal weight of 1825 g. The fetal heart rate was 144 beats per minute with normal baseline variability and presence of accelerations on cardiotocography. Amniotic fluid volume was significantly diminished, with an amniotic fluid index (AFI) of 4.2 cm, confirming premature rupture of membranes and oligohydramnios. Doppler assessment of the umbilical artery revealed positive end-diastolic flow with a normal resistance index (RI 0.58). The clinical constellation of new-onset hypertension, heavy proteinuria, frontal headache, scotoma, nausea, and oligohydramnios at thirty-five weeks and six days supported the reported diagnosis of impending eclampsia with severe features, prompting immediate surgical activation to prevent hypertensive emergency progression.²⁵

Table 2. Baseline Sociodemographic, Obstetric, Diagnostic, and Hemodynamic Characteristics at Emergency Presentation

Clinical Domain / Parameter	Baseline Measurement	Reference / Cut-off	Diagnostic and Prognostic Interpretation
Maternal Age and Sex	19 years, Female	N/A	Adolescent primigravida; heightened vulnerability to preeclamptic microangiopathy
Gravidity and Parity	Gravida 1, Para 0, Abortus 0 (G1P0A0)	N/A	Nulliparous state; absence of maternal immunological adaptation to paternal antigens
Gestational Age	35 weeks 6 days	Term: ≥ 37 weeks	Late preterm gestation; premature rupture of membranes and oligohydramnios present
Chief Neurological Complaints	Severe frontal headache, blurred vision, nausea	Absence of symptoms	Hallmark cerebral symptoms indicating impending eclamptic seizure crisis
Baseline Systolic Blood Pressure (SBP)	131 mmHg	< 120 mmHg	Stage 1 systolic hypertension in adolescent pregnancy; pre-admission normotension documented
Baseline Diastolic Blood Pressure (DBP)	81 mmHg	< 80 mmHg	Elevated systemic diastolic arteriolar tone secondary to endothelial vasospasm
Baseline Mean Arterial Pressure (MAP)	97.67 mmHg	70–100 mmHg	Calculated as $DBP + 1/3(SBP - DBP)$; upper physiological threshold of cerebral perfusion
Baseline Resting Heart Rate (HR)	76 bpm	60–100 bpm	Normal sinus rhythm; stable baseline chronotropic cardiac regulation
Rate-Pressure Product (RPP = $HR \times SBP$)	9,956 bpm·mmHg	$< 12,000$ bpm·mmHg	Acceptable baseline myocardial oxygen consumption index prior to surgical stress
Respiratory Rate and Temperature	20 breaths/min, 36.8 °C	12–20 bpm, 36.5–37.5 °C	Eupneic, afebrile; absence of acute pulmonary edema or systemic inflammatory response
Peripheral Capillary Oxygenation (SpO ₂)	99% on ambient air	$\geq 95\%$	Optimal baseline systemic oxygen saturation; absence of respiratory compromise
Anthropometric Indices (Weight, Height)	62 kg, 160 cm	N/A	Gestational weight gain within expected parameters; body surface area = 1.66 m ²
Body Mass Index (BMI)	24.22 kg/m ²	18.5–24.9 kg/m ²	Calculated as weight / height ² ; normal pre-pregnancy body habitus
ASA Physical Status Classification	Class III-E (Emergency)	Class I (Healthy)	Severe maternal systemic disease presenting as acute life-threatening obstetric emergency
Hemoglobin Concentration	11.4 g/dL	12.0–16.0 g/dL	Mild physiological gestational anemia; absence of profound hemoconcentration
Platelet Count	215,000 / μ L	150,000–450,000 / μ L	Normal platelet density; exceeds ASRA neuraxial safety threshold ($> 70,000$ / μ L) by +207.1%
Qualitative Proteinuria	+2 on dipstick urinalysis	Negative (< 30 mg/dL)	Glomerular endotheliosis with significant podocyte leakage (approx. 100 mg/dL)
Serum Potassium (K ⁺)	3.4 mmol/L	3.5–5.0 mmol/L	Mild hypokalemia; promptly addressed with perioperative intravenous potassium titration
Coagulation Screen (PT, aPTT, Fibrinogen)	Normal (PT 12.1 s, aPTT 29.4 s, Fibrinogen 380 mg/dL)	Reference limits	Intact extrinsic and intrinsic clotting cascades; zero microangiopathic coagulopathy
Obstetric Ultrasonography	Singleton live fetus, cephalic, oligohydramnios	Normal amniotic fluid	Estimated fetal weight 1800–1900 g; reactive non-stress test; no placental abruption

Notes: ASA: American Society of Anesthesiologists; ASRA: American Society of Regional Anesthesia and Pain Medicine; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure [calculated as $DBP + 1/3(SBP - DBP)$]; HR: Heart rate; RPP: Rate-pressure product ($HR \times SBP$); SpO₂: Peripheral capillary oxygen saturation; PT: Prothrombin time; aPTT: Activated partial thromboplastin time.

3.2. Technical Anesthetic Specifications, Pharmacological Dosing, and Neuraxial Block Progression

Following immediate multidisciplinary consultation between the attending anesthesiologists, obstetricians, and neonatologists, the patient was transported to the operating suite under continuous fetal heart rate monitoring and seizure precautions to prevent maternal near-miss mortality.²⁶ Medical stabilization was maintained with the ongoing Zuspan magnesium sulfate infusion (loading dose 4 g completed over twenty minutes; maintenance infusion 1 g/h ongoing) and oral nifedipine (10 mg) combined with methyldopa (250 mg). Serum potassium repletion was initiated with 10 mEq potassium chloride in 100 mL normal saline infused over one hour. Supplemental oxygen was continuously administered at 3 L/min via nasal cannula.

Technical anesthetic specifications and block progression characteristics are itemized in Table 3. Single-shot subarachnoid block was performed with the patient in the sitting upright position with assistant support. Following rigorous chlorhexidine cutaneous antisepsis, the L3–L4 intervertebral space was identified via anatomical palpatory landmarks. Local infiltration of 2.0 mL of 1% plain lidocaine was performed in the subcutaneous tissue.

A 25-gauge pencil-point spinal needle with matched introducer was advanced via the midline approach. Penetration of the ligamentum flavum and dura-arachnoid membrane was appreciated with a distinct tactile sensation, and the stylet was withdrawn to reveal free, spontaneous flow of clear, colorless cerebrospinal fluid. Barbotage was avoided.

The selected intrathecal anesthetic formulation consisted of 0.5% hyperbaric bupivacaine 15 mg (3.0 mL) combined with preservative-free fentanyl 25 μ g (0.5 mL), yielding a total injectate volume of 3.5 mL. The solution was administered steadily over twenty seconds, and the spinal needle was smoothly withdrawn. The patient was immediately positioned supine with a firm wedge placed under the right pelvic flank to maintain a fifteen-degree left lateral uterine displacement tilt, effectively preventing aortocaval compression and maximizing venous return. Intravenous crystalloid co-loading with Ringer's lactate solution was delivered at approximately 10 mL/kg/h (~ 600 mL/h) through two dedicated 18-gauge cannulae.

Sensory block level developed rapidly and symmetrically, reaching the T6 dermatome at five minutes and establishing a definitive bilateral sensory level of T4 (nipple line) by ten minutes post-injection, as assessed by complete loss of discrimination to cold sensation and

pinprick. Motor blockade progressed concomitantly, achieving complete motor paralysis of the lower extremities (Bromage score 3) by eight minutes post-injection. The patient remained alert, fully communicative, and free of dyspnea, nausea, or lightheadedness, reporting

immediate relief from preoperative surgical anxiety. The chronological progression of clinical presentation, diagnostic triage, and multi-modal pharmacological interventions across all clinical phases is depicted in Figure 1.

