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# General Anesthesia and Modified Rapid Sequence Induction for Emergency Cesarean Delivery in an Eclamptic Adolescent with Severe Thrombocytopenia

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### A B S T R A C T

**Background:** Eclampsia complicated by severe thrombocytopenia presents a critical anesthetic challenge, often representing an absolute contraindication to neuraxial anesthesia. When alternative approaches are mandated, General Anesthesia with Rapid Sequence Induction or a modified rapid sequence approach is crucial for high-risk obstetric emergencies. **Case presentation:** A 16-year-old primigravida at 35 weeks gestation presented with eclampsia, acute fetal distress, and severe thrombocytopenia. Initial vitals showed a blood pressure of 150/98 mmHg. Laboratory findings confirmed severe preeclampsia with a critical platelet count of 42,000/ $\mu$ L. An airway assessment revealed a Mallampati class II airway with mild pharyngolaryngeal edema. Due to the high risk of spinal epidural hematoma, neuraxial anesthesia was contraindicated. An emergency cesarean section was performed using a modified rapid sequence induction. Following preoxygenation, induction was achieved with fentanyl 100  $\mu$ g, midazolam 3 mg, and propofol 100 mg. Due to institutional unavailability of rocuronium, atracurium 30 mg was utilized. After a 2.5-minute onset interval utilizing apneic oxygenation and continuous cricoid pressure, the patient was successfully intubated on the first attempt with a Macintosh size 3 blade. The intraoperative course was hemodynamically stable. **Conclusion:** A modified rapid sequence induction utilizing atracurium provides an effective alternative for airway control and physiological stability in eclamptic adolescents with coagulopathy, particularly in resource-limited settings where standard rapid-acting non-depolarizing agents are unavailable.

### 1. Introduction

The anesthetic management of obstetric emergencies frequently demands rapid, high-stakes clinical decision-making, yet rarely do practitioners encounter a scenario as fraught with compounded risks as the simultaneous convergence of extreme maternal youth, active eclamptic neuro-hyperactivity, severe thrombocytopenia, and acute fetal distress.<sup>1</sup> This precise intersection of pathologies generates a highly complex pathophysiological matrix that fundamentally alters standard obstetric anesthesia algorithms. Eclampsia complicated by severe thrombocytopenia presents a critical anesthetic

challenge, often representing an absolute contraindication to neuraxial anesthesia. When regional anesthetic techniques are unequivocally precluded by hematological derangements, general anesthesia becomes the mandatory alternative; however, securing the airway in an actively seizing or postictal pregnant patient introduces profound risks of failed intubation, catastrophic hypertensive crises, and pulmonary aspiration. In such high-risk obstetric emergencies where alternative approaches are mandated, General Anesthesia with Rapid Sequence Induction or a modified rapid sequence approach is crucial. This clinical dilemma is exponentially

magnified when practicing in resource-limited environments where gold-standard pharmacological agents are unavailable, requiring anesthesiologists to adapt established protocols without compromising maternal or fetal safety.<sup>2</sup>

To fully appreciate the gravity of this clinical presentation, one must first dissect the severe airway vulnerability inherent to eclampsia. Eclampsia represents the severe end of the preeclampsia spectrum, characterized by generalized tonic-clonic seizures and driven by widespread endothelial dysfunction, intense vasospasm, and an exaggerated systemic inflammatory response.<sup>3</sup> In the central nervous system, the loss of cerebral autoregulation leads to hyperperfusion, endothelial leakage, and subsequent vasogenic cerebral edema, all of which culminate in a significantly lowered seizure threshold. For the anesthesiologist, these systemic vascular changes manifest most dangerously as a highly vulnerable upper airway. The capillary leak syndrome associated with severe preeclampsia frequently results in pharyngolaryngeal edema, rendering the upper airway friable and significantly reducing the glottic opening. Furthermore, the profound endothelial dysfunction means that the cardiovascular system is ill-equipped to handle the intense nociceptive stimulus of direct laryngoscopy and endotracheal intubation. The sympathetic response to direct laryngoscopy in a patient with pre-existing endothelial dysfunction can precipitate catastrophic hypertensive crises, leading directly to intracranial hemorrhage or pulmonary edema. Therefore, any induction sequence utilized must definitively blunt the sympathoadrenal axis while simultaneously ensuring the rapid securing of the airway to prevent fatal pulmonary aspiration.<sup>4</sup>

