

CASE REPORT

Neuroprotective General Anesthesia and Invasive Hemodynamic Monitoring for Emergency Cesarean Section in a Parturient with a Giant Vestibular Schwannoma and Obstructive Hydrocephalus: An Observational Analytic Case Study

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ABSTRACT

Background. A giant vestibular schwannoma with obstructive hydrocephalus in late pregnancy is a neuro-obstetric dilemma: neuraxial anesthesia risks tonsillar herniation, and general anesthesia must protect cerebral perfusion.

Objective. To describe the anesthetic management, invasive hemodynamic course and conservative postoperative treatment of a parturient with a Koos Grade IV vestibular schwannoma, obstructive hydrocephalus and complete visual loss who needed emergency cesarean section.

Methods. This single case report follows the CARE guideline and was approved by an ethics committee (2025/145). A 35-year-old woman (Gravida 3 Para 1 Abortus 1) at 36 weeks plus 5 days presented with membrane rupture, fetal compromise and blindness from chronic papilledema. Neuraxial anesthesia was avoided. General anesthesia used propofol, atracurium, a remifentanyl infusion and sevoflurane, with normocapnia, a controlled oxytocin infusion, and invasive arterial and central venous monitoring.

Results. Mean arterial pressure ranged from 80.3 to 102.7 mmHg (baseline 98.7 mmHg; largest fall -18.6%), and end-tidal carbon dioxide from 35 to 38 mmHg. A male infant (2,600 g; Apgar 8 and 9) was delivered at minute 12, and the patient was extubated awake without coughing. Computed tomography showed a 4.2 × 3.8 × 3.5 cm schwannoma with upward herniation. The family declined shunting; dexamethasone and acetazolamide were given, and she was discharged on day 5. At one year her visual loss was permanent and the infant developed normally.

Conclusion. In this patient, slow-induction general anesthesia with invasive monitoring was followed by hemodynamic stability and a healthy neonate, and medical therapy by stabilization after neurosurgery was declined. One case cannot establish efficacy.

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1. Introduction

A primary tumor of the central nervous system that coincides with advanced pregnancy is rare and carries high stakes. It occurs in approximately ten per one hundred thousand pregnancies.¹ The hormonal and physiological adaptations of pregnancy create a unique biological milieu, and this milieu can change the natural history, the microvascular architecture and the clinical course of a pre-existing intracranial lesion.² Circulating progesterone and estrogen rise sharply. Maternal intravascular blood volume and resting cardiac output expand by forty to fifty percent. These effects act together to accelerate tumor cell proliferation, promote pathological angiogenesis and trigger severe peritumoral vasogenic edema.³ A mass that was indolent or quiescent before pregnancy can therefore enlarge abruptly in the second or third trimester. A stable,

subclinical condition becomes a life-threatening neuro-obstetric emergency, with fulminant intracranial hypertension, cranial neuropathy and a threat to both mother and fetus.⁴

Vestibular schwannomas are benign, slow-growing tumors. They arise from the myelinating Schwann cells of the superior or inferior vestibular division of the eighth cranial nerve and make up approximately seven to eight percent of all primary intracranial tumors in adults.⁵ Symptoms during gestation are relatively infrequent. Even so, pregnancy can speed expansion of the tumor through a two-hit endocrine-hemodynamic mechanism.⁶ Cultured human vestibular schwannoma specimens express abundant functional progesterone receptors. When gestational progesterone surges, receptor activation stimulates transcriptional signaling cascades that govern cellular hypertrophy and protein synthesis.⁷ At the same time, gestational hypervolemia raises the hydrostatic

capillary filtration pressure across immature, structurally defective tumor capillaries that lack tight endothelial zonulae occludentes. The result is extensive vasogenic edema in the surrounding white matter tracts.⁸

A vestibular schwannoma that reaches significant size inside the confined cerebellopontine angle presses on neighboring eloquent neurovascular structures, including the facial nerve, the trigeminal nerve and the anterolateral brainstem.⁹ Growth continues across the cerebellomedullary cistern. The fourth ventricle is compressed and displaced, which blocks the physiological outflow of cerebrospinal fluid from the fourth ventricle through the lateral apertures of Luschka and the median aperture of Magendie and compresses the lower aqueduct of Sylvius.¹⁰ Severe non-communicating (obstructive) hydrocephalus follows. The third and lateral ventricles distend upstream, and intracranial pressure climbs steeply.

At this stage intracranial compliance is entirely exhausted. The patient sits on the steep, vertical limb of the Monro-Kellie volume-pressure relationship, where even a minute increase in intracranial volume produces an exponential rise in pressure. Sustained intracranial hypertension is transmitted along the optic nerve sheaths. It disrupts axoplasmic transport across the lamina cribrosa, causes severe chronic papilledema and secondary microvascular ischemia of the optic nerve, and can end in irreversible bilateral blindness.¹¹⁻¹³

A parturient with severe obstructive hydrocephalus and an urgent obstetric indication for delivery presents one of the most hazardous problems in modern obstetric anesthesia. Standard practice strongly favors neuraxial blockade. Spinal, epidural and combined spinal-epidural techniques avoid the airway hazards of general anesthesia and the transfer of depressant drugs to the fetus. With severe intracranial hypertension, non-communicating hydrocephalus and posterior fossa mass effect, however, neuraxial techniques are absolutely contraindicated. A dural puncture, whether deliberate in spinal anesthesia or inadvertent during epidural placement, leaves a low-resistance drainage fistula in the lumbar theca and sets up a steep hydrostatic gradient between the supra-tentorial and infra-tentorial compartments and the spinal canal. When a posterior fossa lesion obstructs the pathway and compliance is exhausted, that gradient can force the cerebellar tonsils down through the foramen magnum. The consequences can be catastrophic: tonsillar coning, acute medullary compression, cardiovascular collapse and immediate maternal death.¹⁴

General anesthesia is therefore the only permissible technique for surgical delivery in this situation. It brings physiological hazards of its own. Direct laryngoscopy and tracheal intubation provoke an intense sympathoadrenal reflex, so systemic arterial pressure and maternal heart rate surge and cerebral blood volume and intracranial pressure rise suddenly. Upward or downward transtentorial herniation or intratumoral hemorrhage may follow. Maternal hypercapnia from hypoventilation causes potent cerebral arteriolar vasodilation and dangerously raises intracranial pressure. Aggressive hyperventilation does the opposite, because excessive vasoconstriction puts the maternal brain at risk of ischemia. Hypotension during surgical delivery, especially after rapid oxytocin administration, severely reduces cerebral perfusion pressure and exposes the maternal brain to global hypoperfusion. Cerebral perfusion pressure equals mean

arterial pressure minus intracranial pressure ($CPP = MAP - ICP$). Finally, volatile anesthetics and opioids cross the placenta and threaten neonatal respiratory depression. The drug regimen must be calibrated with care.

Detailed empirical evaluations remain exceedingly scarce in the literature. Few reports combine continuous invasive hemodynamic tracking, serial physiological delta calculations and postpartum neuroprotective management. Earlier accounts have mostly addressed definitive surgical resection during pregnancy or postpartum ventriculoperitoneal shunt placement. They say little about the clinical trajectory when the patient refuses definitive neurosurgical intervention. An earlier report by the same authors described the anesthetic management of this patient.¹¹ This case report describes the maternal-fetal neuro-hemodynamic course, the systemic physiological adaptations and the multidisciplinary management of a near-term parturient with a massive vestibular schwannoma, obstructive hydrocephalus and complete visual loss from papilledema who underwent emergency cesarean section under tailored neuroprotective general anesthesia. It documents beat-to-beat arterial pressure stability, tight control of ventilatory carbon dioxide, controlled oxytocin administration and conservative medical reduction of intracranial pressure after surgery was refused, so that clinicians who face a similar emergency can examine the approach in detail.

2. Methods

2.1. Report design and reporting guideline

This is a single case report. It presents high-density longitudinal hemodynamic data and serial percentage changes from baseline for one patient, and it is reported in accordance with the Consensus-based Clinical Case Reporting (CARE) guideline.¹²

2.2. Setting

The case was managed in the Emergency Obstetric Surgical Suite and the Intensive Care Unit of a tertiary academic medical center affiliated with the Faculty of Medicine, Universitas Brawijaya, Malang, East Java, Indonesia. The patient was followed for one year after delivery.

2.3. Patient information and case selection

The patient was a 35-year-old Gravida 3 Para 1 Abortus 1 woman at 36 weeks plus 5 days of gestation (height 155 cm, weight 68 kg, body mass index 28.3 kg/m^2). She presented with premature rupture of membranes of 15 hours duration. Acute fetal compromise was present. So was complete bilateral blindness secondary to chronic papilledema. Her American Society of Anesthesiologists Physical Status was Class 3E.¹⁴ She had no preeclampsia, uncorrected coagulopathy or known maternal cardiovascular malformation, and she gave informed consent.¹⁵

The case is reported because severe obstructive hydrocephalus from a posterior fossa tumor is rare in a near-term parturient, because an acute obstetric indication coexisted with an absolute contraindication to neuraxial anesthesia, and because the patient and her family declined neurosurgery, which allowed the conservative course to be documented.¹

2.4. Case timeline

The chronology from the first symptoms to the one-year follow-up is set out in Table 1, using relative time points.

2.5. Clinical findings and diagnostic assessment

Visual function, cranial nerve function, vital signs, laboratory values and cardiocography were assessed at admission. Papilledema was graded on the Frisén scale and facial nerve function on the House-Brackmann scale. Non-contrast brain computed tomography was performed on postoperative day one. Neurological status (Glasgow Coma Scale, papilledema grade) and laboratory values were repeated on postoperative days 1, 3 and 5 and at one year.