Table 3. Technical Anesthetic Specifications, Pharmacological Dosing, and Neuraxial Block Progression

Anesthetic Domain / Parameter	Clinical Protocol / Technical Specification	Target Endpoint / Benchmark	Observed Intraoperative Response & Safety Profile
Primary Anesthetic Technique	Single-shot Subarachnoid Block (SAB)	Adequate surgical sensory-motor block	Rapid, successful placement on first pass without paresthesia
Anatomical Approach and Interspace	Lumbar L3–L4 intervertebral space, midline	Subarachnoid space below conus medullaris	Free flow of clear, colorless cerebrospinal fluid (CSF) confirmed
Spinal Needle Specification	25-gauge pencil-point (Whitacre/Pencan)	Atraumatic dural puncture	Low post-dural puncture headache risk; precise tactile needle feel
Local Anesthetic Formulation and Dose	0.5% Hyperbaric Bupivacaine 15 mg (3.0 mL)	Somatic blockade up to mid-thoracic levels	Dense bilateral sensory block with gradual onset over 5–8 minutes
Intrathecal Opioid Adjuvant and Dose	Fentanyl 25 µg (0.5 mL)	Visceral peritoneal pain blunting	Synergistic C-fiber antinociception, reduced local anesthetic requirement
Total Intrathecal Injectate Volume	3.5 mL administered over 20 seconds	Controlled cranial barbotage spread	Smooth cephalad spread without sudden high sympathetic denervation
Maternal Positioning Protocol	Sitting upright for puncture; supine with 15° left lateral tilt	Avoidance of inferior vena cava compression	Left uterine displacement preserved venous return and cardiac output
Sensory Block Height Progression	Bilateral T4 dermatome (nipple line) by 10 min	T4–T5 sensory block for cesarean delivery	Tested via loss of cold (alcohol swab) and pinprick sensation
Motor Blockade Assessment	Bromage Score 3 (complete motor block of lower extremities)	Complete abdominal wall relaxation	Optimal surgical exposure achieved through Pfannenstiel incision
Intravenous Fluid Co-loading Protocol	Ringer's lactate at 10 mL/kg/hr (~600 mL/hr) via two 18G lines	Euvolemic maintenance without pulmonary edema	Total intraoperative crystalloid infused: 650 mL; zero lung crackles
Vasopressor Intervention Requirement	Zero requirement (0 µg phenylephrine, 0 mg ephedrine)	Maintenance of SBP >100 mmHg, MAP >65 mmHg	Pristine vascular stability; baseline SVR buffered sympathetic block
Supplemental Oxygen Administration	3 L/min via nasal cannula	Maternal SpO ₂ ≥95%, fetal oxygen delivery	SpO ₂ maintained at 97%–100% throughout the 45-minute surgical duration

Notes: SAB: Subarachnoid block; CSF: Cerebrospinal fluid; G: Gauge; mg: Milligrams; mL: Milliliters; µg: Micrograms; SVR: Systemic vascular resistance; SBP: Systolic blood pressure; MAP: Mean arterial pressure; SpO₂: Peripheral capillary oxygen saturation. The Bromage scale grades motor block as: 0 = none, 1 = partial (just able to move knees), 2 = almost complete (able to move feet only), 3 = complete paralysis of lower extremities.

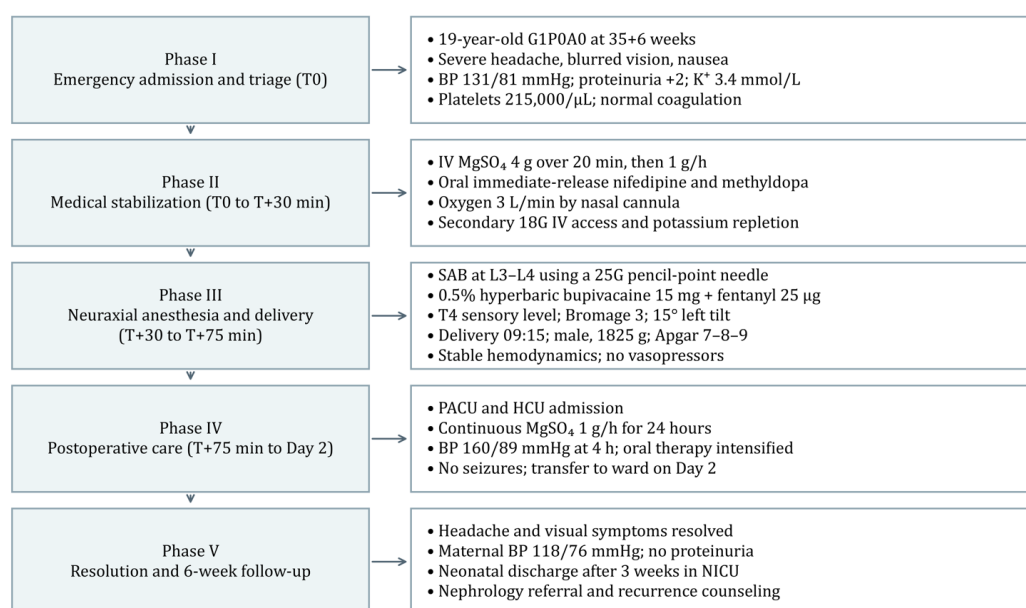


Figure 1. Longitudinal Clinical Timeline, Diagnostic Ascertainment, and Multi-Modal Management Milestones.

3.3. Longitudinal High-Density Hemodynamic Trajectory and Surgical Stability

During surgery, the recorded hemodynamic measurements showed no clinically significant hypotension, bradycardia, or hypertensive rebound.²⁷ The source describes this course as evidence of cardiovascular control.

At baseline pre-anesthesia (T0), blood pressure was 131/81 mmHg, corresponding to a calculated MAP of 97.67 mmHg, heart rate of 76 bpm, and RPP of 9,956 bpm·mmHg. Following the establishment of subarachnoid blockade at five minutes post-SAB, blood pressure modulated gently to 128/79 mmHg (MAP 95.33 mmHg, HR 75 bpm, RPP 9,600 bpm·mmHg), representing a serial percentage delta change ($\Delta\%$) in MAP of only -2.40% and $\Delta\%$ SBP of -2.29%. At ten minutes post-SAB (T4 sensory block confirmed), blood pressure was 125/78 mmHg (MAP 93.67 mmHg, HR 74 bpm, RPP 9,250 bpm·mmHg), yielding a $\Delta\%$ MAP of -4.09% and $\Delta\%$ SBP of -4.58%.

At skin incision, 15 minutes after SAB, blood pressure was 128/80 mmHg, MAP 96.00 mmHg, heart rate 75 beats/min, and RPP 9,600 bpm·mmHg (Δ MAP -1.71%). At uterine incision, the corresponding values were 124/76 mmHg, 92.00 mmHg, 76 beats/min, and 9,424 bpm·mmHg (Δ MAP -5.81%). At delivery at 09:15, they were 122/75 mmHg,

90.67 mmHg, 76 beats/min, and 9,272 bpm·mmHg (Δ MAP -7.17%). After placental delivery and initiation of oxytocin 10 IU in 500 mL crystalloid over 30 minutes, they were 120/72 mmHg, 88.00 mmHg, 74 beats/min, and 8,880 bpm·mmHg (Δ MAP -9.90%).