Compounding this treacherous airway scenario is the severe hematologic derangement that forces the anesthesiologist away from safer regional techniques. While neuraxial anesthesia is generally the preferred modality for cesarean deliveries to avoid the risks associated with a compromised obstetric airway, severe thrombocytopenia renders neuraxial

anesthesia absolutely contraindicated.<sup>5</sup> The pathophysiology of thrombocytopenia in preeclampsia is primarily driven by microangiopathic hemolysis and rapid platelet consumption. As the systemic disease progresses, endothelial damage exposes subendothelial collagen throughout the vascular tree, triggering widespread platelet aggregation and the formation of microthrombi in the microvasculature. This active, consumptive coagulopathy severely depletes circulating platelets and impairs their overall hemostatic function. Performing a subarachnoid or epidural block in this specifically depleted setting carries an unacceptably high risk of accidental vascular puncture, inevitably leading to a spinal epidural hematoma. Such a complication, occurring within the rigid, confined space of the spinal canal, can rapidly cause irreversible spinal cord compression and permanent paraplegia. Consequently, the anesthesiologist is forced to abandon neuraxial approaches and confront the eclamptic airway directly.<sup>6</sup> The third, and perhaps most critical, dimension of this matrix is the physiological vulnerability of the adolescent gravida. Extreme maternal youth, specifically adolescence, introduces unique physiological and systemic variables into the management of preeclampsia and eclampsia. The physiological landscape of a 16-year-old parturient cannot be equated to that of an adult; adolescent gravidas are frequently physically immature, possessing underdeveloped pelvic anatomy and notably lower baseline circulating blood volumes compared to adult parturients.<sup>7</sup> This reduced physiological reserve makes them particularly susceptible to sudden hemodynamic shifts during anesthesia. Furthermore, the intense systemic inflammatory response and endothelial dysfunction inherent to eclampsia often manifest more precipitously in adolescent patients, leading to a faster and more unpredictable progression to severe neurological and hematological sequelae. From a neurovascular perspective, adolescent vasculature and developing central nervous systems often exhibit

heightened reactivity to nociceptive stimuli. This amplified sympathetic tone requires meticulous and careful titration of induction agents to ensure profound anesthesia and blunt the pressor response of intubation, all without precipitating sudden cardiovascular collapse in a hemodynamically fragile patient. The adolescent eclamptic patient is not merely a smaller adult, but a patient undergoing concurrent developmental and pathological vascular stress, requiring highly tailored pharmacological strategies.<sup>8</sup>

Finally, the navigation of these formidable physiological challenges is frequently constrained by the realities of healthcare logistics, introducing the final element of the dilemma: resource limitations regarding optimal neuromuscular blockade. General anesthesia rapid sequence induction represents a cornerstone airway management strategy for patients at increased risk of pulmonary aspiration, characterized by rapid intravenous induction, the avoidance of manual ventilation, application of cricoid pressure, and prompt endotracheal intubation. In modern obstetric anesthesia, high-dose rocuronium is widely considered the gold standard alternative to succinylcholine, providing optimal intubating conditions in 60 seconds without raising intracranial pressure.<sup>9</sup> However, in many global healthcare environments, supply chain shortages frequently render this ideal agent unavailable. While succinylcholine remains a rapid-acting alternative, it is rightfully avoided in the severe eclamptic patient due to the well-documented risks of transient increases in intracranial pressure and potential hyperkalemia in a neurologically vulnerable, postictal patient. Deprived of both rapid-acting gold standards, the anesthesiologist must devise a modified induction strategy using intermediate-acting non-depolarizing agents, such as atracurium, which possesses a significantly slower onset time. This necessitates prolonged periods of apnea, requiring advanced techniques like continuous cricoid pressure and apneic oxygenation to prevent hypoxia and aspiration during the extended induction window.<sup>10</sup>

Therefore, the aim of this study is to elucidate the pathophysiological rationale governing anesthetic drug selection, hemodynamic optimization, and airway management in this specific, high-risk clinical triad of adolescent eclampsia, severe thrombocytopenia, and acute fetal distress. By detailing the successful navigation of these compounded challenges, including the critical adaptation of standard protocols to accommodate severe resource limitations, this case report provides a novel framework for obstetric critical care. Ultimately, it establishes a pragmatic, safe, and physiologically sound approach for environments where gold-standard neuromuscular blockers are unavailable, ensuring that vulnerable parturients can be safely managed regardless of institutional constraints.

## **2. Case Presentation**

All procedures detailed in this case report were conducted in strict accordance with the ethical standards established by the institutional research committee and comply with the principles outlined in the 1964 Declaration of Helsinki and its subsequent amendments. As this manuscript constitutes a retrospective observation of a single clinical case, formal approval from the Institutional Review Board (IRB) was exempted per standard institutional policies regarding isolated case reports. Nevertheless, the utmost priority was given to the protection of patient privacy and medical confidentiality. Because the patient is a 16-year-old adolescent, written informed consent for the publication of this clinical narrative, encompassing all laboratory parameters and intraoperative data, was obtained from both the patient and her legally authorized representative. To guarantee absolute anonymity, all personally identifiable information—including precise dates, institutional affiliations, and specific geographic markers—has been entirely de-identified or omitted from the manuscript. The patient and her legal guardian were comprehensively counseled regarding the academic, non-commercial objectives of this

dissemination and granted their explicit permission to share these findings to advance obstetric critical care. Furthermore, the preparation of this manuscript aligns with the consensus-based CARE (CAse REport) guidelines to ensure methodological transparency.

