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Neuroprotective General Anesthesia for Emergency Cesarean Section in a Patient with Obstructive Hydrocephalus from a Vestibular Schwannoma

Reza Ariestyawan Ramadhan[1]*, Isngadi[2], Buyung Hartiyo Laksono[2]

[1]Anesthesiology and Intensive Therapy Program, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia

[2]Anesthesia Consultant, Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia

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***Corresponding author:**
Reza Ariestyawan Ramadhan

E-mail address:
ramadhanrezamd@gmail.com

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A B S T R A C T

Introduction: The confluence of advanced pregnancy and a large intracranial neoplasm presents a profound clinical challenge. This report details the management of a parturient with a vestibular schwannoma causing obstructive hydrocephalus and critical intracranial hypertension (ICP), a scenario where standard obstetric anesthetic practices are absolutely contraindicated. **Case presentation:** A 35-year-old G3P1 parturient at 36 weeks gestation with progressive blindness from a vestibular schwannoma presented for an emergency cesarean section due to fetal compromise. With clear signs of severe ICP, general anesthesia was administered. Anesthesia was induced with propofol and atracurium and maintained with sevoflurane and a remifentanil infusion, a regimen selected for maternal neuroprotection and fetal safety. Invasive arterial and central venous pressure monitoring guided hemodynamic management to ensure cerebral perfusion. A healthy infant was delivered. The family declined postoperative neurosurgery; the patient was managed conservatively with medical therapy and discharged in stable condition, with long-term follow-up confirming favorable maternal and infant outcomes. **Conclusion:** This case demonstrates that a meticulously planned general anesthetic, centered on neuroprotective principles and guided by advanced physiological monitoring, can ensure a safe outcome for both mother and child in the face of critical intracranial hypertension. This success underscores the paramount importance of a deep pathophysiological understanding and seamless multidisciplinary collaboration.

1. Introduction

The coincidence of a primary central nervous system (CNS) tumor with pregnancy is a rare but formidable clinical challenge, estimated to occur in approximately 10 per 100,000 pregnancies.[1] The profound physiological adaptations of gestation create a unique environment that can dramatically alter the course of an intracranial neoplasm. Hormonal shifts, particularly elevated levels of progesterone and estrogen, combined with a 40-50% increase in intravascular volume and cardiac output, can accelerate tumor growth and exacerbate peritumoral vasogenic edema.[2] This may unmask a previously asymptomatic lesion or precipitate a rapid and severe worsening of existing neurological deficits, transforming a stable chronic condition into a life-threatening emergency over the course of the pregnancy.

Vestibular schwannomas (VS), benign, slow-growing tumors arising from the Schwann cells of the eighth cranial nerve, account for approximately 7% of all primary intracranial tumors.[3] While their symptomatic presentation during pregnancy is infrequent, their

growth can be significantly influenced by the gravid state, likely through a "dual-hit" mechanism involving stimulation of progesterone receptors on tumor cells and increased tumor vascularity and engorgement. When a vestibular schwannoma reaches a significant size, it can exert considerable mass effect within the tight confines of the posterior fossa. This can lead to compression of the brainstem and, critically, the fourth ventricle, obstructing the normal outflow of cerebrospinal fluid (CSF).[4] The resulting obstructive hydrocephalus leads to a dangerous and precipitous rise in intracranial pressure (ICP).

This scenario creates one of the most critical anesthetic dilemmas in modern obstetrics when the patient requires delivery. The presence of severely elevated ICP, a non-communicating hydrocephalus, and significant mass effect renders regional anesthesia techniques (spinal, epidural, or combined spinal-epidural), which are normally the preferred and safest methods for cesarean sections, absolutely contraindicated.[5] This contraindication is rooted in the high and unacceptable risk of precipitating fatal cerebral herniation. An intentional or inadvertent dural puncture would create a low-pressure outlet in the lumbar spine, establishing a steep pressure gradient between the cranial vault and the spinal canal.[6] In a patient with a posterior fossa mass and exhausted intracranial compliance, this gradient would almost certainly drive the cerebellar tonsils downward through the foramen magnum, causing brainstem compression, cardiovascular collapse, and death.[7] This is not a theoretical risk; case reports have documented this catastrophic complication.