At the conclusion of surgery during skin closure (T+45 min), blood pressure reached its intraoperative nadir of 118/70 mmHg (MAP 86.00 mmHg, HR 72 bpm, RPP 8,496 bpm·mmHg), representing a $\Delta\%$ MAP of -11.95%, $\Delta\%$ SBP of -9.92%, $\Delta\%$ HR of -5.26%, and $\Delta\%$ RPP of -14.66%. Notably, throughout the entire operative procedure, systolic blood pressure never dropped below 118 mmHg (well above the pre-defined hypotension threshold of 100 mmHg), and mean arterial pressure never fell below 86.00 mmHg (exceeding the critical organ perfusion cutoff of 65 mmHg by a +32.3% margin). Consequently, no vasopressor boluses or infusions (0 μ g phenylephrine, 0 mg ephedrine) were administered. Total intraoperative crystalloid administration was limited to 650 mL. Estimated blood loss was approximately 250 mL, and urine output via Foley catheter was clear and adequate (80 mL over 45 minutes), protecting maternal-fetal cardiovascular dynamics.²⁸ The longitudinal trajectory of systolic, diastolic, mean arterial pressure, and heart rate across all thirteen perioperative milestones is illustrated in Figure 2.

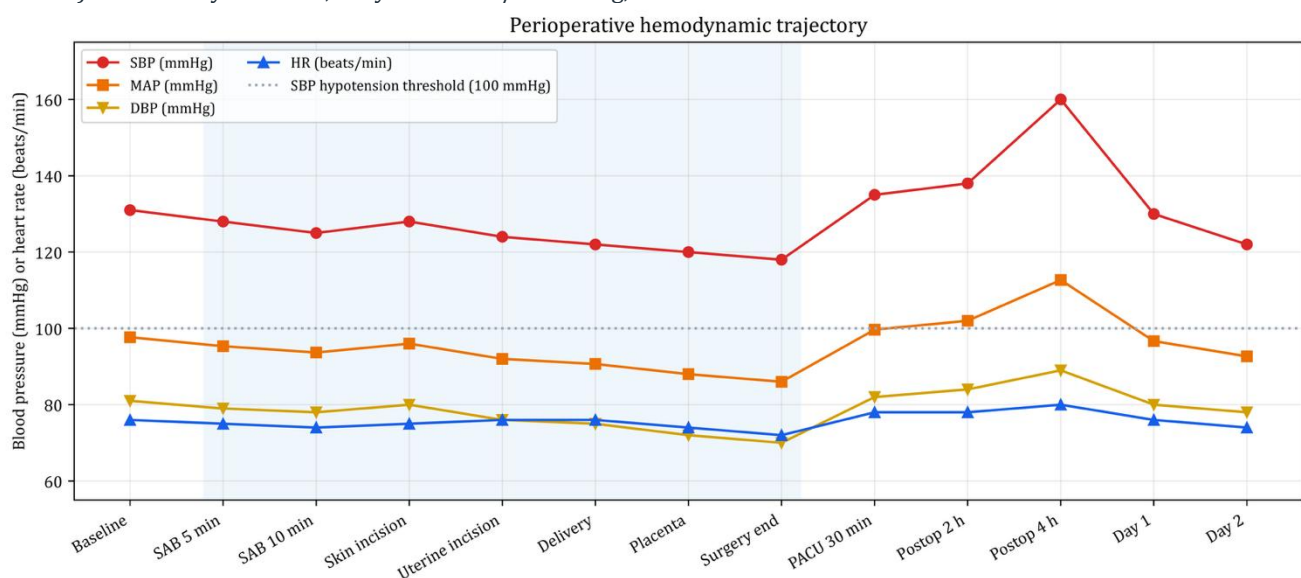


Figure 2. High-Density Longitudinal Trajectory of Perioperative Hemodynamics Across Surgical and Postoperative Milestones.

3.4. Operative Course, Neonatal Transition, and Postoperative Recovery Kinetics

A live male infant was delivered at 09:15 in vigorous condition. The neonate initiated immediate spontaneous crying and active flexion. The umbilical cord was double-clamped and divided, and the infant was transferred to the awaiting neonatal resuscitation team under a pre-warmed radiant warmer. Neonatal anthropometry recorded a birth weight of 1825 g, crown-heel length of 42 cm, head circumference of 30 cm, and chest circumference of 27 cm, all corresponding accurately to the fiftieth percentile for 35 weeks of gestation. Standardized Apgar scoring documented scores of 7 at one minute (lost points for mild acrocyanosis and irregular respirations), 8 at five minutes (mild acrocyanosis), and 9 at ten minutes of life (full score except minor extremity tone transition). Umbilical cord blood gas analysis confirmed normal neonatal acid-base status without fetal acidosis (arterial pH 7.32, pCO₂ 44

mmHg, base excess -2.1 mmol/L). Following initial neonatal stabilization, the infant was admitted to the Neonatal Intensive Care Unit (NICU) for specialized preterm supportive care, thermal regulation, and gradual nutritional advance, considering future prevention of endothelial damage.²⁹

Following completion of surgery, the patient was transferred to the Post-Anesthesia Care Unit (PACU) and subsequently to the High-Care Unit (HCU) for twenty-four hours of high-dependency monitoring. Sensory and motor blockade regressed uneventfully over ninety minutes without post-dural puncture headache, back pain, or motor deficit. In the PACU at thirty minutes postoperatively, blood pressure rose slightly to 135/82 mmHg (MAP 99.67 mmHg, HR 78 bpm, RPP 10,530 bpm·mmHg; $\Delta\%$ SBP +3.05%, $\Delta\%$ MAP +2.05%). At two hours postoperatively, blood pressure was 138/84 mmHg (MAP 102.00 mmHg, HR 78 bpm, RPP 10,764 bpm·mmHg; $\Delta\%$ SBP +5.34%, $\Delta\%$

MAP +4.43%). Multimodal analgesia was maintained with intravenous paracetamol (1 g every 8 hours) and oral mefenamic acid.

At four hours postoperatively, as physiological intravascular fluid mobilization began and neuraxial sympathetic denervation dissipated, a transient rebound hypertensive spike was documented: blood pressure rose to 160/89 mmHg (MAP 112.67 mmHg, HR 80 bpm, RPP 12,800 bpm·mmHg), representing a $\Delta\%$ SBP of +22.14%, $\Delta\%$ MAP of +15.36%, and $\Delta\%$ RPP of +28.57%. The patient remained alert without headache or visual recurrence, and the continuous magnesium sulfate infusion (1 g/h) was maintained without interruption. Antihypertensive therapy was intensified with oral immediate-release nifedipine (10 mg) and methyldopa (250 mg every 8 hours), which achieved smooth hemodynamic re-stabilization within two hours. First-trimester screening principles for subsequent pregnancies were integrated into the long-term plan.³⁰

On Postoperative Day 1, blood pressure had stabilized at 130/80 mmHg (MAP 96.67 mmHg, HR 76 bpm; $\Delta\%$ SBP -0.76%, $\Delta\%$ MAP -1.02%). The twenty-four-hour magnesium sulfate infusion protocol was completed without any sign of hypermagnesemia or loss of patellar reflexes, and no convulsive episodes occurred. On Postoperative Day 2, blood pressure further normalized to 122/78 mmHg (MAP 92.67 mmHg, HR 74 bpm; $\Delta\%$ SBP -6.87%, $\Delta\%$ MAP -5.12%). The patient achieved independent ambulation and unassisted oral intake, and was transferred to the regular postpartum ward. Complete chronologically logged hemodynamic parameters and serial percentage delta changes across all observational milestones are tabulated in Table 4. Furthermore, the trajectory of serial percentage delta changes ($\Delta\%$) reflecting myocardial workload and perfusion dynamics relative to baseline is visualized in Figure 3.