2.6. Therapeutic intervention

The anesthetic plan, the drugs and doses, and the reason for each choice are summarized in Table 2 and described below. The drugs and monitoring were selected to prevent surges in intracranial pressure and to protect cerebral perfusion and the fetus.

The radial arterial catheter transducer was zeroed to atmospheric pressure and leveled at the external auditory meatus (tragus) rather than the standard mid-axillary phlebostatic axis, to keep hemodynamic and neuro-perfusion monitoring as precise as possible.¹⁶ The foramen of Monro is approximated externally by the external auditory meatus. In patients with elevated intracranial pressure, aligning the pressure transducer there gives an accurate reading of the mean arterial pressure entering the intracranial vault and removes the hydrostatic column discrepancy caused by head elevation. Beat-to-beat pressure waveforms were analyzed continuously for arterial pulse pressure variation and estimates of systemic

vascular resistance. This allowed real-time detection of vasoplegia or sympathetic hyperreactivity.

The right internal jugular vein was catheterized under real-time ultrasound guidance, which prevents inadvertent carotid puncture and hematoma formation. Either would impede cerebral venous outflow. Continuous central venous pressure monitoring served as a hemodynamic guardrail to keep the mother strictly euvolemic. In the third trimester, aggressive crystalloid loading risks worsening peritumoral brain edema and pulmonary hydrostatic transudation. Hypovolemia, by contrast, triggers maternal hypotension and uteroplacental hypoperfusion. The target central venous pressure was 6 to 8 mmHg. It was maintained throughout the perioperative course and supplemented by dynamic stroke volume evaluations.

The neuroprotective general anesthetic was built around complementary pharmacodynamic properties. Propofol uncouples cerebral metabolism from blood flow in a dose-dependent manner. It suppresses the cerebral metabolic rate of oxygen and promotes precapillary vasoconstriction, which lowers cerebral blood volume and intracranial pressure without impairing cerebral autoregulation at therapeutic concentrations. Atracurium besylate was chosen for its organ-independent degradation. Ester hydrolysis and non-enzymatic Hofmann elimination break it down spontaneously, and this proceeds reliably despite gestational changes in maternal hepatic enzyme induction or renal clearance. Remifentanyl is an ultra-short-acting opioid. Its context-sensitive half-time is three to four minutes. It provided dense analgesia and sympathoadrenal ablation during laryngoscopy and peritoneal incision. Non-specific blood and tissue esterases hydrolyze it rapidly, which limits residual respiratory depression in the mother and the delivered neonate.¹⁷

Table 2. Neuroanesthetic dilemmas, drug interventions and rationale during emergency cesarean section.

Clinical Challenge	Deployed Anesthetic Intervention	Mechanistic Physiological Rationale & Safety Profile
Severe Intracranial Hypertension & Herniation Risk	Mandatory selection of General Anesthesia; strict avoidance of neuraxial techniques (spinal / epidural / CSE)	Dural puncture creates a low-pressure lumbar CSF sink, generating a steep trans-tentorial / trans-foraminal pressure gradient. In a patient with an obstructing posterior fossa mass and exhausted compliance, this can precipitate catastrophic downward cerebellar tonsillar herniation (coning), irreversible brainstem ischemia, and fatal arrest.
Hypertensive Surge & Intubation Sympathoadrenal Response	Slow IV Propofol 100 mg (≈ 1.5 mg/kg) titrated over 60 s combined with Atracurium 25 mg (≈ 0.37 mg/kg) and continuous Remifentanyl infusion (0.1 mcg/kg/min); gentle direct laryngoscopy (Cormack-Lehane 1)	Blunts the potent sympathoadrenal surge of airway manipulation. Propofol decreases cerebral metabolic rate of oxygen (CMRO ₂ by $\approx 40\%$) and couples it with cerebral vasoconstriction, reducing cerebral blood flow (CBF) and ICP. Remifentanyl potently ablates hemodynamic surges without maternal histamine release.
Maintenance of Cerebral Perfusion Pressure (CPP)	Invasive beat-to-beat arterial line monitoring (left radial artery); target Mean Arterial Pressure (MAP) 75–90 mmHg (epochs above target are given in Table 4)	Cerebral perfusion pressure is governed by $CPP = MAP - ICP$. In the setting of severe occult intracranial hypertension (≈ 25 – 35 mmHg), allowing MAP to drop below 70 mmHg risks ischemic brainstem injury, whereas hypertensive spikes exacerbate peritumoral vasogenic edema and upward herniation.
Cerebral Vasomotor Tone & Carbon Dioxide Homeostasis	Controlled mechanical ventilation targeting strict normocapnia: End-Tidal Carbon Dioxide (EtCO ₂) 35–38 mmHg	PaCO ₂ exerts a steep linear effect on cerebral arteriolar caliber. Hypercapnia triggers profound cerebral vasodilation and intracranial volume expansion, triggering acute herniation. Conversely, aggressive hyperventilation (EtCO ₂ < 30 mmHg) causes extreme vasoconstriction and secondary cerebral ischemia. Normocapnia ensures safe equilibrium.
Uterine Atony vs. Hemodynamic & CPP Collapse	Controlled oxytocin infusion (20 IU in 500 mL Ringer's Lactate at 10–15 mU/min); absolute omission of rapid IV bolus	Rapid oxytocin boluses trigger profound systemic vasodilation, peripheral pooling, reflex tachycardia, and abrupt precipitous hypotension, which would immediately crash MAP and CPP. Slow controlled infusion achieves sustained myometrial tone without hemodynamic instability.
Fetal Well-Being & Neonatal Depressant Minimization	Low-dose Sevoflurane (0.8–1.0 MAC) supplemented by ultra-short-acting Remifentanyl infusion; prompt delivery within 12 min of induction	Sevoflurane below 1.0 MAC preserves cerebral autoregulation while ensuring maternal surgical amnesia. Remifentanyl undergoes rapid non-specific tissue and plasma esterase hydrolysis in both maternal and fetal circulations, limiting neonatal respiratory depression (Apgar 8 at 1 min, 9 at 5 min).
Neuromuscular Blockade Predictability & Safety	Atracurium Besylate 25 mg (≈ 0.37 mg/kg) for tracheal intubation and maintenance	Undergoes organ-independent spontaneous chemical degradation (Hofmann elimination, 80%) and ester hydrolysis (20%), ensuring predictable clearance independent of hepatic or renal fluctuations and preventing prolonged post-delivery weakness.

Clinical Challenge	Deployed Anesthetic Intervention	Mechanistic Physiological Rationale & Safety Profile
Emergency Strain, Coughing & Extubation Management	Cessation of Remifentanyl and Sevoflurane at skin closure; 100% O ₂ ; extubation strictly upon full consciousness and obedience to commands	Coughing, straining, or bucking against the endotracheal tube dramatically elevates intrathoracic pressure, blocks cerebral venous outflow, and precipitates an acute post-surgical ICP surge. Remifentanyl allows smooth emergence and immediate postoperative neurological assessment.

Notes: CBF, cerebral blood flow; CMRO₂, cerebral metabolic rate of oxygen; CPP, cerebral perfusion pressure; CSE, combined spinal-epidural; EtCO₂, end-tidal carbon dioxide; ICP, intracranial pressure; MAC, minimum alveolar concentration; MAP, mean arterial pressure; PaCO₂, arterial partial pressure of carbon dioxide; RL, Ringer's lactate.

Table 2 sets out the main neuroanesthetic dilemmas of the emergency surgical delivery and the tailored drug interventions used to limit maternal intracranial hypertension, prevent cerebral herniation and preserve fetal well-being. The decision to avoid neuraxial techniques rested on preventing a lethal trans-foraminal pressure sink. The induction regimen was formulated to decouple cerebral metabolism from cerebral blood flow and so lower intracranial pressure during tracheal manipulation.

2.7. Outcome measures and follow-up

Hemodynamic and laboratory values were summarized descriptively and graphically¹⁸ as the percentage change from the baseline value (T_b, taken at T-5 min before induction): $\Delta\% = [(T_n - T_b) / T_b] \times 100\%$. Because this is a single case (n = 1), no inferential test was applied.¹⁹ Cerebral perfusion pressure was estimated as mean arterial pressure minus an assumed intracranial pressure. Intracranial pressure was not measured. Baseline intracranial pressure was assumed to be 32 mmHg from the clinical papilledema and ventricular distension, and values of 22 to 28 mmHg were assumed intraoperatively under neuroprotective anesthesia.²⁰ The resulting perfusion pressures are therefore illustrative estimates, not measurements. Neurological outcome at one year was graded with the Glasgow Outcome Scale (GOS) and functional disability with the modified Rankin Scale (mRS); the infant was assessed by Apgar score and developmental milestones.