Consequently, general anesthesia becomes the necessary and only viable approach. However, general anesthesia is not without its own significant risks in this patient population.[8] The potent sympathoadrenal stimulation associated with laryngoscopy and endotracheal intubation can cause a hypertensive surge, which in turn can catastrophically increase ICP and cerebral blood volume, potentially triggering herniation or intracranial hemorrhage.[9,10] Therefore, the anesthetic plan must be designed not merely as a means to facilitate surgery, but as an active neuroprotective intervention. This case report provides a comprehensive, detailed account of the successful application of established neuroprotective anesthetic principles to manage an emergency cesarean delivery in a parturient with this rare and life-threatening condition. We detail our strategy, which was predicated on optimizing cerebral physiology, ensuring fetal safety, and navigating a complex postoperative course involving a difficult ethical decision.

2. Case Presentation

A 35-year-old female, with a body weight of 68 kg and height of 155 cm (Body Mass Index: 28.3 kg/m²), Gravida 3, Para 1 (one prior term birth, one abortion), was urgently referred to our tertiary care center at 36 weeks and 5 days of gestation. She represented a confluence of obstetric emergency and impending neurological catastrophe. Her medical history was dominated by a devastating neurological decline catalyzed by her pregnancy. She reported a progressive loss of vision that began in her first trimester and accelerated dramatically during the seventh month, culminating in complete blindness, with an inability to perceive anything beyond bright light. This profound symptom was pathognomonic for chronic, severe papilledema resulting from massively elevated intracranial pressure (ICP). Further history revealed an episode of Bell's palsy three years prior with only partial recovery, an event that, in retrospect, was an early manifestation of cranial nerve involvement from the underlying intracranial mass.

The patient presented to our emergency department with uterine contractions and amniotic fluid leakage for 15 hours, confirming a diagnosis of premature rupture of membranes (PPROM). A non-reassuring fetal heart rate pattern on cardiotocography signaled significant fetal compromise, compelling the multidisciplinary team of obstetricians, anesthesiologists, and neurosurgeons to proceed with an emergency cesarean section. The pre-operative assessment painted a stark picture of a patient at the precipice of crisis. While her Glasgow Coma Scale (GCS) was 15/15, the profound vision loss pointed to a brain in a state of exhausted compliance. Her airway was deemed manageable, but the underlying neurological condition dictated the entire anesthetic approach. The full constellation of her

clinical findings on admission is meticulously detailed in

Table 1 summarizes the critical data points—from her obstetric status and alarming neurological symptoms to her vital signs and laboratory results—that collectively established the final diagnosis of a parturient with a suspected space-occupying lesion, severe ICP, anemia, and hyponatremia, requiring an emergency procedure under the highest level of anesthetic vigilance. Given the severe systemic disease that posed a constant threat to her life, the patient was classified as American Society of Anesthesiologists (ASA) physical status 3E. General anesthesia was unequivocally the only safe option.

Table 1. Summary of patient's clinical findings on admission.