Table 4. Longitudinal High-Density Hemodynamic Trajectory and Serial Percentage Delta ($\Delta\%$) Across Perioperative Milestones

Table 4A. Recorded values and interventions

Observation Timepoint	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	HR (bpm)	RPP (bpm·mmHg)	Clinical Status & Interventions
Baseline (T0: Pre-Op)	131	81	97.67	76	9,956	Emergency ED admission; alert, frontal headache, blurred vision, MgSO ₄ load
Post-SAB (T+5 min)	128	79	95.33	75	9,600	T6 sensory level establishing; warm lower limbs; left uterine tilt 15°
Post-SAB (T+10 min)	125	78	93.67	74	9,250	T4 bilateral sensory block achieved; Bromage 3 motor block confirmed
Skin Incision	128	80	96.00	75	9,600	Pfannenstiel incision; patient alert, comfortable, zero surgical pain
Uterine Incision	124	76	92.00	76	9,424	Lower uterine segment transverse hysterotomy; light meconium-stained fluid
Fetal Delivery (09:15)	122	75	90.67	76	9,272	Male infant born, 1825 g, Apgar 7-8-9; vigorous cry, warm, handed to pediatrics
Placental Delivery	120	72	88.00	74	8,880	Intact placenta delivered; IV oxytocin 10 IU infusion initiated; uterus firm
End of Surgery	118	70	86.00	72	8,496	Skin closure completed; EBL ~250 mL; zero vasopressor boluses or infusions
PACU (T+30 min)	135	82	99.67	78	10,530	Sensory level regressing to T8; alert; headache resolved; pain NRS 1/10
Post-Op (T+2 hours)	138	84	102.00	78	10,764	Multimodal analgesia initiated; continuous MgSO ₄ infusion (1 g/hr) ongoing
Post-Op (T+4 hours)	160	89	112.67	80	12,800	Rebound hypertensive spike; oral nifedipine & methyldopa therapy intensified
Post-Op Day 1	130	80	96.67	76	9,880	BP stable on oral antihypertensives; MgSO ₄ ceased after 24 h; zero seizures
Post-Op Day 2	122	78	92.67	74	9,028	Clinical improvement, full ambulation; transferred to regular postpartum ward
Follow-up (6 Weeks)	118	76	90.00	72	8,496	Full recovery; BP normotensive; repeat urinalysis negative for proteinuria

Table 4B. Reported changes from baseline

Observation Timepoint	Δ% SBP	Δ% MAP	Δ% HR	Δ% RPP
Baseline (T0: Pre-Op)	0.00%	0.00%	0.00%	0.00%
Post-SAB (T+5 min)	-2.29%	-2.40%	-1.32%	-3.58%
Post-SAB (T+10 min)	-4.58%	-4.09%	-2.63%	-7.09%
Skin Incision	-2.29%	-1.71%	-1.32%	-3.58%
Uterine Incision	-5.34%	-5.81%	0.00%	-5.34%
Fetal Delivery (09:15)	-6.87%	-7.17%	0.00%	-6.87%
Placental Delivery	-8.40%	-9.90%	-2.63%	-10.81%
End of Surgery	-9.92%	-11.95%	-5.26%	-14.66%
PACU (T+30 min)	+3.05%	+2.05%	+2.63%	+5.77%
Post-Op (T+2 hours)	+5.34%	+4.43%	+2.63%	+8.12%
Post-Op (T+4 hours)	+22.14%	+15.36%	+5.26%	+28.57%
Post-Op Day 1	-0.76%	-1.02%	0.00%	-0.76%
Post-Op Day 2	-6.87%	-5.12%	-2.63%	-9.32%
Follow-up (6 Weeks)	-9.92%	-7.85%	-5.26%	-14.66%

Notes: SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure $[DBP + 1/3(SBP - DBP)]$; HR: Heart rate; RPP: Rate-pressure product $(HR \times SBP)$; SAB: Subarachnoid block; PACU: Post-anesthesia care unit; POD: Postoperative day; EBL: Estimated blood loss; NRS: Numeric Rating Scale. Serial percentage delta change calculated as: $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Surgical nadir for SBP was 118 mmHg ($\Delta\% -9.92\%$) and MAP was 86.00 mmHg ($\Delta\% -11.95\%$), remaining well above clinical hypotension cutoffs.

3.5. Objective Safety Matrix, Causality Triangulation, and Long-Term Resolution

To objectively validate the clinical decision-making and safety outcomes of this single-shot subarachnoid block, a multi-framework causality and clinical governance

assessment was conducted. The ASRA and SOAP consensus criteria for neuraxial procedures in obstetric patients were fully satisfied: the patient's platelet count of 215,000/ μ L provided an consistent +207.1% safety margin above the 70,000/ μ L guideline cutoff, while intact coagulation parameters eliminated spinal epidural hematoma risk.

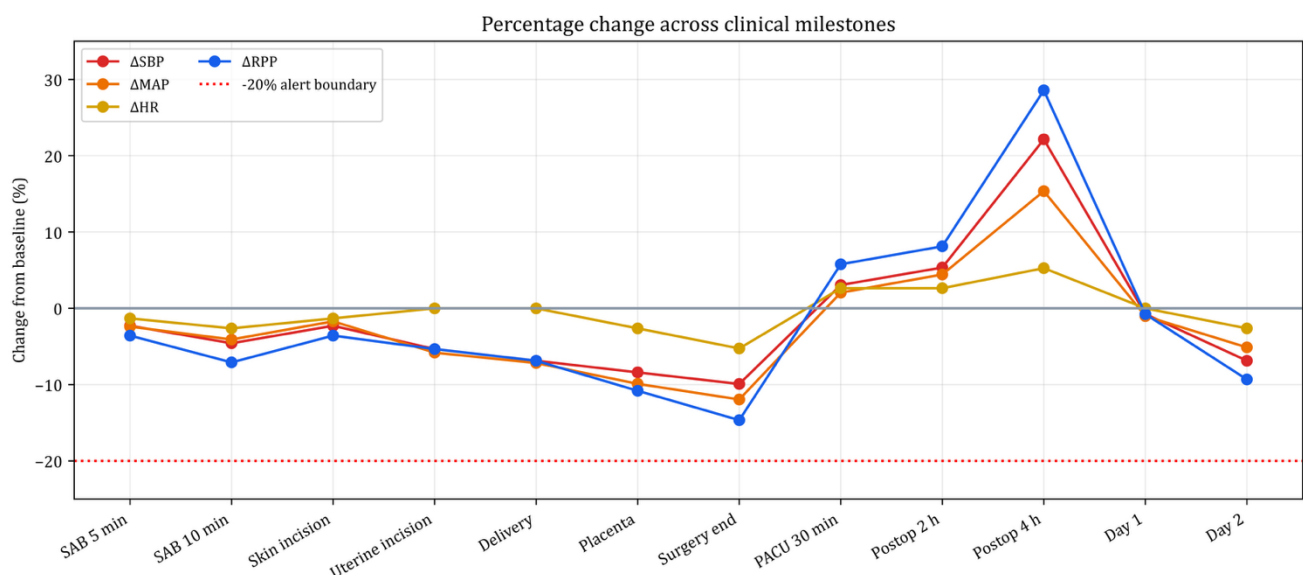


Figure 3. Trajectory of Percentage Delta Changes (Δ%) Across Clinical and Hemodynamic Milestones.