2.8. Ethics and consent

The Central Medical and Health Ethics Committee, Indonesia, oversaw the ethical governance of this report. It granted institutional review board approval under protocol approval registration number 2025/145. All clinical procedures, diagnostic evaluations and therapeutic interventions complied with the ethical tenets of the Declaration of Helsinki of 1975, as revised in 2013, and with the guidelines of the International Committee of Medical Journal Editors.^{21,22} After comprehensive counseling, the patient and her legal surrogate gave

written informed consent to publication of anonymized clinical data, diagnostic neuroimaging, hemodynamic recordings and laboratory trajectories. Patient privacy was protected through complete de-identification, in line with international editorial standards.²³

3. Results

3.1. Presentation and baseline findings

The patient came to the emergency obstetric unit with a devastating, progressive neurological decline that had developed throughout the pregnancy. Bilateral visual blurring began insidiously in the first trimester. It worsened acutely in the seventh month and ended in complete bilateral visual loss with light perception only (amaurosis). Cranial nerve examination showed bilateral optic nerve dysfunction with severe Frisén Grade 5 papilledema, and left facial nerve weakness with a positive Bell's sign (House-Brackmann Grade III). The facial weakness corresponded to a previously misdiagnosed episode of Bell's palsy three years earlier. On admission, systemic blood pressure was 124/86 mmHg (mean arterial pressure 98.7 mmHg), heart rate 90 beats/min and regular, respiratory rate 18 breaths/min, and resting oxygen saturation 88% on ambient air. This baseline arterial hypoxemia mandated immediate pre-oxygenation. Laboratory analysis showed mild gestational dilutional anemia (hemoglobin 10.3 g/dL, hematocrit 31.2%), physiological leukocytosis (11,200/μL) and preserved platelets (228,000/μL). Serum biochemistry showed hypo-osmolar hyponatremia with sodium 129 mmol/L. Potassium was normal (3.8 mmol/L). Chloride was 96 mmol/L, blood urea nitrogen 10.0 mg/dL and serum creatinine 0.62 mg/dL. Obstetric examination confirmed ruptured membranes with clear fluid leakage for 15 hours and regular uterine contractions. Cardiotocography showed persistent variable decelerations and minimal fetal heart rate variability (baseline 130 to 135 beats/min). This established acute fetal compromise and mandated emergency cesarean section, as detailed in Table 3. The timeline of events is given in Table 1.

Table 1. Clinical timeline of the case: events, investigations and interventions from the first symptoms to the one-year follow-up (relative time points).

Time point	Clinical event	Investigation and result	Intervention
About 3 years before delivery	Left facial weakness, diagnosed at the time as Bell's palsy	—	—
First trimester	Bilateral visual blurring begins	—	—
Seventh month of gestation	Acute worsening to complete bilateral visual loss (light perception only)	—	—
Day 0, admission (36 weeks + 5 days)	Premature rupture of membranes for 15 h, regular contractions, fetal compromise	BP 124/86 mmHg (MAP 98.7), HR 90/min, RR 18/min, SpO ₂ 88% on room air; Hb 10.3 g/dL; Na 129 mmol/L; Frisén Grade 5 papilledema; House-Brackmann III; cardiotocography with variable decelerations and minimal variability	Emergency cesarean section under general anesthesia planned
Day 0, T-5 to T0 min	Positioning, monitoring, pre-oxygenation, induction	SpO ₂ 88% → 99%; MAP 98.7 → 88.3 mmHg	15° left tilt and 15° head-up tilt; radial arterial line; internal jugular central venous catheter; 100% O ₂ ; propofol 100 mg, atracurium 25 mg, remifentanyl 0.1 mcg/kg/min

Time point	Clinical event	Investigation and result	Intervention
Day 0, T+5 min	Tracheal intubation	MAP 102.0 mmHg; SBP 130 mmHg; HR 88/min	Gentle laryngoscopy (Cormack-Lehane 1); 7.0 mm cuffed tube
Day 0, T+12 min	Delivery of a male infant	2,600 g; Apgar 8 and 9; MAP 87.3 mmHg	Sevoflurane 0.8 MAC; fentanyl 150 mcg after cord clamping
Day 0, T+15 min	Uterotonic started	MAP 80.3 mmHg (lowest)	Oxytocin 20 IU in 500 mL Ringer's lactate, infusion, no bolus
Day 0, T+48 to T+55 min	Skin closure and awake extubation	MAP 92.0 → 102.7 mmHg; EtCO ₂ 38 mmHg; no coughing	Sevoflurane and remifentanyl stopped at skin closure; awake extubation at T+55
Day 0, T+60 min	Transfer to intensive care	GCS 15; MAP 94.0 mmHg	Arterial line monitoring continued
Postoperative day 1	CT: Koos Grade IV schwannoma, hydrocephalus, upward herniation; shunt advised and declined	Na 133 mmol/L; Frisén Grade 4	Dexamethasone 4 mg IV every 6 h; acetazolamide 250 mg orally every 6 h
Postoperative day 3	Ward care	Na 136 mmol/L; Frisén Grade 3	Dexamethasone 4 mg IV every 8 h; acetazolamide 250 mg every 8 h
Postoperative day 5	Discharge home	Na 138 mmol/L; Frisén Grade 2	Oral dexamethasone taper (4 mg/day); acetazolamide 250 mg every 12 h
One year	Follow-up: GOS 5; mRS 2	Permanent amaurosis with optic atrophy; House-Brackmann II; GCS 15/15; infant with normal milestones	Dexamethasone and acetazolamide discontinued

Notes: BP, blood pressure; CL, Cormack-Lehane; EtCO₂, end-tidal carbon dioxide; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; Hb, hemoglobin; HR, heart rate; IV, intravenous; MAC, minimum alveolar concentration; MAP, mean arterial pressure; mRS, modified Rankin Scale; RR, respiratory rate; SBP, systolic blood pressure; SpO₂, peripheral oxygen saturation; T, minutes from induction (T0).

Table 3. Demographic profile, obstetric characteristics and baseline multi-system clinical findings at emergency admission.

Domain / Parameter	Clinical Finding / Diagnostic Metric	Diagnostic Ascertainment & Pathophysiological Significance
Maternal Demographics		
Chronological Age	35 years	Advanced maternal reproductive age; peak incidence window for vestibular schwannoma
Parity & Obstetric Status	Gravida 3, Para 1, Abortus 1 (G3P1001Ab100)	Previous uneventful term delivery (living child) and one first-trimester miscarriage
Gestational Age	36 weeks + 5 days	Near-term gestation; peak physiological hyperdynamic blood volume expansion (40–50%)
Anthropometry & Body Mass Index	Height 155 cm; Weight 68 kg; BMI 28.3 kg/m ²	Overweight classification; increased intra-abdominal pressure exacerbating venous return resistance
ASA Physical Status Classification	ASA Class 3E (Emergency)	Severe systemic disease posing a constant threat to life requiring an emergency surgical procedure
Chief Complaint & Obstetric Urgency		
Presenting Obstetric Event	Uterine contractions & amniotic fluid leakage for 15 h	Premature Rupture of Membranes (PPROM) with prolonged latency interval
Fetal Status on Cardiotocography	FHR 130–135 bpm; variable decelerations & minimal variability	Confirmed non-reassuring fetal status / fetal compromise mandating emergency Cesarean delivery
Estimated Fetal Weight	2,600 g (ultrasonographic biometry)	Appropriate for gestational age; viability confirmed with urgent delivery readiness
Neurological & Neuro-Ophthalmologic Status		
Visual Acuity & Timeline	Complete bilateral blindness (perception of light only)	Progressive decline from 1st trimester, fulminant acceleration at 7th month from chronic severe papilledema
Cranial Nerve Examination	CN II: Bilateral amaurosis; CN VII: Left peripheral paresis	Positive Bell's sign on left (misdiagnosed Bell's palsy 3 years prior representing early CPA tumor involvement)
Level of Consciousness & GCS	Alert, Glasgow Coma Scale 15/15 (E4V5M6)	Preserved cortical consciousness despite exhausted intracranial compliance (decompensated Monro-Kellie curve)
Airway & Intubation Predictors	Mallampati Class 2; TMD 6.0 cm; full cervical mobility	Manageable anatomic airway; gentle laryngoscopy indicated to prevent sympathoadrenal surge
Vital Signs at Admission		
Blood Pressure (BP)	124/86 mmHg (Mean Arterial Pressure 98.7 mmHg)	Preserved baseline resting systemic arterial pressure; sympathetic tone activated
Heart Rate (HR)	90 beats/min, regular sinus rhythm	Heart rate within the normal range for late pregnancy
Respiratory Rate (RR) & Oxygenation	18 breaths/min; SpO ₂ 88% on ambient air	Resting arterial hypoxemia on room air indicating need for immediate pre-oxygenation and airway control
Baseline Laboratory Investigations		
Hematology (Hb, Hct, Leukocytes)	Hb 10.3 g/dL; Hct 31.2%; WBC 11,200/μL; Platelets 228,000/μL	Mild gestational dilutional anemia; physiological leukocytosis; preserved platelet count
Electrolytes & Metabolic Profile	Serum Na ⁺ 129 mmol/L; K ⁺ 3.8 mmol/L; Blood Glucose 98 mg/dL	Hyponatremia, attributed by the authors to neuroendocrine dysregulation / SIADH-like stress response
Renal, Hepatic & Coagulation Panels	BUN 10 mg/dL; Cr 0.62 mg/dL; PT 11.4 s; aPTT 28.6 s; INR 1.01	Preserved renal excretion, hepatic synthesis, and normal plasma hemostasis
Diagnostic Neuroimaging (Post-Cesarean CT)		
Tumor Topography & Morphology	Solid, extra-axial mass in left cerebellopontine angle (CPA)	Consistent with large vestibular schwannoma (Koos Grade IV, ≈4.2 × 3.8 × 3.5 cm)

Domain / Parameter	Clinical Finding / Diagnostic Metric	Diagnostic Ascertainment & Pathophysiological Significance
Mass Effect & Ventriculomegaly	Moderate non-communicating obstructive hydrocephalus	Direct compression and effacement of fourth ventricle obstructing normal CSF outflow
Herniation Biomechanics	Significant peritumoral vasogenic edema; upward transtentorial herniation	Severe infratentorial compartment hypertension vector displacing cerebellar structures upward

Notes: Data extracted from maternal clinical records. aPTT, activated partial thromboplastin time; ASA, American Society of Anesthesiologists; BMI, body mass index; BUN, blood urea nitrogen; CN, cranial nerve; CPA, cerebellopontine angle; CSF, cerebrospinal fluid; FHR, fetal heart rate; GCS, Glasgow Coma Scale; Hb, hemoglobin; Hct, hematocrit; INR, international normalized ratio; MAP, mean arterial pressure; PPRM, preterm premature rupture of membranes; PT, prothrombin time; SIADH, syndrome of inappropriate antidiuretic hormone secretion; SpO₂, peripheral oxygen saturation; TMD, thyromental distance; WBC, white blood cells.