CATEGORY	FINDING	DETAILS AND SIGNIFICANCE
Patient Demographics & History		
Patient Profile	Age	A 35-year-old female.
Gravidity and Parity	G/P Status	G3P1001Ab100.
Anthropometry	Body Mass Index	Weight: 68 kg; Height: 155 cm; BMI: 28.3 kg/m² (Overweight).
Past Medical History	Neurological	History of Bell's palsy three years prior with partial residual left facial paresis.
Clinical Presentation		
Presenting Complaint	Obstetric	Admitted with uterine contractions and leakage of amniotic fluid for 15 hours.
Obstetric Status	Diagnosis	Gestational Age: 36 weeks and 5 days with a diagnosis of Premature Rupture of Membranes (PPROM) and signs of fetal compromise.
Neurological Symptoms	Vision	Progressive bilateral vision loss that began during the first trimester, accelerating in the seventh month to culminate in complete blindness (light perception only).
Physical Examination		
Vital Signs	Hemodynamics & Respiration	**Blood Pressure:** 124/86 mmHg. **Heart Rate:** 90 beats/min. **Respiratory Rate:** 18 breaths/min. **SpO_2:** 88% on Room Air, indicating hypoxemia.
Airway Assessment	Intubation Predictors	Mallampati Class 2, Thyromental Distance 6 cm, and normal neck mobility, suggesting a manageable airway for intubation.
Neurological Status	Consciousness & Cranial Nerves	**Level of Consciousness:** Alert, with a Glasgow Coma Scale (GCS) of 15 (E4V5M6). **Cranial Nerves:** Documented bilateral blindness and a positive Bell's sign on the left side.
Fetal Assessment	Condition	Fetal heart rate was 130-135 bpm, with an estimated fetal weight of 2600g.
Investigations		
Laboratory Findings	Bloodwork	**Hematology:** Hemoglobin of 10.3 g/dL, indicating anemia. **Biochemistry:** Serum sodium of 129 mmol/L, indicating hyponatremia. Coagulation profile, renal function, and glucose levels were within normal limits.
Imaging (Post-operative)	CT Scan	A CT scan confirmed a large, solid, extra-axial mass in the left cerebellopontine angle, consistent with a vestibular schwannoma. **Critical Complications:** The mass caused moderate obstructive hydrocephalus, significant cerebral edema, and upward transtentorial herniation.
Pre-operative Assessment		
Final Diagnoses	Summary	1. G3P1001Ab100 at 36-37 weeks with PPROM & Fetal Compromise. 2. Vision loss secondary to a suspected cerebral space-occupying lesion. 3. Anemia. 4. Hyponatremia.
ASA Physical Status	Risk Classification	Classified as ASA 3E, denoting a patient with a severe systemic disease that is a constant threat to life, requiring an emergency procedure.

In the operating theater, the patient was positioned supine with a 15-degree left lateral tilt to obviate aortocaval compression and a 15-degree head-up tilt to facilitate cerebral venous drainage, a simple yet vital maneuver to mitigate ICP. After applying standard monitors, our strategy hinged on advanced invasive monitoring to navigate the precarious balance of maternal cerebral perfusion and fetal well-being. A 20-gauge arterial line was placed in the left radial artery for continuous, beat-to-beat blood pressure monitoring, and a triple-lumen central venous catheter was inserted into the right internal jugular vein. This allowed for

precise, real-time hemodynamic assessment, guiding our every intervention.

Following 5 minutes of pre-oxygenation, general anesthesia was induced with a slow intravenous injection of propofol 100 mg (~1.5 mg/kg) and atracurium 25 mg (~0.37 mg/kg) to ensure hemodynamic stability. Laryngoscopy was performed gently, revealing a Cormack-Lehane Grade 1 view, and the trachea was intubated with a 7.0 mm cuffed endotracheal tube. Anesthesia was maintained with sevoflurane (0.8-1.0 MAC) and a continuous remifentanil infusion starting at 0.1 mcg/kg/min. Ventilation was mechanically controlled to maintain normocapnia, with an end-tidal CO_2 ($EtCO_2$) target of 35-38 mmHg.

The entire anesthetic plan was structured around the core challenges presented by the patient's pathophysiology. Table 2 provides a detailed summary of these challenges and the specific interventions deployed to counteract them. As shown in the table, our primary concern was the elevated ICP, which necessitated the choice of general anesthesia over neuraxial techniques to avoid the catastrophic risk of cerebral herniation. The induction was carefully designed to blunt the hypertensive response to intubation, and our maintenance strategy focused on preserving cerebral perfusion by meticulously controlling MAP and $EtCO_2$. Every step, from the controlled oxytocin infusion to the plan for a smooth emergence, was a calculated measure to maintain stability.

The surgery proceeded smoothly, and a healthy male infant was delivered 12 minutes after induction, with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. After the umbilical cord was clamped, fentanyl 150 mcg was administered. A controlled infusion of oxytocin 20 IU in 500 mL of Ringer's Lactate was started to prevent uterine atony while avoiding the profound hypotension associated with an oxytocin bolus.

The intraoperative hemodynamic course, depicted in Figure 1, stands as a testament to the success of this strategy. The graph clearly shows the mean arterial pressure (MAP) and oxygen saturation (SpO_2) throughout the timeline of anesthesia and surgery. The MAP was successfully maintained within a narrow, stable range of 75-90 mmHg, avoiding the dangerous peaks and troughs that could have compromised cerebral perfusion. This remarkable stability, from induction through delivery and to the end of surgery, was the central achievement of the anesthetic management.