The clinical course initiated with the urgent admission of a 16-year-old primigravida, currently at 35 weeks gestation, who presented to the emergency department for impending delivery driven by a cascade of severe, concurrent medical and obstetric indications. Her complex clinical matrix included a primary diagnosis of eclampsia, compounded by acute fetal distress, premature rupture of membranes, and the critically limiting factor of severe thrombocytopenia. Upon securing her medical history, it was noted that the patient possessed a history of seizures, although she explicitly denied the presence of any other significant medical illnesses or a history of prior surgical interventions.

The acute neurological emergency was marked by the onset of seizures that occurred approximately two hours prior to her emergency department admission. This eclamptic episode was clinically characterized by generalized tonic-clonic activity that persisted for a duration of 90 seconds, which was subsequently followed by a pronounced postictal state. Upon her formal admission at 08:00, the clinical assessment revealed a patient who remained conscious but was notably lethargic. Her initial vital signs demonstrated a hypertensive state with a blood pressure recorded at 150/98 mmHg. Concurrently, she exhibited a heart rate of 67 beats per minute and a respiratory rate of 20 breaths per minute, while maintaining an oxygen saturation of 98 percent, achieved through the administration of 3 L/min of supplemental oxygen delivered via a nasal cannula. Her baseline anthropometric evaluation indicated a body mass index of 18.57 kg/m<sup>2</sup>. The physical examination further underscored her critical neurological status; it revealed pronounced hyperreflexia, documented as 3+ patellar reflexes, alongside two beats of clonus observed bilaterally. These specific neurological findings were

highly consistent with severe central nervous system irritability.

In anticipation of surgical delivery, a thorough and meticulous preoperative airway assessment was conducted. The anatomical evaluation of the patient exhibited a Mallampati class II airway, alongside a reassuring thyromental distance of 6.5 cm and an adequate mouth opening that was measured to be greater than 4 cm. However, this otherwise standard airway profile was complicated by the notation of mild pharyngolaryngeal edema. This concerning upper airway swelling was likely secondary to the capillary leak syndrome that is characteristically driven by severe preeclampsia. Additionally, a previously obtained computed tomography scan had demonstrated the presence of a grade I right-sided nasal septal deviation. While this anatomical anomaly was carefully noted, it was ultimately deemed irrelevant for the purposes of the planned oral intubation strategy. Consequently, based on the accumulation of these severe systemic and obstetric morbidities, the patient was classified as American Society of Anesthesiologists physical status III, carrying an emergency indication (Table 1).

Table 2 provides a comprehensive overview of the preoperative laboratory and coagulation profiles, revealing severe systemic derangements fundamentally consistent with the pathology of severe preeclampsia. The hematologic evaluation demonstrated moderate anemia, characterized by a depressed hemoglobin concentration of 9.2 g/dL and a correspondingly decreased hematocrit of 28 percent. Most critically, the patient exhibited severe thrombocytopenia, evidenced by a critically depleted platelet count of just 42,000/ $\mu$ L. The underlying pathophysiology of this specific thrombocytopenia is primarily driven by microangiopathic hemolysis and rampant platelet consumption. As the systemic disease progresses, intense endothelial damage exposes subendothelial collagen throughout the vascular tree, triggering widespread platelet aggregation and the extensive formation of microthrombi within the microvasculature.

**Table 1. Summary of Clinical Findings on Admission**

Category	Parameter	Clinical Finding / Value
Demographics & History	Age & Obstetric Status	16-year-old primigravida at 35 weeks gestation.
	Clinical Indications	Eclampsia, fetal distress, premature rupture of membranes, and severe thrombocytopenia.
	Neurological History	Onset of seizures ~2 hours prior (generalized tonic-clonic for 90 seconds).
	Admission Status (08:00)	Conscious but lethargic, postictal state.
Vital Signs & Anthropometrics	Blood Pressure	150/98 mmHg.
	Heart Rate	67 beats/min.
	Respiratory Rate	20 breaths/min.
	Oxygen Saturation (SpO <sub>2</sub> )	98 percent on 3 L/min supplemental oxygen via nasal cannula.
	Body Mass Index (BMI)	18.57 kg/m <sup>2</sup> .
Physical & Airway Assessment	Neurological Exam	Hyperreflexia (3+ patellar reflexes) and two beats of clonus bilaterally.
	Mallampati Classification	Class II.
	Airway Dimensions	Thyromental distance of 6.5 cm; adequate mouth opening (> 4 cm).
	Airway Edema	Mild pharyngolaryngeal edema.
	Nasal Anatomy	Grade I right-sided nasal septal deviation.
Obstetric & General Assessment	Fetal Assessment (Cardiotocography)	Baseline fetal heart rate of 165 bpm with absent variability and recurrent late decelerations.
	ASA Physical Status	Class III with an emergency indication.