3.2. Diagnostic findings

Non-contrast brain computed tomography on postoperative day one showed a large, well-circumscribed, solid extra-axial mass in the left cerebellopontine angle. It measured 4.2 × 3.8 × 3.5 cm and matched a Koos Grade IV vestibular schwannoma. The lesion severely displaced and compressed the pons and medulla and completely effaced the fourth ventricle. There was marked non-communicating obstructive hydrocephalus of the third and lateral ventricles, extensive peritumoral vasogenic edema and clear upward transtentorial herniation.

3.3. Treatment course and outcome

In the operating theater the patient was positioned with a fifteen-degree left lateral pelvic tilt to relieve aortocaval compression and a fifteen-degree head-up tilt to aid cerebral venous drainage through the internal jugular

veins. Standard monitors were attached. Invasive arterial cannulation and central venous catheterization were established uneventfully. After five minutes of denitrogenation with one hundred percent oxygen, general anesthesia was induced at minute zero (T0). Propofol 100 mg (~1.5 mg/kg) and atracurium 25 mg (~0.37 mg/kg) were titrated slowly under a continuous remifentanyl infusion, which blunted the intubation reflex. Gentle direct laryngoscopy at minute five (T+5) gave a Cormack-Lehane Grade 1 view, and a 7.0 mm cuffed tube was placed promptly. Systolic blood pressure went from a baseline of 124 mmHg to 115 mmHg at induction (delta -7.3%) and 130 mmHg at intubation (delta +4.8%). Mean arterial pressure went from 98.7 mmHg to 88.3 mmHg at induction (delta -10.5%) and 102.0 mmHg at intubation (delta +3.3%). Heart rate stayed between 82 and 88 beats/min. The hypertensive surges and tachycardia typical of conventional rapid-sequence intubation in obstetrics were completely prevented, as illustrated in Figure 1.

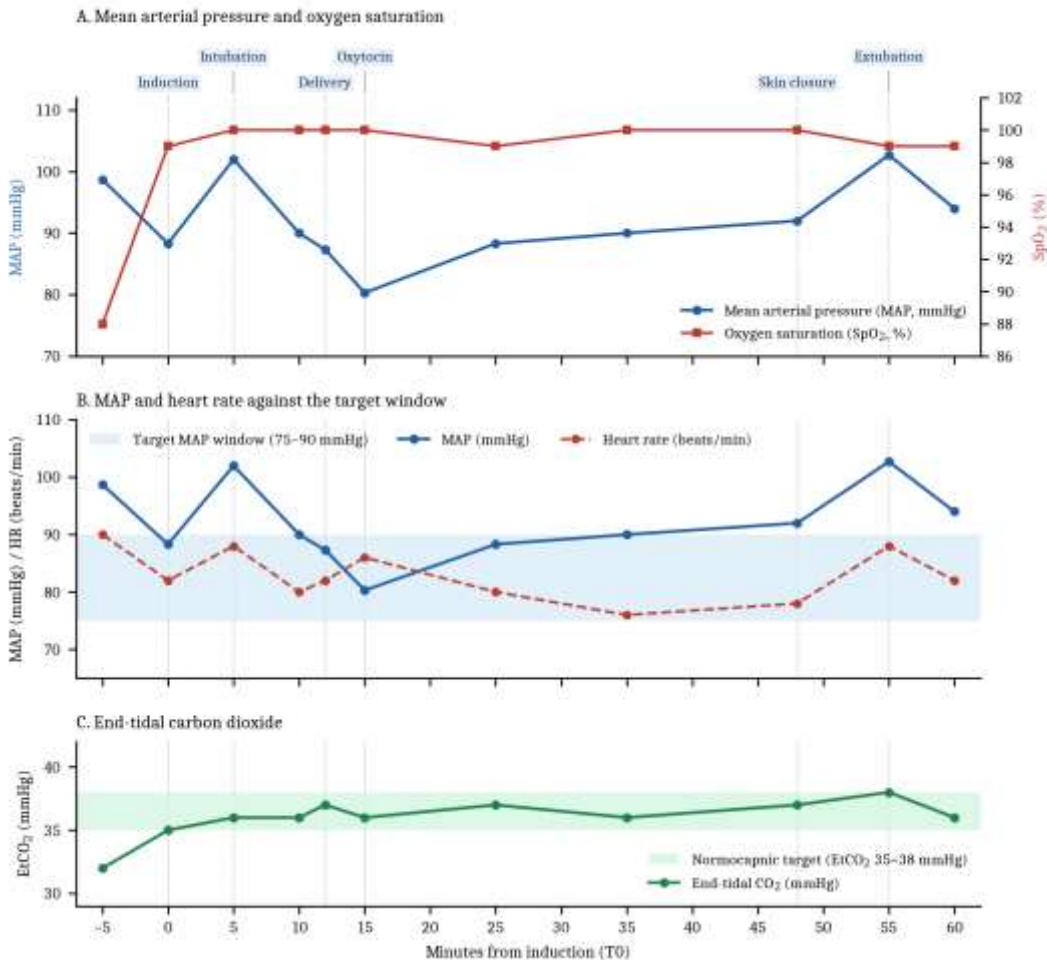


Figure 1. Hemodynamic, oxygenation and ventilatory profile across the surgical milestones. Panels: A, mean arterial pressure and oxygen saturation; B, mean arterial pressure and heart rate against the 75–90 mmHg target window; C, end-tidal carbon dioxide. Values are those of Table 4.

Epoch-by-epoch evaluation showed stable physiology throughout the surgical intervention. At baseline before induction (T-5 min), the patient had resting arterial oxygen

desaturation (SpO₂ 88% on ambient air). Five minutes of denitrogenation with one hundred percent oxygen through an anatomical face mask completely corrected this to 99%.

After the propofol-atracurium-remifentanyl induction (T0), mean arterial pressure fell modestly and in a controlled way, by -10.5% (from 98.7 mmHg to 88.3 mmHg). This reflected mild peripheral vasodilation. Fluid maintenance counterbalanced it easily, with no vasopressor bolus.

During direct laryngoscopy and endotracheal intubation (T+5 min), the pre-emptive continuous remifentanyl infusion blunted the customary hypertensive spike. Systolic blood pressure rose by only +4.8% (to 130 mmHg). Heart rate fell slightly from 90 to 88 beats/min (-2.2%), confirming suppression of the sympathetic axis. The uterine incision and delivery of the infant at minute twelve (T+12 min) proceeded without maternal awareness or surgical movement, with deep surgical anesthesia maintained by low-dose sevoflurane (0.8 MAC) and remifentanyl. After the umbilical cord was clamped, fentanyl 150 mcg was given promptly. It sustained stable analgesia while remifentanyl continued.

Surgical delivery proceeded rapidly through a Pfannenstiel incision. At minute twelve (T+12), a vigorous male infant weighing 2,600 grams was delivered, with Apgar scores of 8 at one minute and 9 at five minutes. After umbilical cord clamping, intravenous fentanyl 150 mcg was given. A controlled oxytocin infusion (20 international units in 500 mL Ringer's lactate) was started at minute fifteen (T+15). It established excellent uterine tone while

mean arterial pressure was held at 80.3 mmHg (delta -18.6% from baseline), and it entirely avoided the precipitous vasoplegic hypotension typical of rapid oxytocin boluses. Illustrative cerebral perfusion pressure, calculated from the assumed intracranial pressure, did not fall below 58.3 mmHg and averaged 67.5 mmHg across the eleven epochs. These values stayed above the ischemic threshold of 50 mmHg.

Mechanical ventilation maintained strict normocapnia. After induction, end-tidal carbon dioxide stayed between 35 and 38 mmHg (32 mmHg before induction), which avoided hypercapnia-induced intracranial vasodilation and hypocapnia-induced cerebral ischemia. Surgical blood loss was approximately 350 mL, and central venous pressure held steady at 6 to 8 mmHg under balanced crystalloid hydration. At skin closure (minute forty-eight), sevoflurane and remifentanyl were stopped, and spontaneous respiratory drive returned promptly. At minute fifty-five (T+55) the patient was fully conscious and oriented and followed complex commands. Awake tracheal extubation was smooth, without coughing, bucking or straining. Mean arterial pressure was 102.7 mmHg (delta +4.1%). No coughing, bucking or straining was observed after extubation. The complete longitudinal hemodynamic and ventilatory trajectories are given in Table 4.

Table 4. Longitudinal hemodynamic, ventilatory and illustrative cerebral perfusion trajectory across perioperative epochs (T-5 min to T+60 min).