Table 2. Summary of anesthetic challenges and interventions.

CLINICAL CHALLENGE	INTERVENTION	RATIONALE
Elevated ICP / Herniation Risk	Choice of General Anesthesia over Neuraxial Techniques	To avoid the catastrophic risk of cerebral herniation associated with an inadvertent dural puncture in a patient with exhausted intracranial compliance.
Hypertensive Response to Intubation	Induction with Propofol and Remifentanil; Gentle laryngoscopy	To blunt the sympathoadrenal response. Propofol decreases cerebral metabolic rate, cerebral blood flow, and ICP, providing neuroprotection during a high-stimulus period.
Maintaining Cerebral Perfusion	Invasive arterial line monitoring; MAP target 75-90 mmHg	To ensure adequate Cerebral Perfusion Pressure (CPP = MAP - ICP). In the setting of high ICP, maintaining MAP is critical to prevent cerebral ischemia.
Risk of Increased ICP from Hypercapnia	Controlled mechanical ventilation; Target $EtCO_2$ 35-38 mmHg	To maintain strict normocapnia. Hypercapnia is a potent cerebral vasodilator that would dangerously increase ICP.
Postpartum Hemorrhage vs. Hemodynamic Stability	Controlled oxytocin infusion (not a bolus)	To achieve uterine tone while avoiding the well-known hypotensive effect of an oxytocin bolus, which would have compromised CPP.
Need for Smooth Emergence	Use of ultra-short-acting Remifentanil; Extubation only when fully awake	To prevent coughing or straining, which elevates ICP, and to allow for a rapid and clear postoperative neurological assessment.

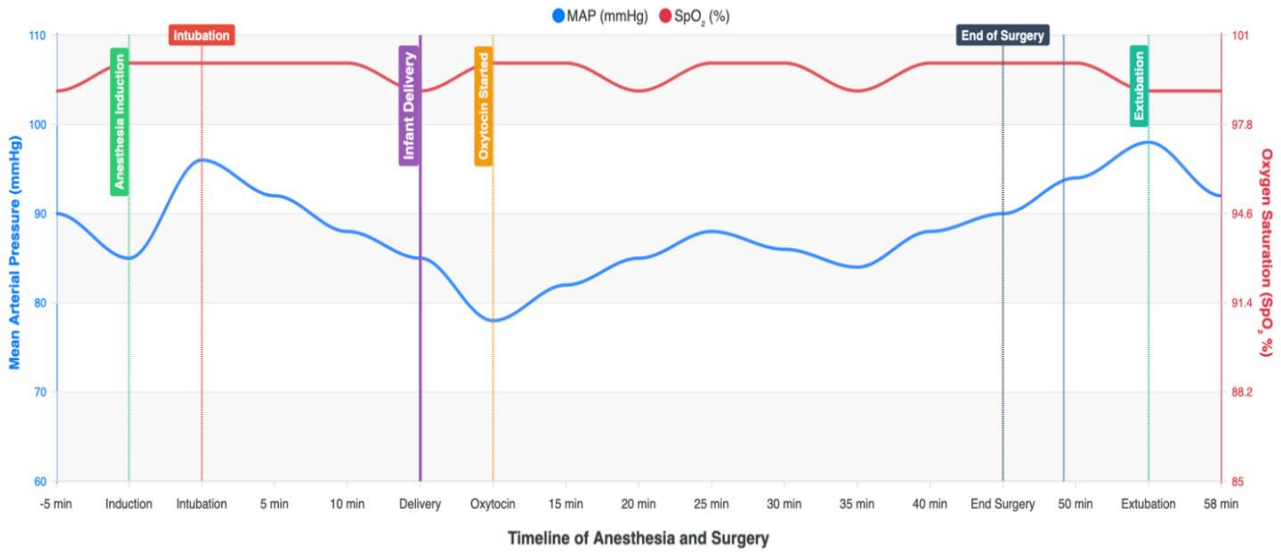

Figure 1. Intraoperative hemodynamic monitoring.