This active, consumptive coagulopathy severely depletes the circulating platelet reservoir and heavily impairs their overall hemostatic function. Reflecting this profound consumptive state, the patient's coagulation profile was markedly abnormal. The prothrombin time was notably prolonged at 16.2 seconds, well above the reference range of 11.0 to 13.5 seconds, and the activated partial thromboplastin time was also extended to 45.0 seconds. These prolonged coagulation parameters highlight a significantly compromised hemostatic capacity, firmly cementing the absolute contraindications to neuraxial anesthesia. Attempting a regional technique under these hematologic conditions carries an unacceptably high risk of accidental vascular puncture, leading to a spinal epidural hematoma. Hepatic involvement, a classic hallmark of severe preeclamptic progression, was clearly quantified through significant transaminitis. The aspartate aminotransferase was markedly elevated at 112 U/L, and the alanine aminotransferase was concurrently raised to 98 U/L. Finally, significant renal endothelial dysfunction

was aggressively manifested by severe proteinuria, documented as a +4 on the admission urinalysis. Collectively, the comprehensive metabolic and hematologic data within Table 2 elucidate a highly precarious clinical scenario characterized by simultaneous hematologic, hepatic, and renal distress.

Further laboratory investigations, encompassing comprehensive metabolic and hematologic profiling, revealed severe systemic derangements that were entirely consistent with a diagnosis of severe preeclampsia. The urgency of the case was ultimately cemented by the continuous fetal monitoring. The cardiotocography demonstrated a highly concerning baseline fetal heart rate of 165 beats per minute. This tachycardia was accompanied by an absent baseline variability and the presence of recurrent late decelerations. Together, these ominous cardiotocographic patterns were strictly indicative of profound fetal distress, necessitating immediate and definitive intervention.

**Table 2. Preoperative Laboratory and Coagulation Profiles**

Laboratory Parameter	Patient Value	Reference Range	Interpretation
Hemoglobin	9.2 g/dL	12.0 - 16.0 g/dL	Moderate Anemia
Hematocrit	28 percent	36 - 48 percent	Decreased
Platelet Count	42,000 / $\mu$ L	150,000 - 450,000 / $\mu$ L	Severe Thrombocytopenia
Prothrombin Time	16.2 seconds	11.0 - 13.5 seconds	Prolonged
Activated PTT	45.0 seconds	25.0 - 35.0 seconds	Prolonged
Aspartate Aminotransferase	112 U/L	8 - 33 U/L	Elevated
Alanine Aminotransferase	98 U/L	7 - 55 U/L	Elevated
Urinalysis (Protein)	+4	Negative	Severe Proteinuria

Given the presence of eclampsia, fetal distress, preterm labor, and absolute contraindications to neuraxial anesthesia related to severe thrombocytopenia, the decision was made to proceed with an emergency deep transperitoneal cesarean section. A modified general anesthesia rapid sequence induction was selected due to the advanced gestational age and elevated risk of pulmonary aspiration. In the operating theater, standard monitors were applied, and a 20-gauge radial arterial line was inserted under local anesthesia for continuous hemodynamic monitoring.

Adequate preoxygenation was achieved using a tight-fitting face mask for 3 minutes to extend safe apnea time. Due to institutional stock shortages, high-dose rocuronium was unavailable. Therefore, anesthetic induction was achieved with intravenous fentanyl 100  $\mu$ g, midazolam 3 mg, and propofol 100 mg, followed by atracurium 30 mg. Because atracurium possesses a slower onset time than succinylcholine or rocuronium, a modified rapid sequence approach was utilized. Cricoid pressure was immediately applied by a trained assistant upon loss of consciousness. To maintain oxygenation during the prolonged onset phase without increasing gastric insufflation risk, high-flow apneic oxygenation was maintained, and positive pressure ventilation was strictly avoided. After a 2.5-minute onset interval, direct laryngoscopy was performed, and the patient was successfully intubated on the first attempt using

a Macintosh size 3 blade. Fluid management was controlled to avoid exacerbating cerebral or pulmonary edema. A restrictive fluid strategy was employed, with a total of 500 mL of Lactated Ringer's administered intraoperatively. A norepinephrine infusion was maintained on standby but was ultimately not required.

Figure 1 delineates the comprehensive intraoperative hemodynamic timeline, providing a chronological quantification of the patient's cardiovascular stability throughout the emergency surgical intervention. Upon the patient's initial arrival in the operating room at 08:30, the baseline physiological parameters accurately reflected her severe preeclamptic state, demonstrating a hypertensive profile with a systolic blood pressure of 148 mmHg and a diastolic blood pressure of 95 mmHg. At this juncture, the baseline heart rate was recorded at 70 beats per minute, and peripheral oxygen saturation was optimal at 99 percent. Following the execution of the modified rapid sequence induction, a deliberate and carefully controlled attenuation of the patient's heightened sympathetic tone was achieved. In the immediate post-induction phase at 08:32, the systemic blood pressure appropriately decreased to a normotensive range of 115/72 mmHg, accompanied by a slight bradycardic shift to a heart rate of 65 beats per minute. Crucially, oxygen saturation peaked at 100 percent, validating the efficacy of the high-flow apneic oxygenation