Perioperative Epoch	Time (min)	SBP (mmHg) [Δ%]	DBP (mmHg) [Δ%]	MAP (mmHg) [Δ%]	HR (bpm) [Δ%]	SpO ₂ (%) [Δ%]	EtCO ₂ (mmHg)	CPP* (mmHg)	Key Interventions & Clinical Events
Pre-Induction Baseline	T-5	124 [0.0%]	86 [0.0%]	98.7 [0.0%]	90 [0.0%]	88 [0.0%]	32	66.7	15° left lateral tilt + 15° head-up tilt; 100% O ₂ pre-oxygenation; radial A-line zeroed
Anesthesia Induction	T0	115 [-7.3%]	75 [-12.8%]	88.3 [-10.5%]	82 [-8.9%]	99 [+12.5%]	35	64.3	Slow IV Propofol 100 mg + Atracurium 25 mg + Remifentanyl 0.1 mcg/kg/min initiated
Tracheal Intubation	T+5	130 [+4.8%]	88 [+2.3%]	102.0 [+3.3%]	88 [-2.2%]	100 [+13.6%]	36	74.0	Gentle CL-1 direct laryngoscopy; 7.0 mm ETT secured; no cough or hypertensive surge
Uterine Incision / Pre-Delivery	T+10	118 [-4.8%]	76 [-11.6%]	90.0 [-8.8%]	80 [-11.1%]	100 [+13.6%]	36	65.0	Sevoflurane 0.8 MAC + Remifentanyl; mechanical ventilation normocapnic; surgical entry
Infant Delivery Milestone	T+12	114 [-8.1%]	74 [-14.0%]	87.3 [-11.6%]	82 [-8.9%]	100 [+13.6%]	37	62.3	Male infant delivered (2,600 g, Apgar 8/9); umbilical cord clamped; Fentanyl 150 mcg IV
Uterotonic Initiation	T+15	105 [-15.3%]	68 [-20.9%]	80.3 [-18.6%]	86 [-4.4%]	100 [+13.6%]	36	58.3	Oxytocin 20 IU in 500 mL RL slow infusion started; no bolus given; MAP preserved >75 mmHg
Placental Delivery & Synthesis	T+25	115 [-7.3%]	75 [-12.8%]	88.3 [-10.5%]	80 [-11.1%]	99 [+12.5%]	37	65.3	Firm uterine contraction; minimal blood loss (~350 mL); crystalloid euvolemia maintained
Myometrial & Fascial Suture	T+35	118 [-4.8%]	76 [-11.6%]	90.0 [-8.8%]	76 [-15.6%]	100 [+13.6%]	36	68.0	Stable surgical anesthesia; CVP guided fluid therapy (CVP 6–8 mmHg); normothermic
Subcuticular Skin Closure	T+48	120 [-3.2%]	78 [-9.3%]	92.0 [-6.8%]	78 [-13.3%]	100 [+13.6%]	37	70.0	Surgery concluded; sevoflurane and remifentanyl discontinued; spontaneous ventilation returns
Awake Tracheal Extubation	T+55	132 [+6.5%]	88 [+2.3%]	102.7 [+4.1%]	88 [-2.2%]	99 [+12.5%]	38	76.7	Patient fully awake, following verbal commands; smooth extubation without straining/bucking
Transfer to Intensive Care	T+60	122 [-1.6%]	80 [-7.0%]	94.0 [-4.8%]	82 [-8.9%]	99 [+12.5%]	36	72.0	Admitted to ICU; GCS 15; stable neurological baseline; arterial line monitoring continued

Notes: Δ% = [(Tn - Tb) / Tb] × 100%, where Tb is the value at T-5 min (pre-induction baseline). *Illustrative cerebral perfusion pressure (CPP) = MAP - assumed intracranial pressure (ICP). ICP was not measured; it was assumed to be 32 mmHg at baseline and 22–28 mmHg intraoperatively, and the value used at each epoch is the difference between the MAP and CPP columns. These values are illustrative, not measurements. A-line, arterial line; CL-1, Cormack-Lehane Grade 1; CVP, central venous pressure; DBP, diastolic blood pressure; ETT, endotracheal tube; EtCO₂, end-tidal carbon dioxide; GCS, Glasgow Coma Scale; HR, heart rate; ICU, intensive care unit; MAP, mean arterial pressure; RL, Ringer's lactate; SBP, systolic blood pressure; SpO₂, peripheral oxygen saturation.

Figure 2 plots the arterial pressures, the illustrative cerebral perfusion pressure and the serial percentage

changes at the eleven epochs of Table 4. The target mean arterial pressure of 75 to 90 mmHg was met at six of the

ten post-induction epochs. Mean arterial pressure was above 90 mmHg at the intubation (102.0 mmHg), skin

closure (92.0 mmHg), extubation (102.7 mmHg) and ICU transfer (94.0 mmHg) epochs.

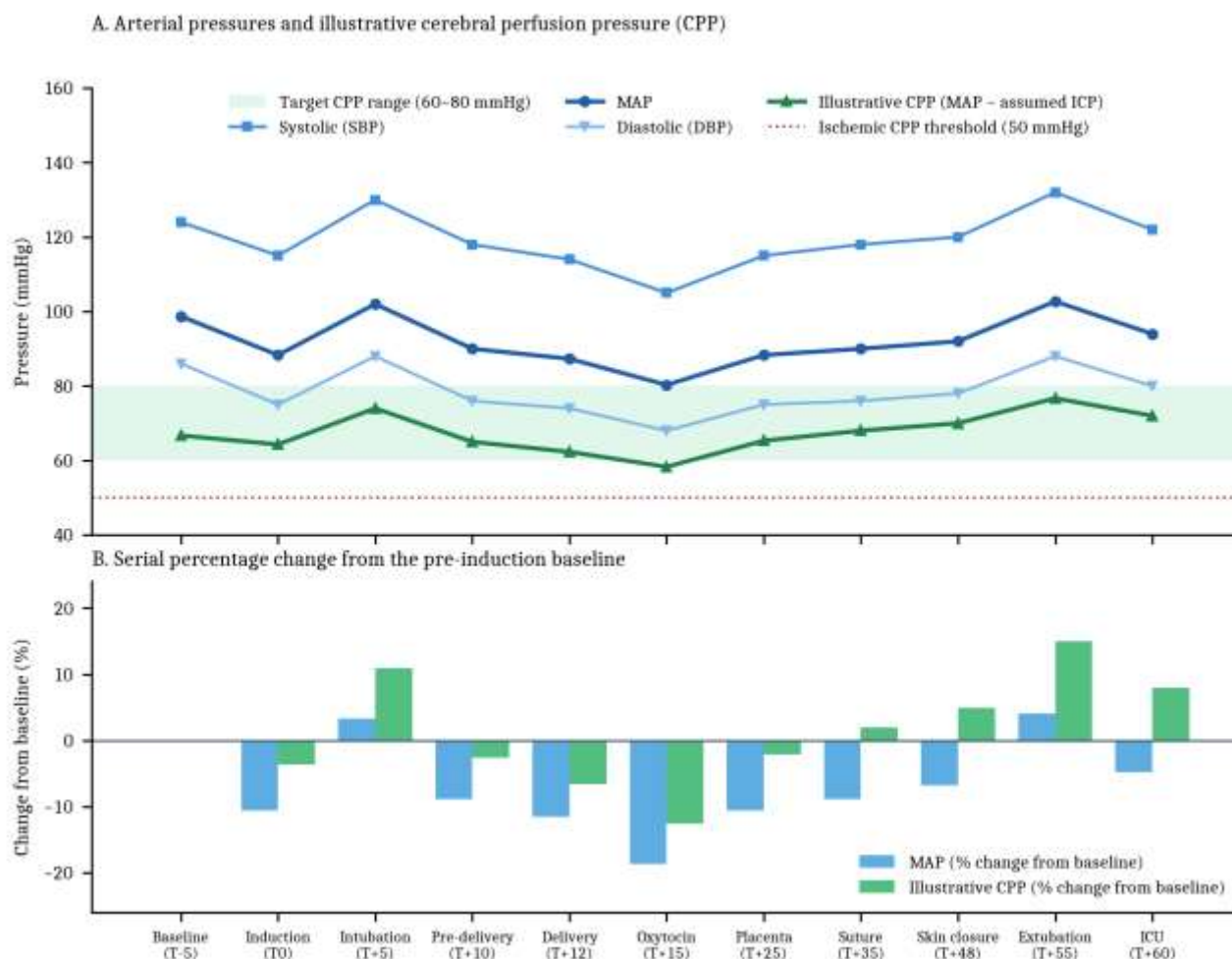


Figure 2. Arterial pressures and illustrative cerebral perfusion pressure (CPP; MAP minus an assumed intracranial pressure) across the perioperative epochs (A) and their percentage change from baseline (B).

The patient was transferred to the intensive care unit after surgery. The neurosurgical team recommended immediate external ventricular drainage or ventriculoperitoneal shunt placement. After extensive multidisciplinary counseling, however, the patient and her family formally exercised their autonomy and declined all neurosurgical procedures. They asked for non-invasive medical management.