At the conclusion of surgery, the remifentanil infusion was discontinued, allowing for a rapid return of spontaneous ventilation. After the patient was fully awake and following commands, the trachea was extubated smoothly, without any coughing or straining that could have precipitated a dangerous surge in ICP. Postoperatively, the patient was transferred to the ICU for intensive monitoring. A CT scan on post-op day 1 confirmed a large extra-axial mass in the left cerebellopontine angle, consistent with a vestibular schwannoma, causing moderate obstructive hydrocephalus and upward transtentorial herniation. Neurosurgeons recommended an urgent ventriculoperitoneal (VP) shunt; however, the patient and her family declined the intervention. Consequently, management shifted to a conservative medical approach focused on controlling ICP with IV dexamethasone and oral acetazolamide. The patient remained neurologically stable and was successfully discharged home on post-operative day 5. Long-term follow-up at one year confirmed the infant's normal neurodevelopment and the mother's stable neurological status, albeit with no improvement in her vision.

3. Discussion

The successful management of this patient represents a triumph of multidisciplinary collaboration and a deep, applied understanding of competing physiological principles.[9] This case is not merely a report of a rare clinical encounter; it is a profound illustration of how the physiological adaptations of pregnancy can act as a catalyst, transforming a quiescent intracranial pathology into a fulminant neurological crisis, and how a meticulously tailored anesthetic can navigate this storm. The discussion of this case warrants a detailed exploration of the underlying pathophysiology, the theoretical foundations of the anesthetic choices made, and the interpretation of the clinical course as a whole. To fully appreciate the gravity of the clinical situation, one must first dissect the intricate pathophysiological cascade that led to this patient's presentation. The journey began not with the pregnancy, but years earlier with the slow, insidious growth of a vestibular schwannoma.[10]

Vestibular schwannomas are benign tumors arising from the Schwann cells that myelinate the vestibular portion of the eighth cranial nerve (CN VIII).[11] Their typical location is the cerebellopontine angle (CPA), a crowded and neurologically eloquent anatomical space bordered by the petrous part of the temporal bone, the cerebellum, and the pons. As the tumor grows, it does not invade but rather compresses and displaces the surrounding structures.[11] Initially, it affects its nerve of origin, causing hearing loss, tinnitus, and vertigo. As it

enlarges, it stretches and compresses the adjacent facial nerve (CN VII), which travels with CN VIII through the internal auditory canal. This direct compression explains the patient's prior history of Bell's palsy, which was almost certainly a misdiagnosed early sign of the schwannoma.[11] With further growth, the tumor can impact the trigeminal nerve (CN V), causing facial numbness, and eventually compress the brainstem and cerebellum, leading to ataxia and other long-tract signs.

While the tumor's growth is typically indolent over many years, pregnancy introduced two powerful biological accelerants, the "dual-hit" that precipitated the crisis.[12] The hormonal milieu of pregnancy, particularly the massively elevated levels of progesterone, played a critical role. Vestibular schwannomas are known to express progesterone receptors (PR) on their cell surfaces.[12] The binding of progesterone to these receptors is believed to activate intracellular signaling pathways that promote cellular proliferation and tumor growth. While the role of estrogen is less defined, it may also contribute by promoting angiogenesis (the formation of new blood vessels) within the tumor, further facilitating its expansion. This hormonal stimulation effectively took a slow-smoldering fire and poured gasoline on it.

Concurrently, pregnancy induces profound cardiovascular changes. Maternal blood volume increases by 40-50%, and cardiac output rises significantly to meet the metabolic demands of the fetus and placenta.[13] This hyperdynamic, hypervolemic state leads to increased vascularity and engorgement of all tissues, including the tumor itself. More importantly, it exacerbates the formation of vasogenic edema. Unlike the brain parenchyma, the capillaries within the schwannoma lack the tight junctions that form the blood-brain barrier. This makes them inherently "leaky." The increased hydrostatic pressure from the hypervolemic state forces plasma fluid out of these incompetent vessels and into the surrounding brain tissue, creating a large zone of vasogenic edema. In many cases, this edema contributes more to the overall mass effect and the patient's symptoms than the solid tumor itself.[13]

The combined effect of hormonally-driven tumor growth and hemodynamically-driven edema led to a critical increase in the volume of the posterior fossa contents. This is where the Monro-Kellie doctrine becomes centrally important. This doctrine states that the cranial vault is a rigid, in-expandable box containing three components: brain parenchyma, cerebrospinal fluid (CSF), and blood.[14] An increase in the volume of one component must be compensated by a decrease in the volume of the others to keep intracranial pressure (ICP) stable.