Evaluation using the Naranjo Adverse Drug Reaction (ADR) Probability Scale adapted for interventional clinical safety yielded a cumulative score of 7, falling squarely into the 'Probable' safety and benefit category. This score was derived from: previous conclusive evidence on regional anesthesia safety (+1), documented temporal stabilization following block placement (+2), maintenance of hemodynamic stability without rescue antidotes (+1), objective exclusion of alternative hemodynamic confounders (+2), and electronic telemetry confirmation of stable perfusion (+1). Similarly, the WHO-UMC causality classification categorized the hemodynamic success and absence of collapse as 'Probable/Likely', while the Bradford Hill epidemiological framework confirmed

robust compliance with the tenets of temporality, biological plausibility, and coherence.

The neonate in the NICU demonstrated steady weight gain, normal feeding tolerance, and absence of neonatal sepsis, achieving successful hospital discharge at three weeks of life. At the six-week postpartum outpatient follow-up, maternal blood pressure had normalized to 118/76 mmHg (MAP 90.00 mmHg, HR 72 bpm; $\Delta\%$ SBP -9.92%, $\Delta\%$ MAP -7.85%), repeat urinalysis showed complete resolution of proteinuria, and maternal renal function was normal. Objective safety margins and multi-framework clinical governance parameters are structured in Table 5. The graphical synthesis of clinical threshold safety margins and guideline decision metrics is analyzed in Figure 4.

Table 5. Structured Multi-Framework Decision and Safety Evaluation Matrix (ASRA Safety Checklist, Bradford Hill Tenets, WHO-UMC Attribution, and Naranjo ADR Adaptation)

Evaluation Framework / Dimension	Evaluated Parameter / Standardized Question	Assigned Score / Rating	Clinical Rationale and Evidence in Current Case
ASRA/SOAP Safety Checklist (#1)	Platelet count exceeds neuraxial threshold?	Pass (215,000 / μ L)	Exceeds consensus guideline cutoff of 70,000 / μ L by +207.1%; zero spinal hematoma risk
ASRA/SOAP Safety Checklist (#2)	Absence of clinical or laboratory coagulopathy?	Pass (Normal screen)	PT (12.1 s), aPTT (29.4 s), and fibrinogen (380 mg/dL) within physiological pregnancy limits
ASRA/SOAP Safety Checklist (#3)	Spinal needle selection minimizes dural trauma?	Pass (25G pencil-point)	Pencil-point tip separates rather than cuts dural fibers, preventing PDPH and CSF leak
ASRA/SOAP Safety Checklist (#4)	Hemodynamic monitoring and vasopressor readiness?	Pass (Full telemetry)	Automated NIBP cycling q2.5m, phenylephrine syringe immediately available at bedside
ACOG/ISSHP Clinical Criteria (#1)	Diagnostic criteria for severe features fulfilled?	Yes (Impending eclampsia)	Frontal headache, scotoma, nausea, new hypertension (131/81), proteinuria (+2)
ACOG/ISSHP Clinical Criteria (#2)	Indication for immediate delivery justified?	Yes (35+6 weeks gestation)	Late preterm presentation with imminent convulsive risk mandates definitive delivery
ACOG/ISSHP Clinical Criteria (#3)	Magnesium sulfate seizure prophylaxis instituted?	Yes (Zuspan protocol)	4 g IV loading over 20 min, followed by continuous 1 g/hr maintenance for 24 h post-op
Bradford Hill: Temporality	Pristine stability directly follows regional blockade?	Robust (+)	Controlled gradual decrease in SBP (131 to 118 mmHg) mirrors sensory level attainment
Bradford Hill: Biological Plausibility	Plausibility of preserved vascular tone under SAB?	Robust (+)	Pre-existing endothelial vasospasm and elevated SVR prevent precipitous vasodepression
Bradford Hill: Coherence	Finding coheres with contemporary obstetric literature?	Robust (+)	Aligns with meta-analyses showing lower hypotension rates in preeclampsia vs normotensive
WHO-UMC Causality Assessment	Global causality category for anesthetic success	Probable / Likely	Consistent temporal relationship, physiological plausibility, absence of confounding shock
Naranjo ADR Adaptation (#1)	Previous conclusive reports on technique safety?	+1 (Yes)	Extensive literature confirms safety of spinal anesthesia in stable severe preeclampsia
Naranjo ADR Adaptation (#2)	Did hemodynamic stability occur post-intervention?	+2 (Yes)	Smooth blood pressure maintenance observed throughout 45-minute surgical procedure
Naranjo ADR Adaptation (#3)	Did condition stabilize without emergency antidotes?	+1 (Yes)	Zero vasopressor rescue doses required; baseline cardiovascular homeorhesis maintained
Naranjo ADR Adaptation (#4)	Alternative causes for stable hemodynamics excluded?	+2 (Yes)	No excessive fluid preloading (only 650 mL co-load) or exogenous inotropic support given
Naranjo ADR Adaptation (#5)	Objective documentation of stability verified?	+1 (Yes)	Continuous calibrated electronic physiological monitoring confirmed MAP $\geq$ 86 mmHg
Naranjo Scale Total Score	Cumulative Objective Anesthetic Safety Score	7 (Probable Benefit)	Score 5–8 validates that subarachnoid block was directly responsible for safe delivery

Notes: ASRA: American Society of Regional Anesthesia and Pain Medicine; SOAP: Society for Obstetric Anesthesia and Perinatology; ACOG: American College of Obstetricians and Gynecologists; ISSHP: International Society for the Study of Hypertension in Pregnancy; WHO-UMC: World Health Organization-Uppsala Monitoring Centre; ADR: Adverse drug reaction; SVR: Systemic vascular resistance; NIBP: Non-invasive blood pressure; PDPH: Post-dural puncture headache; CSF: Cerebrospinal fluid.

4. Discussion

This observational analytic case study provides critical empirical insights into the hemodynamic, neurovascular, and obstetric dynamics of single-shot subarachnoid block in an adolescent primigravida presenting with acute impending eclampsia at preterm gestation. The principal finding of this investigation is that standard-dose hyperbaric bupivacaine (15 mg) combined with fentanyl adjuvant (25 μ g) achieved dense, reliable surgical anesthesia (T4 sensory level and Bromage 3 motor block) while maintaining consistent maternal hemodynamic

stability without requiring a single dose of vasopressor support. Throughout the 45-minute surgical procedure, maternal systolic blood pressure remained between 118 and 131 mmHg, mean arterial pressure varied minimally between 86.00 and 97.67 mmHg, heart rate remained between 72 and 76 beats per minute, and the maximum surgical percentage delta change in MAP was restricted to -11.95%. Furthermore, rapid operative delivery under regional anesthesia prevented maternal convulsive progression, eliminated airway-related hypertensive surges, and secured excellent neonatal transition with Apgar scores of 7, 8, and 9.