An intensive conservative medical neuroprotective protocol was therefore started. Intravenous dexamethasone 16 mg daily (4 mg every six hours) targeted the vasogenic peritumoral edema. Oral acetazolamide 1,000 mg daily (250 mg every six hours) suppressed cerebrospinal fluid production by the choroid plexus. Serum sodium rose steadily from 129 mmol/L at admission to 133 mmol/L on postoperative day one (delta

+3.1%), 136 mmol/L on day three (delta +5.4%) and 138 mmol/L at discharge on day five (delta +7.0%). Neurological monitoring showed sustained alertness with a Glasgow Coma Scale score of 15/15, stable left facial paresis and progressive attenuation of papilledema from Frisén Grade 5 to Grade 2. The patient was discharged home on postoperative day five with an oral dexamethasone taper and acetazolamide. Table 5 gives the longitudinal laboratory and functional recovery metrics. At one year the visual loss was permanent, with optic nerve atrophy; the left facial paresis had improved to House-Brackmann Grade II; the Glasgow Coma Scale score was 15/15; and the infant showed normal developmental milestones. Table 6 gives the American Society of Anesthesiologists class and the one-year functional outcome (Glasgow Outcome Scale 5; modified Rankin Scale 2, attributed to bilateral visual impairment).

Table 5. Serial hematological, biochemical and neurological findings from emergency presentation through intensive care and one-year follow-up.

Clinical / Laboratory Parameter	Baseline (ED)	Post-Op Day 1 (ICU)	Post-Op Day 3 (Ward)	Post-Op Day 5 (Discharge)	1-Year Follow-Up	Normal Reference Interval
Hematological Parameters						
Hemoglobin (Hb, g/dL) [$\Delta\%$]	10.3 [0.0%]	9.8 [-4.9%]	10.1 [-1.9%]	10.5 [+1.9%]	12.4 [+20.4%]	12.0–15.5 g/dL
Hematocrit (Hct, %) [$\Delta\%$]	31.2 [0.0%]	29.8 [-4.5%]	30.6 [-1.9%]	31.8 [+1.9%]	37.5 [+20.2%]	36.0–46.0 %
White Blood Cells (WBC, / μL)	11,200 [0.0%]	12,800 [+14.3%]	9,400 [-16.1%]	7,800 [-30.4%]	6,500 [-42.0%]	4,500–11,000 / μL
Platelet Count ($\times 10^3$ / μL)	228 [0.0%]	215 [-5.7%]	242 [+6.1%]	260 [+14.0%]	255 [+11.8%]	150–450 $\times 10^3$ / μL

Clinical / Laboratory Parameter	Baseline (ED)	Post-Op Day 1 (ICU)	Post-Op Day 3 (Ward)	Post-Op Day 5 (Discharge)	1-Year Follow-Up	Normal Reference Interval
Electrolytes & Metabolic Profile						
Serum Sodium (Na ⁺ , mmol/L) [Δ%]	129 [0.0%]	133 [+3.1%]	136 [+5.4%]	138 [+7.0%]	140 [+8.5%]	135–145 mmol/L
Serum Potassium (K ⁺ , mmol/L)	3.8 [0.0%]	4.0 [+5.3%]	4.1 [+7.9%]	4.2 [+10.5%]	4.3 [+13.2%]	3.5–5.0 mmol/L
Serum Chloride (Cl ⁻ , mmol/L)	96 [0.0%]	99 [+3.1%]	102 [+6.3%]	103 [+7.3%]	101 [+5.2%]	96–106 mmol/L
Blood Urea Nitrogen (BUN, mg/dL)	10.0 [0.0%]	12.4 [+24.0%]	11.0 [+10.0%]	10.5 [+5.0%]	12.0 [+20.0%]	7.0–20.0 mg/dL
Serum Creatinine (Cr, mg/dL)	0.62 [0.0%]	0.68 [+9.7%]	0.64 [+3.2%]	0.60 [-3.2%]	0.72 [+16.1%]	0.50–0.90 mg/dL
Random Blood Glucose (mg/dL)	98 [0.0%]	128 [+30.6%]	112 [+14.3%]	104 [+6.1%]	92 [-6.1%]	70–140 mg/dL
Neurological & Functional Metrics						
Glasgow Coma Scale (GCS)	15/15 (E4V5M6)	15/15 (E4V5M6)	15/15 (E4V5M6)	15/15 (E4V5M6)	15/15 (E4V5M6)	15/15
Visual Acuity (Bilateral)	Light perception only	Light perception only	Light perception only	Light perception only	Permanent amaurosis	20/20 Snellen
Facial Nerve Paresis (CN VII)	House-Brackmann III	House-Brackmann III	House-Brackmann III	House-Brackmann III	House-Brackmann II	House-Brackmann I
Vestibulocochlear Function (CN VIII)	Severe sensorineural loss	Severe sensorineural loss	Severe sensorineural loss	Severe sensorineural loss	Profound unilateral loss	Normal audition
Papilledema Grade (Frisén Scale)	Grade 5 (Severe)	Grade 4 (Marked)	Grade 3 (Moderate)	Grade 2 (Mild)	Optic nerve atrophy	Grade 0 (Normal)
Medical ICP Management Regimen						
IV Dexamethasone Regimen	Pre-op 10 mg load	4 mg IV q6h (16 mg/d)	4 mg IV q8h (12 mg/d)	Oral taper (4 mg/d)	Discontinued	N/A
Oral Acetazolamide Regimen	None	250 mg PO q6h (1000 mg/d)	250 mg PO q8h (750 mg/d)	250 mg PO q12h (500 mg/d)	Discontinued	N/A
Neonatal Neurodevelopmental Trajectory						
Infant Apgar Score / Status	Fetal compromise	Apgar 8/9; birth wt 2600g	Feeding well, rooming-in	Discharged healthy	Normal milestones at 1 yr	Normal development

Notes: Δ% = $[(T_n - T_b) / T_b] \times 100\%$ relative to the baseline emergency department (ED) value. CN, cranial nerve; ED, emergency department; Frisén scale, clinical optic disc edema grading (0 to 5); Hb, hemoglobin; Hct, hematocrit; House-Brackmann, facial nerve grading system (I = normal, VI = total paralysis); ICP, intracranial pressure; ICU, intensive care unit; PO, per os (orally); q6h/q8h/q12h, every 6/8/12 hours; WBC, white blood cells.

Table 6. American Society of Anesthesiologists class and functional outcome at one year.

Assessment framework	Domain / evaluation criterion	Category assigned	Evidence
ASA Physical Status Classification	Preoperative Risk Stratification	Class 3E (Emergency)	Severe systemic disease posing a constant threat to life in the setting of critical intracranial hypertension and threatened maternal-fetal demise
Glasgow Outcome Scale (GOS)	1-Year Neurological Recovery	Category 5 (Good Recovery)	Patient resumed independent daily living and maternal caregiving activities without focal motor paralysis, despite irreversible optic nerve atrophy
Modified Rankin Scale (mRS)	Functional Disability Stratification	Score 2 (Slight Disability)	Able to look after own affairs without assistance; unable to carry out all previous activities due solely to bilateral visual impairment

Notes: ASA, American Society of Anesthesiologists; GOS, Glasgow Outcome Scale; mRS, modified Rankin Scale.

4. Discussion

4.1. Principal lesson of the case

In a near-term parturient with a giant vestibular schwannoma and obstructive hydrocephalus, avoiding neuraxial anesthesia and delivering the infant under general anesthesia was followed by a hemodynamically stable course and a healthy neonate. The anesthetic used slow induction, a remifentanyl infusion, strict normocapnia, a controlled oxytocin infusion and invasive arterial and central venous monitoring. This describes what happened in one patient; it does not show that the approach is better than the alternatives.

Successful management of an emergency cesarean section in a parturient with a giant vestibular schwannoma, obstructive hydrocephalus and complete blindness is a profound clinical demonstration of applied neuro-obstetric physiology.¹ The case also shows how the systemic

adaptations of pregnancy can turn a quiescent, slow-growing posterior fossa tumor into an acute neurological catastrophe. It also shows the indispensable role of a carefully customized anesthetic plan. That plan preserved maternal cerebral perfusion and ensured neonatal survival.²⁴ The sections below examine the underlying pathophysiology, the biomechanical hazards of neuraxial anesthesia, the pharmacological rationale for the general anesthetic regimen, the conservative reduction of intracranial pressure after surgery was refused, and the epidemiological benchmarking against the global literature.

4.2. Comparison with the published literature

Published international series show how rare and therapeutically complex a giant vestibular schwannoma is in advanced gestation. In a systematic review of vestibular schwannomas diagnosed in pregnancy, Al-Mufargi and colleagues² reported that over seventy percent of patients

presented with progressive hearing loss or facial weakness. Fewer than ten percent developed complete gestational blindness or acute transtentorial herniation before delivery. Most earlier parturients had either a second-trimester craniotomy or an elective cesarean section followed at once by neurosurgical resection or ventriculoperitoneal shunt placement. The present case is different. An emergency cesarean section was carried out under neuroprotective general anesthesia, and long-term stabilization was then achieved by conservative medical management alone, after the patient autonomously refused neurosurgery. The neonatal outcome of this case (Apgar scores of 8 and 9) and the absence of hypotension are in line with published standards for general anesthesia at cesarean delivery²⁵ and with guidance on the treatment of cerebral edema.²⁶

4.3. Pathophysiological or mechanistic interpretation

This section separates what was observed (the clinical course, the arterial pressures and the CT findings) from what is inferred (the hormonal mechanism of tumor growth, the intracranial pressure and the effect of medical therapy on it).