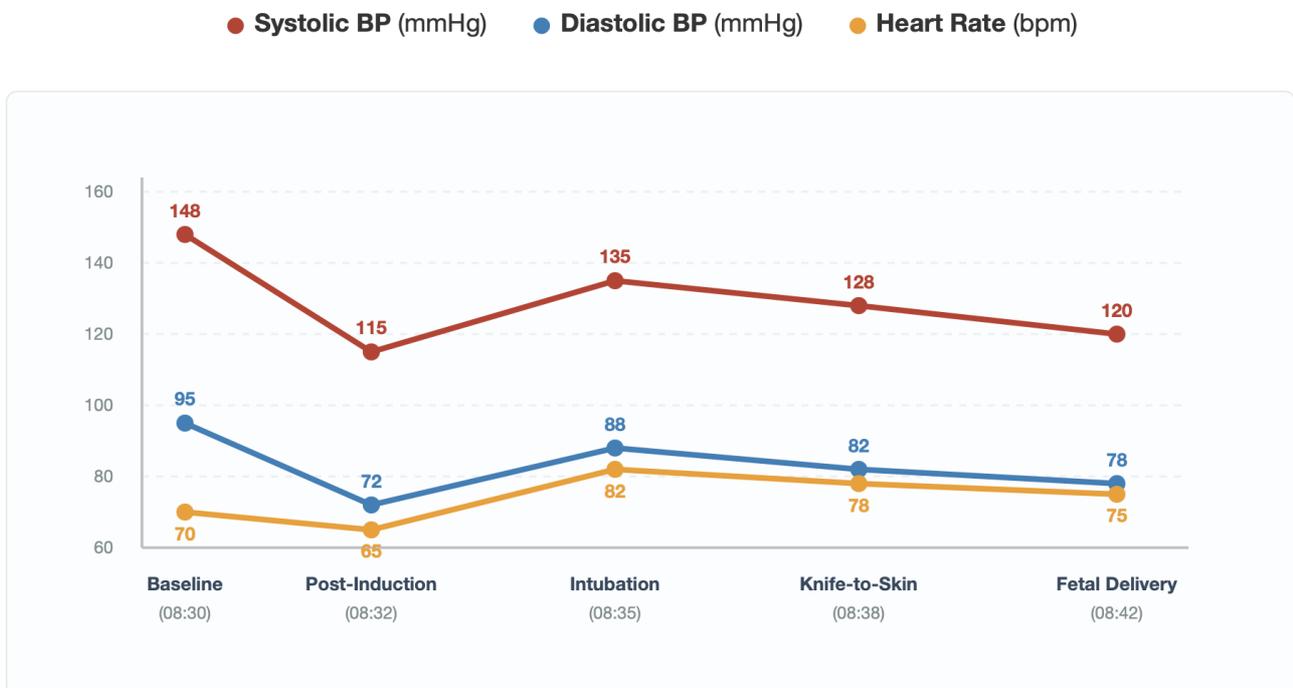
technique utilized during the prolonged 2.5-minute onset interval required for the intermediate-acting non-depolarizing agent, atracurium.

The most perilous phase of airway management—the first-pass endotracheal intubation occurring at 08:35—elicited an intentionally blunted sympathoadrenal response. During this critical maneuver, the blood pressure transiently elevated to 135/88 mmHg, and the heart rate peaked mildly at 82 beats per minute. This highly restrained physiological reaction unequivocally demonstrates the successful mitigation of the potentially fatal pressor response to laryngoscopy, thereby protecting the fragile cerebral vasculature of this eclamptic adolescent from catastrophic hypertensive spikes.

Subsequent surgical stimuli provoked minimal hemodynamic perturbations. At the knife-to-skin incision at 08:38, parameters stabilized at 128/82

mmHg with a heart rate of 78 beats per minute, and upon successful fetal delivery at 08:42, readings settled optimally at 120/78 mmHg and 75 beats per minute. Peripheral oxygen saturation remained flawless at 100 percent during the moment of delivery. Overall, the mean arterial pressure was rigorously maintained within a strict therapeutic window of 85 to 105 mmHg for the entire duration of the procedure. The absolute success of this pharmacological strategy and the restrictive fluid management approach is further underscored by the fact that a preemptively prepared norepinephrine infusion, kept on standby for anticipated hemodynamic collapse, was ultimately not required. This meticulously documented timeline confirms a remarkably hemodynamically stable intraoperative course despite the profound systemic derangements present at admission.

### Intraoperative Hemodynamic Trends



Note: Peripheral oxygen saturation (SpO<sub>2</sub>) remained highly stable between 99% and 100% across all documented clinical phases.

Figure 1. Intraoperative hemodynamic trends.

A live male infant was delivered 4 minutes after the uterine incision. The APGAR scores were 5 and 8 at 1 and 5 minutes, respectively. Following cord clamping, an oxytocin infusion was initiated to promote uterine tone. The mean arterial pressure was maintained between 85 and 105 mmHg intraoperatively. The

procedure was completed without surgical or anesthetic complications. Postoperatively, the patient was admitted to the intensive care unit for monitoring and received paracetamol and fentanyl for analgesia. Her postoperative course was uneventful (Table 3).