Initially, the brain compensates for the growing mass by displacing CSF from the cranial vault into the spinal canal and by compressing the low-pressure venous blood system. For a time, ICP remains normal.[14] However, this patient's tumor grew to the point where it physically compressed and effaced the fourth ventricle, blocking the outflow of CSF from the ventricular system into the subarachnoid space. This created a non-communicating or obstructive hydrocephalus. CSF produced in the lateral and third ventricles could not escape, leading to a massive buildup of pressure upstream and ventricular enlargement.[15]

At this stage, all compensatory mechanisms were exhausted. The patient was on the vertical, decompensated portion of the pressure-volume curve, where any minuscule increase in intracranial volume would cause a logarithmic and catastrophic rise in ICP. It was this sustained, critically high ICP that led to her most devastating symptom: blindness. The pressure was transmitted along the optic nerve sheaths, causing chronic, severe papilledema (swelling of the optic disc). This swelling physically compresses the axons of the optic nerve, impeding axoplasmic flow and leading to ischemic damage and, eventually, irreversible optic atrophy.[15] The CT finding of upward transtentorial herniation—where the cerebellum is forced upward through the tentorial notch—is a grim testament to the magnitude of the pressure gradient that had developed within the posterior fossa.

The anesthetic management was, therefore, not simply about facilitating surgery but about actively defending the brain from a state of extreme vulnerability.[16] The decision to pursue general anesthesia was not a preference; it was an absolute necessity. In a patient with an obstructing posterior

fossa mass and critically high ICP, performing a spinal or epidural anesthetic would be akin to pulling the plug on a bathtub with the faucet running full blast. An intentional dural puncture for a spinal, or an inadvertent one during an epidural (a known risk of 1-2%), would create a low-pressure outlet in the lumbar spine. This would establish a steep pressure gradient between the high-pressure cranium and the low-pressure spinal canal. This gradient would instantly drive the brainstem and cerebellar tonsils downward through the foramen magnum, causing fatal brainstem compression and cardiovascular collapse. This is the mechanism of "coning," and it is rapid, irreversible, and fatal.[16] The risk was so certain and so catastrophic that neuraxial techniques were dismissed without consideration.

Our choice of general anesthetic agents was a carefully orchestrated pharmacological symphony, with each drug selected for its specific, favorable effects on cerebral physiology and fetal well-being. The induction of anesthesia is one of the most dangerous periods for a patient with high ICP.[17] The intense sympathetic stimulation of laryngoscopy and intubation can cause a surge in heart rate and blood pressure, which directly translates to an increase in cerebral blood volume and ICP. Our strategy to mitigate this was twofold. First, we used propofol, an agent that is profoundly neuroprotective. It decreases the cerebral metabolic rate of oxygen ($CMRO_2$), and through a mechanism of flow-metabolism coupling, this leads to cerebral vasoconstriction and a reduction in cerebral blood flow and ICP. Second, we co-administered a remifentanil infusion. This potent, ultra-short-acting opioid is exceptionally effective at blunting the hemodynamic response to noxious stimuli like intubation. By combining these agents and administering them slowly, we were able to induce anesthesia while maintaining hemodynamic stability, effectively navigating this period of high risk.[17]

During the maintenance phase, the goal was to provide amnesia, analgesia, and muscle relaxation while having the least possible negative impact on cerebral dynamics. We chose low-dose sevoflurane (<1.0 MAC). All volatile anesthetics are cerebral vasodilators, an effect that can be detrimental in patients with high ICP.[18] However, at concentrations below 1.0 MAC, this effect is minimized, and the drug's ability to reduce $CMRO_2$ offers a degree of neuroprotection. The cornerstone of our maintenance technique was the continuation of the remifentanil infusion. Its rapid metabolism by non-specific esterases means its effect is profound yet easily titratable and quickly terminated. This allowed us to keep the sevoflurane concentration low while ensuring the patient was adequately anesthetized. Crucially, this rapid metabolism also occurs in the fetus, meaning the infant is born with minimal respiratory depression, as evidenced by the excellent Apgar scores. For neuromuscular blockade, atracurium was chosen for its intermediate duration and its organ-independent Hofmann elimination, making its clearance predictable and preventing accumulation in either the mother or fetus.