Several endocrine and hemodynamic mechanisms act together to accelerate vestibular schwannoma growth in pregnancy.² Vestibular schwannomas originate from the Schwann cell sheath of the vestibulocochlear nerve. In non-pregnant adults they typically grow at one to two millimeters per year.²⁷ Immunohistochemical and molecular studies, however, have confirmed that they express high concentrations of intracellular progesterone receptors and variable estrogen receptors.²⁸ In the third trimester, maternal plasma progesterone concentrations surge up to twenty-fold. This directly stimulates schwannoma cell proliferation, protein translation and tumor hypertrophy.²⁹ Estrogen upregulates vascular endothelial growth factor transcription within the lesion and promotes neovascularization.³⁰ Maternal blood volume expands by forty to fifty percent at the same time. The hyperdynamic circulation that results leaves the tumor greatly engorged with blood.³¹ The microvasculature of

vestibular schwannomas lacks mature endothelial tight junctions, so elevated capillary hydrostatic pressure drives excess plasma into the surrounding brain tissue. A wide zone of peritumoral vasogenic edema results. It is often larger in volume than the solid tumor itself.³² In our patient, this synergistic process turned a chronic lesion into an acute expanding mass. The lesion had shown itself three years earlier as isolated facial palsy. The mass completely obstructed the fourth ventricle and precipitated fulminant non-communicating hydrocephalus.

The molecular mechanisms behind gestational acceleration of schwannoma growth deserve a closer look. Schwannoma cells arise from the neural crest. Neoplastic transformation is characterized by loss of the NF2 tumor suppressor gene, which encodes the protein merlin (schwannomin). Under high circulating concentrations of pregnancy-associated sex steroids, progesterone binds to overexpressed intracellular progesterone receptors and activates the downstream mitogen-activated protein kinase and phosphoinositide 3-kinase signaling pathways, which drive cell cycle progression and cellular hypertrophy. Estrogen, in parallel, upregulates vascular endothelial growth factor (VEGF) and its tyrosine kinase receptors. Neovascular sprouting becomes dysregulated. The newly formed tumor capillaries lack the tight interendothelial junctional complexes (zonula occludens-1, claudin-5 and occludin) that make up the normal blood-brain barrier. Driven by a 40–50% gestational expansion of blood volume, these leaky vessels allow massive fluid extravasation. The peritumoral vasogenic edema grows. Acute intracranial hypertension results.

Figure 3 shows the pathophysiology schematically. The expanding cerebellopontine angle tumor exerts mass effect within the rigid posterior fossa. Effacement of the fourth ventricle leads to non-communicating obstructive hydrocephalus, and intracranial compliance decompensates along the Monro-Kellie volume-pressure relationship. The anatomic displacement of the brainstem and the upward herniation vector through the tentorial notch show how imminent catastrophic downward coning would have been had a lumbar puncture been attempted.

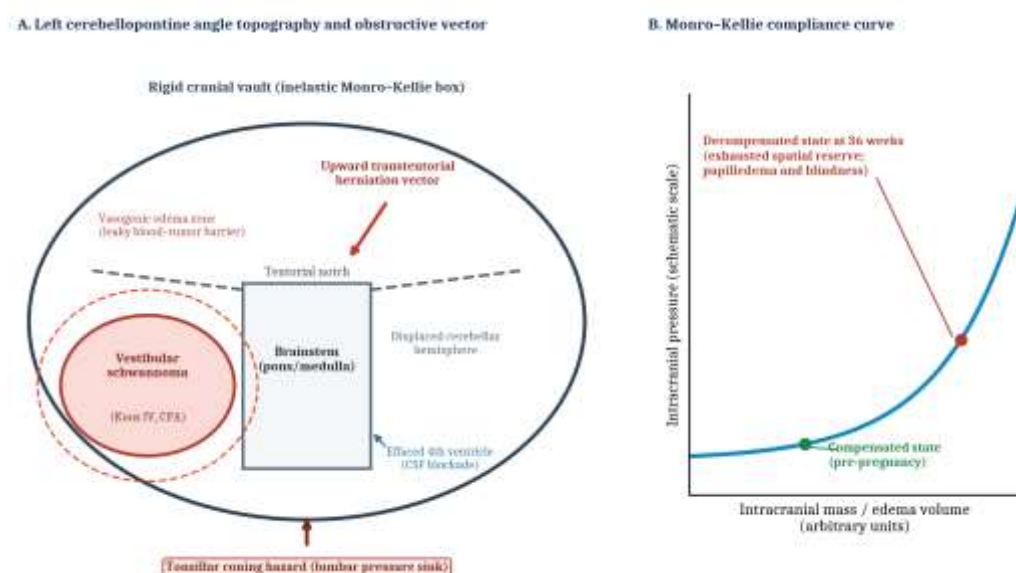


Figure 3. Schematic of the posterior fossa topography, cerebrospinal fluid flow obstruction and Monro-Kellie volume-pressure decompensation. Panel B is a schematic and does not show patient measurements.

Obstructive hydrocephalus that develops within the rigid, unyielding posterior fossa illustrates the principles of the Monro-Kellie doctrine.³³ The intracranial compartment

has a fixed total volume, occupied by brain parenchyma (eighty percent), blood (ten percent) and cerebrospinal fluid (ten percent). Normally an expanding focal mass is

compensated by displacement of cerebrospinal fluid into the spinal subarachnoid space and by compression of the low-pressure cerebral venous sinuses. In our patient, the primary outflow tract for cerebrospinal fluid was completely occluded once the cerebellopontine angle mass had effaced the fourth ventricle. Fluid produced in the lateral and third ventricles could not reach the subarachnoid space. Spatial compensatory reserve was exhausted. The patient moved onto the terminal vertical limb of the intracranial volume-pressure curve, where any minor increase in intracranial blood volume or tissue edema produces an exponential surge in intracranial pressure, as the computed tomography finding of upward transtentorial herniation shows. Elevated cerebrospinal fluid hydrostatic pressure was also transmitted along the optic nerve sheaths. It compressed the retinal ganglion cell axons at the scleral lamina cribrosa and arrested both orthograde and retrograde axoplasmic transport. Prolonged axonal ischemia caused severe chronic papilledema, secondary optic nerve infarction and irreversible bilateral optic atrophy. This explains the patient's complete blindness.

The physiological principles behind pharmacological management of intracranial hypertension in this patient give useful insight into skull base neurobiology under gestational stress. The cranial vault is a rigid, non-distensible compartment of fixed total volume. It holds brain parenchyma, intracranial blood and cerebrospinal fluid. By the Monro-Kellie doctrine, any increase in tumor volume or ventricular CSF volume must be matched by an

equal reciprocal reduction in another compartment, or intracranial pressure rises exponentially along the steep portion of the spatial compensation curve. In our patient, spatial buffering was exhausted. Even minor volume shifts could trigger catastrophic upward or downward herniation vectors. Dexamethasone acts selectively on the disrupted blood-tumor barrier. It upregulates tight junction protein synthesis, inhibits VEGF transcription and decreases capillary endothelial permeability, which directly halts and reverses peritumoral vasogenic edema formation. Acetazolamide works through a separate, non-overlapping cellular pathway. It reversibly inhibits carbonic anhydrase isoenzymes in the choroid plexus epithelium, reducing active transport of bicarbonate and sodium ions into the ventricular lumen and suppressing cerebrospinal fluid production by up to fifty percent. By combining the two drugs, our multidisciplinary team targeted both the parenchymal or edematous compartment and the cerebrospinal fluid compartment of the Monro-Kellie equation. Maternal neurological status remained stable through the perinatal period without invasive surgical shunting. Reversal of the upward herniation vector and restoration of compliance reserves are inferred from this stable course, because intracranial pressure and imaging were not re-measured.

Figure 4 illustrates the multi-tier pathophysiological cascade. It links gestational endocrine priming to obstructive hydrocephalus and irreversible amaurosis through molecular, microvascular, intracranial compliance and neuro-ophthalmological mechanisms.



Figure 4. Multi-tier pathophysiological cascade: gestational endocrine-hemodynamic priming, obstructive hydrocephalus and amaurosis (schematic).

4.4. Implications for practice

From the anesthesiological standpoint, non-communicating hydrocephalus, posterior fossa mass effect and upward transtentorial herniation made neuraxial anesthesia contraindicated.³⁴ Spinal, epidural and combined spinal-epidural techniques are universally recognized as the preferred modalities for cesarean delivery. They avoid maternal airway instrumentation, protect against aspiration and reduce fetal drug exposure. A needle placed in the lumbar subarachnoid space, however, creates a low-resistance fistula through which cerebrospinal fluid escapes into the epidural space. With high intracranial pressure and obstructed ventricular outflow, the lumbar puncture sets up a steep pressure gradient between the posterior fossa and the lumbar theca. This hydrostatic vector inevitably drives the cerebellar tonsils downward through the foramen magnum. The result is acute tonsillar herniation (coning), severe compression of the vital medullary respiratory and vasomotor centers and immediate fatal cardiorespiratory collapse. Even epidural anesthesia carries an unacceptable risk. Accidental dural puncture occurs in approximately one percent of cases and could be fatal in this anatomical setting. General anesthesia was therefore the only viable and ethically justifiable option.