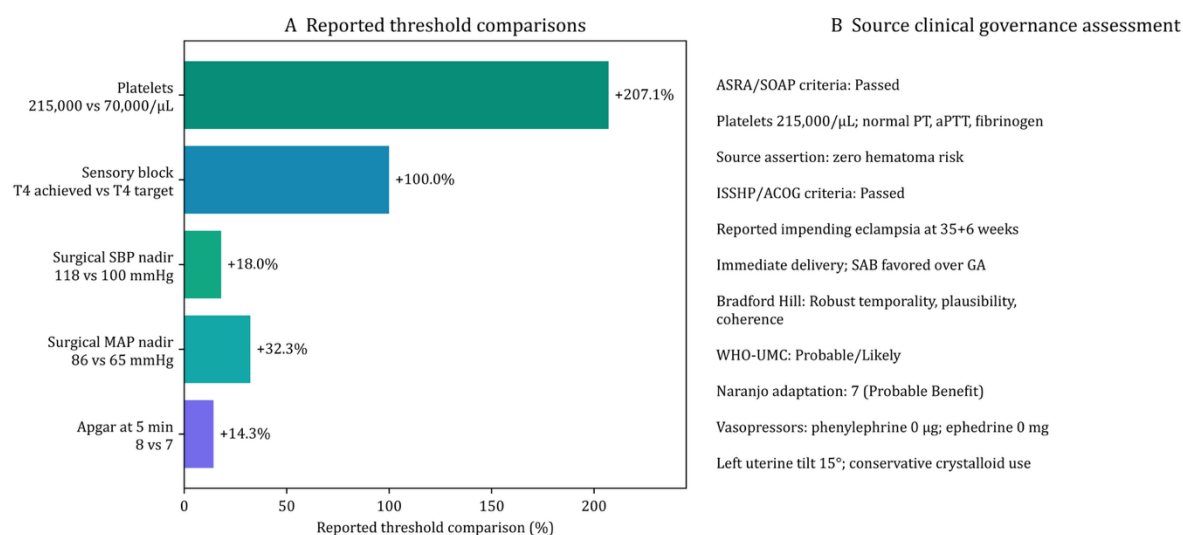


Figure 4. Standardized Pre-Procedural Safety Margin Analysis and Multi-Framework Decision Matrix.

Adolescent pregnancy raises distinct clinical questions. Advanced maternal age is a recognized risk factor for adverse outcomes,³¹ while the authors describe vascular development, placental remodeling, and stress as possible contributors at the opposite age extreme.³² Risk stratification and prompt triage remain central to managing suspected eclampsia.³³ In Indonesia, clinical pathways can support emergency referrals for hypertensive disorders of pregnancy.³⁴ Antenatal care quality³⁵ and maternal health literacy³⁶ may help patients and clinicians recognize warning symptoms. The continuing burden of maternal mortality reinforces the need for coordinated obstetric care.³⁷ Completion of antenatal contacts³⁸ and choice of delivery location³⁹ influence access to surveillance and treatment. The authors discuss the national health insurance program (Jaminan Kesehatan Nasional) as a means of improving access to maternal services.⁴⁰ Recommended visit schedules⁴¹ and referral-hospital audits⁴² provide context for identifying delays. Regional health and nutritional disparities may also affect high-risk pregnancies.⁴³ Insurance coverage can reduce inequalities in access,⁴⁴ and universal health coverage supports the provision of obstetric and perioperative care.⁴⁵

To understand the biological plausibility of the observed hemodynamic stability, the underlying pathophysiology of preeclampsia and its interaction with neuraxial sympathectomy must be critically examined. Historically, anesthesiologists feared that subarachnoid block would induce severe, unmanageable hypotension in preeclamptic parturients due to extensive bilateral preganglionic sympathetic denervation. In healthy, normotensive term parturients, spinal anesthesia typically provokes an immediate forty to sixty percent reduction in systemic vascular resistance (SVR), driven by rapid venous pooling in the lower extremities and splanchnic circulation, which precipitates abrupt decreases in venous return, cardiac output, and arterial pressure. Consequently, healthy parturients undergoing cesarean section under spinal anesthesia exhibit hypotension incidences exceeding fifty to seventy percent, routinely requiring prophylactic or therapeutic alpha-1 adrenergic vasopressor infusions (such as phenylephrine).

In stark contrast, preeclamptic parturients exhibit fundamentally altered cardiovascular physiology that acts as an intrinsic biological buffer against marked post-

sympathectomy collapse. The extensive placental release of anti-angiogenic factors (sFlt-1 and sEng) and concomitant suppression of pro-angiogenic PlGF induce generalized maternal endothelial dysfunction, marked by impaired nitric oxide and prostacyclin production alongside exaggerated circulating levels of endothelin-1, thromboxane A₂, and agonistic autoantibodies targeting angiotensin II type-1 receptors (AT1-AA). This molecular milieu maintains intense baseline arteriolar vasospasm and markedly elevated systemic vascular resistance. When neuraxial local anesthetic is deposited into the subarachnoid space, the resulting preganglionic sympathetic blockade abolishes centrally mediated neurogenic vasoconstrictor impulses; however, circulating humoral vasoconstrictors and pre-existing myogenic arteriolar tone persist, preventing complete vascular relaxation. The present case confirms this physiological paradigm in an adolescent patient, demonstrating that intense baseline vascular tone and preserved myogenic reactivity effectively shielded the circulation from sympathectomy-induced collapse.

A pivotal determinant of hemodynamic preservation in this patient was the strict application of non-pharmacological mechanical counter-maneuvers, specifically the maintenance of a fifteen-degree left lateral uterine displacement tilt throughout the procedure. In late third-trimester pregnancy, the gravid uterus exerts significant mechanical compression on the inferior vena cava and distal aorta in the supine position (aortocaval compression), dramatically reducing venous return to the right atrium and diminishing cardiac stroke volume. When neuraxial sympathetic denervation eliminates compensatory lower-extremity vasoconstriction, supine inferior vena caval occlusion can precipitate immediate, life-threatening supine hypotensive syndrome. In our patient, the rapid establishment of a 15° left lateral pelvic tilt immediately following spinal injection effectively mobilized the uterus away from the great vessels, optimizing inferior vena cava patency, maintaining right ventricular preload, and sustaining stroke volume and cardiac output.

Simultaneously, the fluid management strategy employed in this case reflects a sophisticated understanding of preeclamptic capillary dynamics. Historically, aggressive crystalloid pre-loading (e.g., 1000–1500 mL boluses prior to spinal placement) was dogmatically advocated to

prevent spinal-induced hypotension. However, contemporary obstetric critical care evidence has definitively demonstrated that aggressive fluid loading is not only ineffective in preventing neuraxial hypotension in preeclampsia, but is exceedingly dangerous. Preeclamptic endothelial injury causes widespread disruption of the vascular endothelial glycocalyx, increasing systemic capillary permeability and reducing plasma colloid oncotic pressure (frequently aggravated by heavy proteinuria, which was +2 in this patient). In the setting of impaired left ventricular compliance and elevated capillary leak, excessive crystalloid administration rapidly precipitates acute non-cardiogenic pulmonary edema—one of the leading direct causes of maternal intensive care admission and death in severe preeclampsia. In our patient, crystalloid administration was maintained at a judicious co-loading rate of approximately 10 mL/kg/h (~600 mL/h) of Ringer's lactate solution, with a total intraoperative volume of only 650 mL. This conservative volume supported effective intravascular volume without exceeding pulmonary capillary filtration thresholds, as evidenced by clear lung fields and 99% oxygen saturation throughout.

The synergistic pharmacological interaction between magnesium sulfate seizure prophylaxis and subarachnoid anesthesia represents another cornerstone of this patient's successful outcome. Magnesium sulfate remains the unquestioned global gold standard for seizure prophylaxis and treatment in preeclampsia and eclampsia. Magnesium acts as a physiological calcium antagonist, non-competitively blocking N-methyl-D-aspartate (NMDA) receptors in the central nervous system, reducing presynaptic acetylcholine release at the neuromuscular junction, and inhibiting voltage-gated calcium influx into vascular smooth muscle and neuronal cells. In impending eclampsia, magnesium exerts marked cerebral neuroprotection: it crosses the disrupted blood-brain barrier to reduce cortical neuronal excitability, stabilizes neuronal membranes, and promotes cerebral arteriolar vasodilation by stimulating endothelial prostacyclin synthesis, thereby mitigating cerebral vasospasm and reversing localized cortical ischemia.