**Table 3. Diagnosis, Treatment, Follow-up, and Outcome**

Phase	Details & Interventions
<b>Primary Diagnosis</b>	<ul style="list-style-type: none"> <li>• <b>Severe Preeclampsia/Eclampsia:</b> Generalized tonic-clonic seizures.</li> <li>• <b>Severe Thrombocytopenia:</b> Platelet count of 42,000/<math>\mu</math>L (absolute contraindication to neuraxial anesthesia).</li> <li>• <b>Acute Fetal Distress:</b> Absent variability and recurrent late decelerations.</li> <li>• <b>Obstetric Status:</b> Preterm labor at 35 weeks gestation with premature rupture of membranes.</li> </ul>
<b>Intervention</b>	<ul style="list-style-type: none"> <li>• <b>Surgery:</b> Emergency deep transperitoneal cesarean section.</li> <li>• <b>Anesthetic Approach:</b> Modified general anesthesia rapid sequence induction (RSI).</li> <li>• <b>Pharmacology:</b> Fentanyl 100 <math>\mu</math>g, midazolam 3 mg, propofol 100 mg, and atracurium 30 mg.</li> <li>• <b>Airway Strategy:</b> Apneic oxygenation and continuous cricoid pressure for 2.5 minutes; successfully intubated on the first attempt (Macintosh size 3 blade).</li> <li>• <b>Hemodynamics:</b> Restrictive fluid strategy (500 mL Lactated Ringer's); mean arterial pressure maintained between 85-105 mmHg.</li> </ul>
<b>Follow-up</b>	<ul style="list-style-type: none"> <li>• Admitted to the intensive care unit (ICU) postoperatively for rigorous hemodynamic and neurological monitoring.</li> <li>• Maintained on standard analgesia protocol (paracetamol and fentanyl).</li> </ul>
<b>Outcome</b>	<ul style="list-style-type: none"> <li>• <b>Maternal:</b> Uneventful postoperative course with no surgical or anesthetic complications.</li> <li>• <b>Neonatal:</b> Live male infant delivered 4 minutes post-incision; APGAR scores of 5 (at 1 min) and 8 (at 5 min).</li> </ul>

### 3. Discussion

The anesthetic management of an eclamptic parturient is fundamentally an exercise in navigating severe, multisystem pathophysiological derangements. This case highlights not only the critical nature of the disease pathology itself but also underscores the absolute necessity of clinical

adaptability when standard pharmacological tools—specifically rapid-onset non-depolarizing neuromuscular blockers—are unavailable during high-risk obstetric emergencies.<sup>11</sup>

Eclampsia represents the most severe and clinically volatile end of the preeclampsia spectrum, classically characterized by the onset of generalized

tonic-clonic seizures in a patient with preeclampsia. The underlying pathophysiology driving this systemic crisis involves widespread and profound endothelial dysfunction, intense sympathetic vasospasm, and an exaggerated systemic inflammatory response that affects nearly every organ system (Figure 2).<sup>12</sup> In the central nervous system, this diffuse endothelial injury results in a critical loss of cerebral autoregulation. This failure allows systemic hypertension to translate directly into cerebral hyperperfusion, leading to endothelial leakage and subsequent vasogenic cerebral edema. This progressive cerebral swelling drastically lowers the seizure threshold, culminating in the eclamptic convulsion.

For the anesthesiologist, these systemic vascular changes manifest most alarmingly as a highly vulnerable and difficult airway. The capillary leak syndrome intimately associated with severe preeclampsia frequently extends into the upper respiratory tract, resulting in significant

pharyngolaryngeal edema. This fluid extravasation renders the upper airway tissues extremely friable and significantly reduces the functional glottic opening, making visualization during direct laryngoscopy inherently challenging.<sup>13</sup> Furthermore, the autonomic nervous system is in a state of hyperarousal. The sympathetic response to the nociceptive stimulus of direct laryngoscopy and endotracheal intubation in a patient with pre-existing endothelial dysfunction can precipitate catastrophic hypertensive crises. These sudden spikes in blood pressure carry a severe risk of causing intracranial hemorrhage or acute cardiogenic pulmonary edema. Therefore, the chosen induction sequence must accomplish two diametrically opposed but equally critical goals: it must definitively blunt the sympathoadrenal axis to protect the cerebral and cardiopulmonary systems, while simultaneously ensuring the rapid and definitive securing of the airway to prevent fatal pulmonary aspiration in a postictal pregnant patient.