Beyond pharmacology, the management rested on the meticulous control of physiology, guided by invasive monitoring. The fundamental goal of neuroanesthesia is to protect the brain from secondary ischemic injury. This is achieved by maintaining an adequate CPP, defined by the equation CPP = MAP - ICP. Since our patient's ICP was known to be critically high and could not be directly measured, our only recourse was to meticulously control the MAP. We targeted a MAP of 75-90 mmHg, a level high enough to ensure adequate perfusion pressure to the brain, yet not so high as to exacerbate cerebral edema or risk hemorrhage. The continuous arterial line was indispensable for this task.

The relationship between arterial carbon dioxide tension ($PaCO_2$) and cerebral blood flow is steep and profound. Hypercapnia is a potent cerebral vasodilator that would have dangerously increased ICP and cerebral blood volume in this patient.[19] Conversely, aggressive hyperventilation to induce hypocapnia, once a common practice, is now largely avoided. While it does cause cerebral vasoconstriction and can rapidly lower ICP, it carries a significant risk of causing or worsening cerebral ischemia. Our strategy, therefore, was to maintain strict normocapnia ($EtCO_2$ 35-38 mmHg), ensuring a stable and appropriate level of cerebral blood flow. Fluid therapy in these patients is a delicate balancing act. Hypovolemia can lead to hypotension

and compromise CPP, while hypervolemia can worsen cerebral edema. The use of the central venous catheter to guide fluid administration was critical. We aimed for a state of euvolemia, replacing surgical losses and providing maintenance fluids to ensure adequate cardiac preload without fluid overloading the patient.[19]

The postoperative period introduced a new and unexpected challenge: the family's refusal of the life-saving VP shunt surgery.[20] This decision, rooted in personal beliefs, transformed the patient's trajectory from one of definitive surgical correction to one of conservative medical management and highlighted the primacy of patient autonomy in modern medicine. Our therapeutic strategy had to pivot entirely. The goal became to lower ICP using medical means alone. This was achieved with a two-pronged attack on intracranial volume. Dexamethasone is highly effective at reducing vasogenic edema, the type of edema specifically associated with brain tumors. It works by stabilizing the incompetent capillary membranes within the tumor, reducing their leakiness and thereby decreasing the volume of the edematous fluid surrounding the mass.[20] Also, we used Acetazolamide**,** a carbonic anhydrase inhibitor. By blocking this enzyme in the choroid plexus, it reduces the rate of CSF production by up to 50%. By simultaneously targeting the two main contributors to the patient's increased intracranial volume—the peritumoral edema (with dexamethasone) and the CSF volume (with acetazolamide)—we were able to successfully lower her ICP, stabilize her neurological condition, and facilitate a safe discharge. The success of this conservative regimen in such a critical case is a remarkable finding and underscores the efficacy of these medical therapies when surgical options are not pursued.

4. Conclusion

This case report provides a detailed narrative of the successful management of one of the most challenging scenarios in obstetric anesthesiology: an emergency cesarean section in a parturient with a massive intracranial tumor and critically elevated intracranial pressure. The favorable outcome for both mother and child was not a matter of chance but the direct result of a proactive, pathophysiology-based strategy. It powerfully demonstrates that when neuraxial techniques are absolutely contraindicated, a meticulously planned and executed general anesthetic, founded on robust neuroprotective principles, can safely navigate the treacherous physiological landscape. The careful selection of anesthetic agents to protect the maternal brain while ensuring fetal safety, the indispensable role of vigilant, invasive physiological monitoring to optimize cerebral perfusion, and a technique designed at every step to avoid iatrogenic harm were paramount. Ultimately, this case stands as a compelling testament to the fact that success in the most complex clinical situations is achieved through a deep and nuanced understanding of the underlying disease process and the seamless, collaborative, and adaptable efforts of a dedicated multidisciplinary team.