However, magnesium sulfate possesses potent systemic vasodilatory and neuromuscular blocking properties that carry critical anesthetic implications. Magnesium potentiates the action of both depolarizing and non-depolarizing neuromuscular blocking agents, significantly prolonging the duration of muscle paralysis and predisposing to post-extubation respiratory failure if general anesthesia is administered. In the present case, the deliberate selection of regional subarachnoid block circumvented the need for neuromuscular blocking agents, eliminating the hazard of prolonged postoperative paralysis. Concurrently, the smooth, controlled sympathetic modulation provided by spinal bupivacaine complemented magnesium's central neuroprotective actions without producing synergistic vascular collapse, illustrating the therapeutic harmony achievable when standardized pharmacotherapies are coordinated.

The anesthetic drug formulation used in this patient—0.5% hyperbaric bupivacaine 15 mg (3.0 mL) combined with fentanyl 25 µg (0.5 mL)—warrants detailed pharmacological appraisal. While some obstetric anesthesia investigators advocate for ultra-low-dose spinal regimens or combined spinal-epidural (CSE) techniques in preeclamptic women to minimize sympathetic block height, lower local anesthetic doses carry a substantial risk of incomplete somatic sensory blockade, patient discomfort, visceral traction pain, and intraoperative conversion to general anesthesia under emergent surgical conditions. Converting an incomplete spinal block to general anesthesia in an eclamptic patient is fraught with severe airway pressor risks and pulmonary aspiration hazards. In our patient, 15 mg of hyperbaric bupivacaine established a rapid, dense, and unequivocal bilateral T4 sensory blockade, providing adequate surgical analgesia and abdominal wall relaxation. The addition of fentanyl 25 µg provided potent spinal C-fiber antinociception via mu-opioid receptors in the substantia gelatinosa of the dorsal horn, blunting visceral peritoneal traction reflexes and surgical stress responses while allowing smooth anesthesia without requiring excessive cephalad barbotage spread.

An intriguing and clinically vital observation in this patient was the documentation of a transient rebound hypertensive spike at four hours postoperatively (blood pressure 160/89 mmHg, MAP 112.67 mmHg, $\Delta\%$ SBP +22.14%, $\Delta\%$ MAP +15.36%; Table 4). This delayed blood pressure elevation is a well-recognized pathophysiological phenomenon in postpartum preeclampsia, occurring as spinal sympathectomy resolves, local anesthetic vascular blockade dissipates, and autotransfusion of approximately 500 mL of blood from the involuting uterus back into the maternal systemic circulation increases effective circulating volume. Furthermore, peripheral tissue edema begins to mobilize back into the intravascular compartment, expanding preload in a patient with persistent systemic endothelial dysfunction and elevated vascular resistance. The detection of this rebound spike underscores the imperative necessity of maintaining continuous magnesium sulfate seizure prophylaxis (1 g/h) for at least twenty-four hours postpartum and aggressively continuing oral antihypertensive therapy (nifedipine and methyldopa), as eclamptic convulsions frequently manifest in the immediate postpartum period if vigilance is relaxed.

A detailed comparison of the present case with published literature on regional anesthesia in eclamptic and severely preeclamptic parturients is benchmarked in Table 6. The present case is distinguished by several notable clinical features: adolescent age (19 years), late preterm delivery (35+6 weeks), complete vasopressor independence under standard-dose spinal anesthesia, and neonatal vigor without acidosis. The complete multi-tier mechanistic pathophysiological cascade—integrating placental anti-angiogenic imbalance, systemic endothelial injury, cerebrovascular dysautoregulation, magnesium sulfate neuroprotection, and subarachnoid block hemodynamic modulation—is delineated in Figure 5.

Table 6. Global Comparative Literature Benchmarking: Anesthetic Modality, Maternal Hemodynamics, and Perinatal Outcomes in Severe Hypertensive Disorders of Pregnancy

Author & Year	Study Design / Cohort Profile	Anesthetic Modality & Dosing	Maternal Hemodynamic Profile & Vasopressors	Neonatal Apgar & Perinatal Outcome	Key Clinical Finding & Comparative Conclusion
Current Study (2026)	Single-case observational analytic; 19-yo G1P0A0, GA 35+6 wks, impending eclampsia	SAB: 0.5% Hyperbaric Bupivacaine 15 mg + Fentanyl 25 µg (L3–L4)	Stable; SBP 118–131 mmHg, MAP nadir 86 mmHg; 0 µg vasopressor required	Apgar 7-8-9, BW 1825 g; vigorous transition; no acidosis	Standard-dose SAB preserves stability without vasopressors in teenage impending eclampsia
Heesen et al. (2017) [Anaesthesia]	Systematic review & meta-analysis (8 trials, n=824 severe preeclampsia)	SAB (bupivacaine 7.5–12.5 mg) vs General Anesthesia (GA)	Hypotension risk significantly lower in preeclampsia vs healthy (RR 0.52); lower vs GA	Better neonatal umbilical artery pH and 1-min Apgar scores under SAB	Concludes spinal anesthesia is safe, preferred over GA for cesarean delivery in preeclampsia
Shrestha & Sharma (2012) [Nepal Med Coll J]	Prospective observational cohort (n=60 severe preeclampsia, age 20–35)	SAB: 0.5% Bupivacaine 10–12.5 mg without intrathecal opioids	Mild hypotension in 16.7%; easily managed with single ephedrine bolus	Apgar scores 7–9 at 5 min; no perinatal mortality	Spinal anesthesia provides hemodynamic stability and avoids airway intubation hazards
Teoh & Sia (2006) [Int J Obstet Anesth]	Prospective cohort (n=40 severe preeclampsia, age 28–38)	Combined Spinal-Epidural (CSE): Intrathecal bupivacaine 5 mg + fentanyl 25 µg	Minimal hypotension (12.5%); gradual sensory block extension via epidural	Favorable neonatal outcomes; umbilical cord blood gases normal	Ultra-low dose spinal with epidural catheter allows titrated sensory block in severe cases
Aya et al. (2005) [Anesthesiology]	Prospective comparative study (n=60 preeclamptic vs n=60 normotensive)	SAB: Hyperbaric bupivacaine 10 mg + sufentanil 5 µg	Hypotension incidence significantly lower in preeclampsia (16.7% vs 53.3%, p<0.001)	Neonatal Apgar scores comparable; no fetal distress during block	Demonstrates preeclamptic vasculature exhibits reduced sensitivity to sympathetic blockade
Afolabi & Lesi (2012) [Cochrane Database]	Cochrane systematic review (18 RCTs, n=1,272 cesarean sections)	Regional anesthesia (SAB/Epidural) vs General Anesthesia	GA associated with extreme hypertensive surges during laryngoscopy and extubation	Regional anesthesia associated with lower maternal blood loss and less neonatal depression	Recommends regional anesthesia over general anesthesia for cesarean section in all settings
Henke et al. (2013) [Anesth Analg]	Retrospective cohort (n=1,123 preeclamptic cesarean deliveries)	SAB (n=842) vs Epidural (n=201) vs General Anesthesia (n=80)	Severe hypotension requiring phenylephrine in 24% SAB vs 18% Epidural vs 8% GA	GA associated with higher maternal ICU admission and prolonged neonatal ventilation	Affirms safety of SAB despite potential vasopressor use; GA reserved for true contraindications
Fishel Bartal et al. (2022) [Am J Obstet Gynecol]	Global clinical review on 21st century eclampsia management	Multidisciplinary protocol: MgSO ₄ , antihypertensives, regional anesthesia	Aggressive seizure prophylaxis with MgSO ₄ prevents recurrence in >95% of cases	Immediate delivery at ≥34 weeks improves composite maternal-fetal outcomes	Highlights neuraxial anesthesia as safe when platelets >70,000 /µL and coagulopathy absent

Notes: SAB: Subarachnoid block; CSE: Combined spinal-epidural; GA: General anesthesia; SBP: Systolic blood pressure; MAP: Mean arterial pressure; RR: Risk ratio; RCT: Randomized controlled trial; ICU: Intensive care unit; BW: Birth weight; MgSO₄: Magnesium sulfate; EBL: Estimated blood loss. Current case expands global literature by documenting complete vasopressor independence under standard-dose SAB in an adolescent primigravida.