### Pathophysiology of Eclampsia and Airway Vulnerability

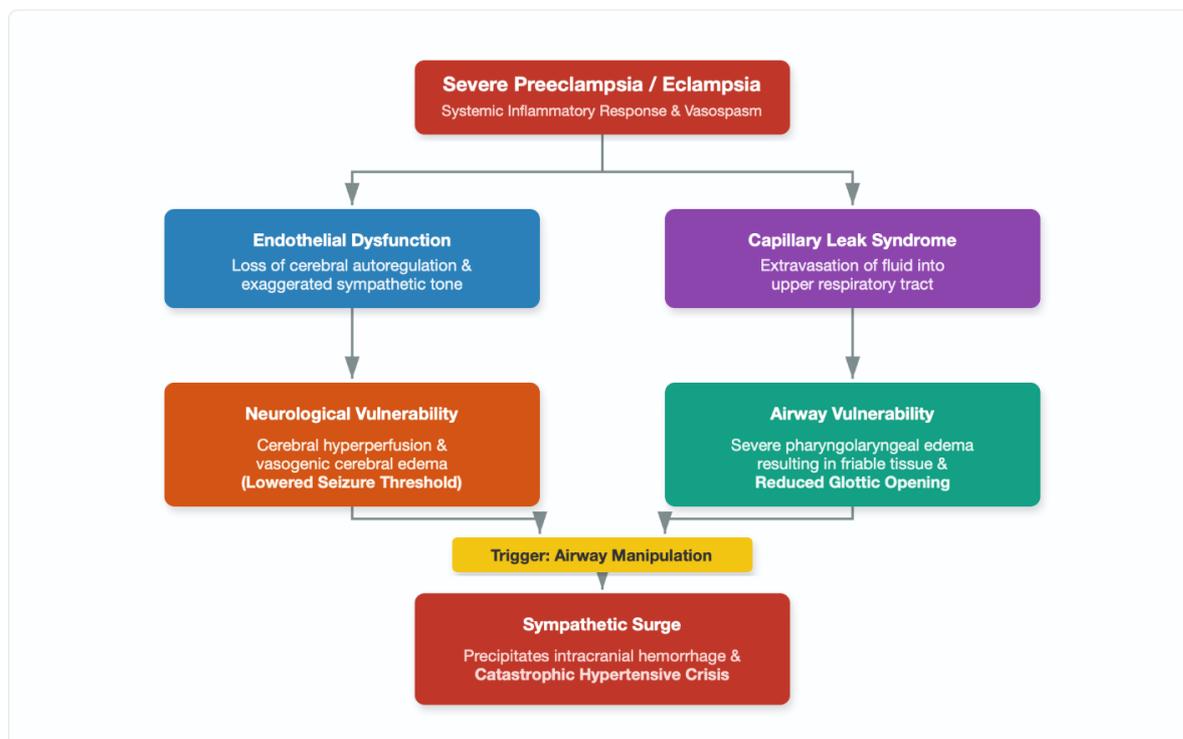


Figure 2. Pathophysiology of eclampsia and airway vulnerability.

The presence of maternal adolescence compounds this already severe risk profile. The adolescent vasculature and developing central nervous systems often exhibit a heightened and less predictable reactivity to nociceptive stimuli compared to adult gravidas. This amplified physiological response requires the anesthesiologist to employ meticulous and careful titration of induction agents; the goal is to ensure profound anesthesia and complete autonomic blunting without precipitating sudden cardiovascular collapse in a patient who already possesses a diminished circulating blood volume and reduced cardiovascular reserve.<sup>14</sup>

In modern obstetric practice, neuraxial anesthesia—most commonly a spinal or epidural block—is generally the preferred modality for cesarean deliveries, as it avoids the inherent risks of maternal airway manipulation and potential fetal depression associated with general anesthesia. However, in this specific clinical presentation, the presence of severe thrombocytopenia rendered neuraxial anesthesia absolutely contraindicated. The pathophysiology of thrombocytopenia in preeclampsia is primarily driven by microangiopathic hemolysis and rampant platelet consumption.<sup>15</sup> As systemic endothelial damage progresses, subendothelial collagen is exposed throughout the maternal microvasculature. This widespread exposure acts as a potent trigger for uncontrolled platelet aggregation and the extensive formation of microthrombi within the capillary beds. This active, consumptive coagulopathy not only severely depletes the total number of circulating platelets—evidenced in this patient by a critical count of 42,000/ $\mu\text{L}$ —but also significantly impairs the hemostatic function of the remaining platelets. Performing a subarachnoid or epidural block in this severely depleted hematologic setting carries an unacceptably high and potentially disastrous risk of accidental vascular puncture. Should a dural or epidural vein be compromised, the patient's inability to form a stable clot leads directly to the formation of a spinal epidural hematoma. Such a complication, expanding within the rigid, confined space of the

spinal canal, can rapidly cause irreversible spinal cord compression, ischemia, and ultimately, permanent paraplegia. Consequently, the anesthesiologist is compelled to abandon regional techniques and manage the immense risks associated with general anesthesia.

Given these profound systemic constraints, the selection of anesthetic agents reflects a highly tailored approach, heavily influenced by both the patient's precarious pathophysiology and the reality of institutional resource limitations. Propofol remains an ideal induction agent in the setting of eclampsia due to its potent anticonvulsant properties. Upon administration, it rapidly decreases both the cerebral metabolic rate of oxygen ( $\text{CMRO}_2$ ) and intracranial pressure (ICP). This action is highly beneficial as it directly counteracts the cerebral hyperperfusion and neuro-excitability associated with the eclamptic state.<sup>16</sup> Furthermore, the administration of fentanyl immediately prior to laryngoscopy was critical. Fentanyl effectively obtunds the pressor response to endotracheal intubation, thereby protecting the patient's fragile cerebral vasculature from sudden, potentially hemorrhagic hypertensive spikes.