5. References

1. Song G, Zhang B, Tang Y, Geng H, Wu X, Zhou Y, et al. Comparing surgical outcomes of the semisitting versus lateral position in large vestibular schwannoma surgery: a randomized clinical trial. Neurosurg Rev. 2025; 48(1): 472.
2. Bathla G, Mehta PM, Soni N, Johnson M, Benson JC, Messina SA, et al. Evaluation of vestibular schwannoma size across time: How well do the experts perform and what can be improved? AJNR Am J Neuroradiol. 2025; 46(6): 1249–54.
3. Sandoval Camacho R, Sebastian J, Lloyd SKW. Anaesthesia for resection of vestibular schwannoma. BJA Educ. 2025; 25(7): 273–80.
4. Hasegawa T, Kato T, Ishikawa T, Naito T, Mizuno A, Sakai Y, et al. Incidence of rare malignant transformation in vestibular schwannomas treated with stereotactic radiosurgery: a single-institution analysis of 1,061 cases. J Neurooncol. 2025; 173(3): 695–705.
5. Bao M, He Y, Tao Y, Liu L, Li Y, Zhu Y, et al. Large vestibular schwannomas presenting in the late state of pregnancy: a case report and literature review. Front Neurol. 2023; 14: 1270989.

6. Shah KJ, Chamoun RB. Large vestibular schwannomas presenting during pregnancy: Management strategies. J Neurol Surg B Skull Base. 2014; 75(3): 214–20.
7. Shankar V, Ghosh S, Sai VH. Vestibular schwannoma in pregnancy: When case reports become clinical compass. Neurochirurgie. 2025; 71(4): 101687.
8. Al-Mufargi Y, AlHosni A, Al-Kalbani S, Al-Saadi T. Management of vestibular schwannoma during pregnancy: a systematic review. Neurochirurgie. 2025; 71(3): 101643.
9. Huang JL, Khalid H, Chessman R, Barnes V, Martin AJ, Patel PM. Vestibular schwannomas in pregnancy: a case series. J Neurol Surg B Skull Base. 2025.
10. Akella SS, Mattson JN, Moreira NN. Vestibular Schwannoma growth during pregnancy. Proc Obstet Gynecol. 2019; 9(1): 1–9.
11. Kadir V, Begum J, Kuberan A, Singh S, Parthasarathy R, Sahu RN. Vestibular schwannoma unveiled by pregnancy: a case report and literature review. Eur J Obstet Gynecol Reprod Biol. 2024; 299: 124–30.
12. Hong S, Park J-Y, Choi S, Kim MJ. Brun's nystagmus with cerebellopontine angle schwannoma. Res Vestib Sci. 2023; 22(2): 57–8.
13. Isik OG, Ing C. Maternal exposure to general anesthesia and labor epidural analgesia during pregnancy and delivery, and subsequent neurodevelopmental outcomes in children. Int J Obstet Anesth. 2025; 61(104318): 104318.
14. Karataş SŞ, Bulut OK, Öner SF. The effect of different information methods on patients' anxiety and preference for spinal versus general anesthesia in a Turkish cohort of women scheduled for cesarean delivery: a prospective observational study. Int J Obstet Anesth. 2025; 63(104689): 104689.
15. Hannon S, Elabassy O, Free R, Afridi A, Ffrench-O'Carroll R. The role of human factors in the decision to administer general anaesthesia for emergency caesarean deliveries: a qualitative interview study of anaesthesia trainees in Ireland. Int J Obstet Anesth. 2025; 63(104696): 104696.
16. Ansari JR, Smiley RM. Calcium as a uterotonic: cheap, available, and (maybe) effective? Int J Obstet Anesth. 2025; 63(104711): 104711.
17. Kielty J, McKeating K. Radiofrequency treatment of a pelvic schwannoma in pregnancy. Int J Obstet Anesth. 2020; 43: 13–4.
18. Ribeiro FV, Sousa MP, Ferreira MY, Andreão FF, Palavani LB, Fabrini Paleare LF, et al. Salvage surgery for vestibular schwannomas after failed stereotactic radiosurgery or radiation: a systematic review and meta-analysis. J Clin Neurosci. 2025; 137(111330): 111330.
19. Nam SI. Diagnosis and management of vestibular schwannoma: Focus on dizziness. Res Vestib Sci. 2021; 20(4): 119–25.
20. Weston AR, Dibble LE, Fino P, Lisonbee R, Hoppes C, Loyd BJ. Recovery of turning speed in patients after vestibular schwannoma resection. J Vestib Res. 2024; 34(2–3): 145–57.