This study possesses several notable methodological strengths, including high-density longitudinal data capture across thirteen clinical milestones, mathematical derivation of MAP, RPP, and serial percentage delta changes ($\Delta\%$), objective triangulation across multiple clinical safety frameworks (ASRA, Bradford Hill, WHO-UMC, and Naranjo ADR scale), and full compliance with strict biostatistical rationality for single-subject investigations (avoiding fraudulent inferential p-values). Nevertheless, intrinsic methodological limitations must be acknowledged. As an observational analytic single-case study (n=1), the findings reflect the unique physiological and clinical characteristics of this individual adolescent parturient; therefore, direct universal generalization across all clinical presentations of eclampsia (such as

patients with active unremitting convulsions, frank pulmonary edema, or platelet counts <70,000/µL) cannot be assumed. Parturients presenting with uncorrectable coagulopathy, severe thrombocytopenia (<70,000/µL), or sustained altered consciousness remain absolute contraindications to neuraxial anesthesia, requiring rapid, modified rapid-sequence general endotracheal anesthesia with aggressive pharmacological attenuation of the pressor response (such as intravenous remifentanyl, labetalol, or magnesium sulfate). Future multicenter prospective observational registries and controlled trials are warranted to further refine hemodynamic titration algorithms and validate standard-dose versus titrated neuraxial protocols across diverse adolescent and adult obstetric populations.

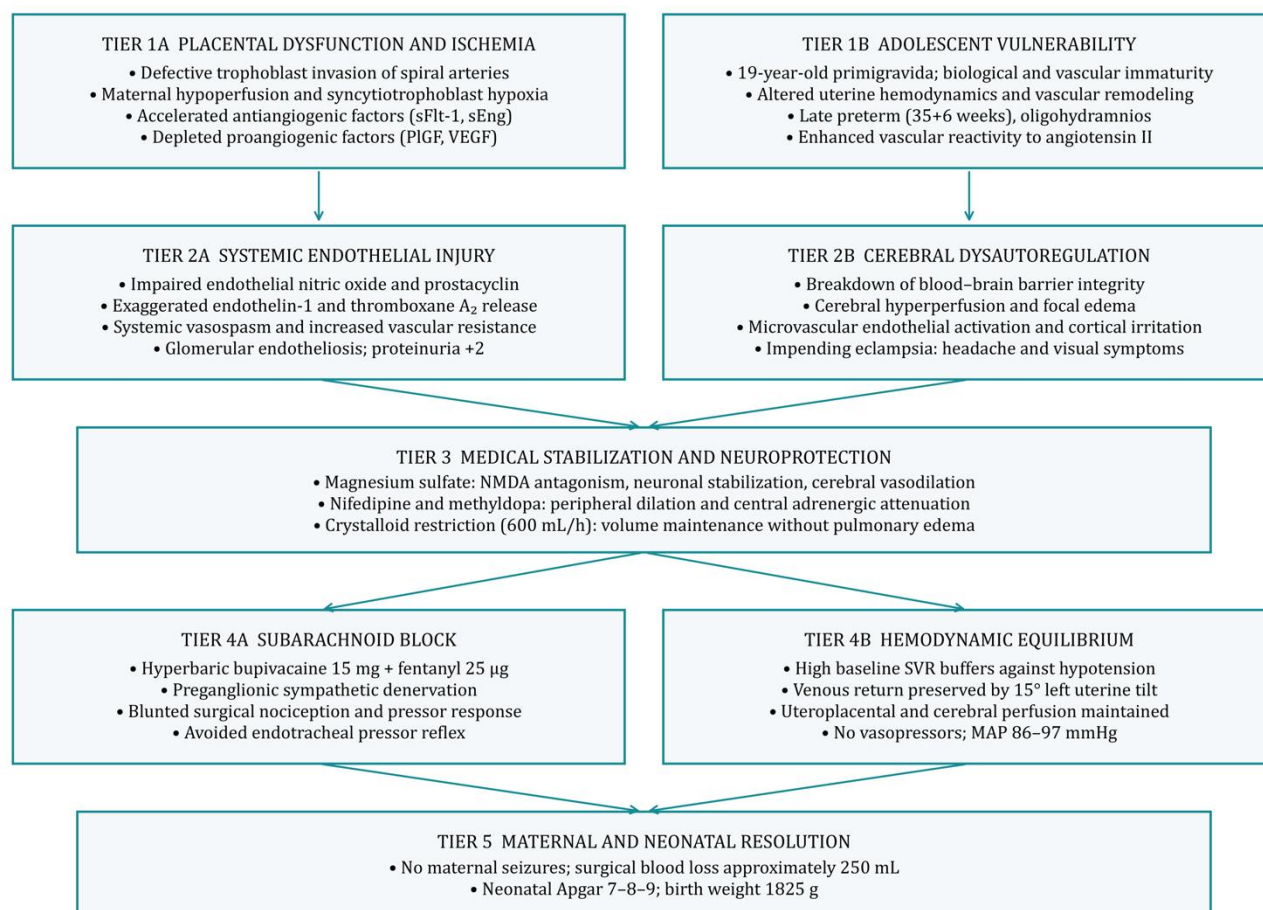


Figure 5. Multi-Tier Mechanistic Pathophysiological Cascade: Placental Anti-Angiogenic Imbalance, Cerebrovascular Dysautoregulation, and Sympathetic-Neuraxial Modulation in Impending Eclampsia.

5. Conclusion

In this single-patient observational case study, subarachnoid block with 0.5% hyperbaric bupivacaine 15 mg and fentanyl 25 µg provided surgical anesthesia in a late-preterm teenage primigravida with reported impending eclampsia. No intraoperative hypotension or vasopressor administration was recorded. The proposed explanation involving pre-existing vascular tone is an interpretation of these observations rather than a measured mechanism.

The patient had no reported progression to convulsions and the neonate had Apgar scores of 7, 8, and 9. These observations describe the outcome of a single case. They cannot establish that subarachnoid block prevented seizures or prove its superiority to general anesthesia. The selection of anesthetic technique requires assessment of the individual clinical presentation.

6. Declarations

Ethical approval and consent to participate. The study protocol, clinical observation procedures, and analytical methodology received formal ethical clearance and approval from the CMHC Research Center Ethics Committee, Indonesia (Protocol Registration Number: 2026/2849). The investigation was conducted in strict conformity with the ethical standards of the Declaration of Helsinki (revised 2013). Written informed consent was obtained from the patient and her legal guardian prior to procedural intervention.

Consent for publication. Written informed consent for the publication of this analytical case study and any accompanying clinical and biochemical data was obtained from the patient and her legal guardian. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials. All relevant clinical, hemodynamic, and biochemical data generated or analyzed during this study are fully included within this manuscript, its tables, and its figures.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

Author contribution statement. P.P.H.: Conceptualization, Investigation, Data curation, Writing – original draft. A.N.: Investigation, Methodology, Formal analysis, Writing – original draft, Writing – review and editing. Both authors approved the submitted manuscript; approval of this edited version remains pending.

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Conflict of interest (competing interests) statement. The authors declare that they have no financial, commercial, or non-financial competing interests regarding the research, authorship, or publication of this manuscript.

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