The administration of 3 mg of midazolam in this specific scenario warrants careful clinical justification. While it is well understood that benzodiazepines readily cross the placental barrier and can cause neonatal central nervous system and respiratory depression, the maternal requirements in this acute crisis unequivocally superseded the potential neonatal risks. The patient was a highly stressed, postictal adolescent facing an emergency surgical intervention; she required immediate neurological stabilization. Midazolam provided necessary amnesia and anxiolysis, which synergistically lowered the required induction dose of propofol, thereby minimizing the risk of propofol-induced hypotension. Crucially, it also offered potent secondary anticonvulsant protection during the critical induction phase. The favorable 5-minute APGAR score of 8 strongly demonstrates that while neonatal respiratory depression was a valid concern, its effects were transient and easily

manageable with standard pediatric resuscitation protocols in the delivery room.<sup>17</sup>

The most notable departure from standard obstetric protocols in this case was the use of atracurium. In modern obstetric anesthesia, high-dose rocuronium (typically 1.0 - 1.2 mg/kg) is widely considered the gold standard alternative to succinylcholine for rapid sequence induction, providing optimal intubating conditions in approximately 60 seconds without raising intracranial pressure. However, in this instance, rocuronium was entirely unavailable due to persistent supply chain shortages. The traditional rapid-acting alternative, succinylcholine, was explicitly avoided due to its known side effects: the risk of transient but significant increases in intracranial pressure, and the potential to induce dangerous hyperkalemia in a neurologically vulnerable, postictal patient with potential subclinical renal impairment.

Therefore, the anesthetic team was forced to initiate a modified rapid sequence approach utilizing the intermediate-acting non-depolarizing agent, atracurium, at a dose of 30 mg. Atracurium is highly advantageous in preeclamptic patients because its degradation relies entirely on Hofmann elimination—a spontaneous, non-enzymatic chemical breakdown in the plasma—rendering its duration of action independent of renal or hepatic function, which are frequently impaired in severe preeclampsia.<sup>18</sup> However, its significant drawback is a much slower onset time compared to rocuronium or succinylcholine. To safely accommodate this delayed onset, the airway had to be meticulously managed during a 2.5-minute induction window. This was achieved through the rigid application of continuous cricoid pressure to physically occlude the esophagus, and the use of high-flow apneic oxygenation to maintain maternal oxygen saturation without initiating positive pressure ventilation. The strict avoidance of manual positive pressure ventilation was paramount; doing so prevented gastric insufflation, thereby significantly mitigating the risk of regurgitation and pulmonary aspiration during the

extended period of paralysis prior to securing the endotracheal tube.<sup>19</sup>

While this case provides significant clinical insight, the primary limitation of this study is its inherent reliance on a single case observation. The N=1 design naturally restricts the broader epidemiological generalizability of the modified induction sequence described. Furthermore, it must be acknowledged that the reliance on a prolonged induction interval introduces inherent risks of maternal hypoxia. While hypoxia was successfully avoided in this specific instance through the meticulous application of apneic oxygenation, it remains a critical concern, particularly if attempted in less optimized settings or by less experienced practitioners. To build upon these findings, future research must transition away from isolated single reports and move toward robust, retrospective cohort analyses. These larger studies are essential for statistically evaluating both maternal and fetal outcomes across diverse populations of eclamptic patients who are forced to undergo modified rapid sequence inductions utilizing intermediate-acting non-depolarizing neuromuscular blockers when the gold-standard rapid-acting agents are rendered unavailable by systemic resource constraints.<sup>20</sup>

#### **4. Conclusion**

This clinical case powerfully demonstrates that while standard rapid sequence induction utilizing rapid-onset paralytics remains the ideal and safest approach for securing the airway in obstetric emergencies complicated by absolute contraindications to neuraxial anesthesia, strict adherence to this ideal is not always possible. In resource-limited environments, a modified rapid sequence induction utilizing atracurium represents not merely a theoretical fallback, but a highly viable, clinically safe alternative. The success of this approach is entirely dependent upon the meticulous and precise execution of compensatory techniques. The careful, uninterrupted implementation of prolonged cricoid pressure, the strategic use of high-flow apneic oxygenation to bridge the delayed onset

time, and the precise, hemodynamically conscious titration of sympatholytic agents like fentanyl and midazolam were the key factors that allowed for profound physiological stability. Ultimately, this tailored strategy facilitated successful and atraumatic airway control in an actively eclamptic, severely thrombocytopenic adolescent. These findings emphatically highlight the absolute necessity of pharmacological adaptability, comprehensive pathophysiological understanding, and rigorous physiological monitoring for anesthesiologists operating in increasingly common resource-limited anesthetic environments.

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