General anesthesia in a patient whose intracranial compliance is exhausted demands an active neuroprotective strategy. It must defend cerebral perfusion pressure and avoid surges in intracranial pressure.³⁵ Cerebral perfusion pressure is mean arterial pressure minus intracranial pressure ($CPP = MAP - ICP$). Direct invasive intracranial pressure monitoring was not available before surgery. A target window of 75 to 90 mmHg for mean arterial pressure was chosen to support cerebral perfusion pressure without inducing hypertensive cerebral edema. For an assumed intracranial pressure of 22 to 28 mmHg, this window corresponds to an estimated cerebral perfusion pressure of 47 to 68 mmHg, so the upper part of the window is needed to keep it above 60 mmHg. Induction is the most hazardous hemodynamic phase. The intense sympathoadrenal reflex triggered by direct laryngoscopy can precipitate hypertensive surges that worsen brain herniation. Slow intravenous propofol combined with a continuous remifentanyl infusion mitigated this danger in this patient. Propofol is a potent neuroprotective agent. It decreases the cerebral metabolic rate of oxygen ($CMRO_2$) by approximately forty percent, which induces coupled cerebral vasoconstriction and reduces cerebral blood flow and intracranial pressure. Remifentanyl is an ultra-short-acting, selective μ -opioid receptor agonist. It potently blunts the hemodynamic response to airway instrumentation without causing maternal histamine release. Non-specific tissue and plasma esterases in both the maternal and fetal circulations metabolize it rapidly, and the infant was delivered without respiratory depression, as the Apgar scores of 8 and 9 at one and five minutes show.³⁶

Careful control of anesthetic depth and ventilation during maintenance prevented secondary brain injury.³⁷

Sevoflurane was kept below 1.0 minimum alveolar concentration. At that dose it preserves cerebral autoregulation and blunts surgical stress, and it avoids the profound cerebral arteriolar vasodilation seen with higher volatile concentrations. Mechanical ventilation was titrated strictly to normocapnia (end-tidal carbon dioxide 35 to 38 mmHg). In neuroanesthesia, arterial carbon dioxide tension and cerebral blood flow are related almost linearly. Hypercapnia induces marked cerebral vasodilation that would have catastrophically increased intracranial pressure. Aggressive hypocapnia (end-tidal carbon dioxide below 30 mmHg) risks severe cerebral arteriolar vasoconstriction and ischemic tissue damage. Strict normocapnia kept cerebral vasomotor tone optimal throughout surgical delivery.

Hemodynamic stewardship around uterotonic administration was an essential part of the intraoperative protocol.³⁸ A rapid intravenous oxytocin bolus is common in routine cesarean delivery. It causes marked peripheral vasodilation, lower systemic vascular resistance, reflex tachycardia and abrupt hypotension. In our patient an oxytocin-induced fall in mean arterial pressure would have collapsed cerebral perfusion pressure at once and caused ischemic brainstem injury. We therefore gave oxytocin as a slow, controlled intravenous infusion (20 international units in 500 mL Ringer's lactate). This achieved excellent myometrial contraction without hemodynamic collapse, with a modest mean arterial pressure nadir of 80.3 mmHg (delta -18.6%). At the end of surgery, the rapid esterase clearance of remifentanyl allowed smooth awake extubation. The patient did not cough, buck or strain. Any of these would have raised intrathoracic pressure sharply, impeded cerebral venous drainage and triggered a dangerous postoperative rise in intracranial pressure.

The debate over volatile maintenance versus total intravenous anesthesia (TIVA) in neuro-obstetric surgery deserves explicit attention in light of our findings.³⁹ High-dose volatile agents (above 1.0 MAC) impair cerebral autoregulation and cause dose-dependent arteriolar vasodilation. This can worsen intracranial hypertension. Low-dose sevoflurane kept below 1.0 MAC, combined with a continuous remifentanyl infusion, preserves cerebral flow-metabolism coupling and gives dependable maternal amnesia. In emergency obstetric settings, starting a target-controlled propofol infusion at once may be limited by equipment availability or by the risk of delayed emergence. In this patient, low-dose volatile maintenance with an ultra-short-acting opioid gave a balance between neuroprotective stability and rapid maternal recovery.

Figure 5 sets out the integrated multidisciplinary management pathway. It is a practical clinical algorithm that runs from the decision between contraindicated neuraxial techniques and indicated neuroprotective general anesthesia, through intraoperative hemodynamic stewardship, to conservative medical neuroprotection. The measures were feasible in a tertiary center with invasive monitoring and neurosurgical support; how far they transfer to settings without such resources is uncertain.

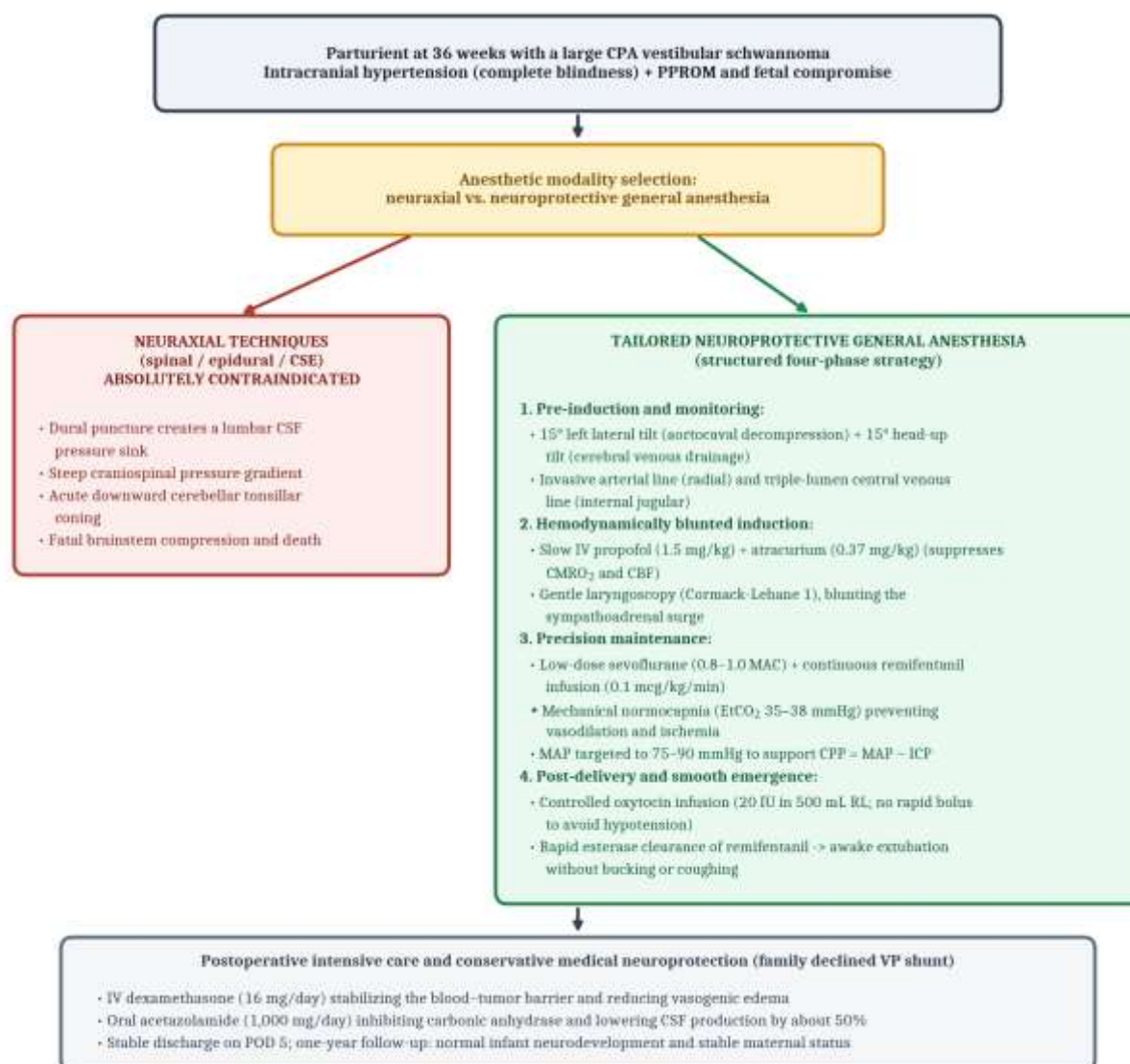


Figure 5. Integrated multidisciplinary anesthetic and neuroprotective algorithm for parturients with intracranial hypertension.

This case also bears on bioethics and clinical decision-making, because it shows the primacy of patient autonomy in life-threatening conditions. The patient and her legal surrogate declined the recommended ventriculoperitoneal shunt surgery after informed counseling. The team did not coerce them paternalistically. It redirected its effort to maximizing conservative medical neuroprotection. The patient was discharged on postoperative day five and remained independent at one-year follow-up. In carefully selected patients under close intensive care surveillance, this confirms that conservative medical management of tumor-induced obstructive hydrocephalus can be an effective and viable alternative when surgical options are not pursued.

The postoperative course brought an intricate bioethical and pharmacological challenge. The patient and her family autonomously declined neurosurgical cerebrospinal fluid diversion (ventriculoperitoneal shunting).³ Respect for patient autonomy is a foundational ethical pillar, even when the medical team considers a proposed operation life-saving. Conservative medical neuroprotection required a targeted pharmacological attack on intracranial volume: dexamethasone acted on the vasogenic edema and acetazolamide on cerebrospinal fluid production, as described in Section 4.3. This two-drug approach was followed by stable neurological function (Glasgow Coma

Scale 15/15) and safe discharge on postoperative day five.⁴⁰

4.5. Strengths

This report has several strengths. They include high-density invasive hemodynamic tracking across eleven standardized surgical epochs, serial percentage change calculations, and complete one-year maternal-infant follow-up.

4.6. Limitations

The principal limitation is the single-patient design, which rules out population-level epidemiological generalization about conservative medical management of giant vestibular schwannomas in pregnancy and cannot establish causation, prevalence or effect. Intracranial pressure was not measured, so the cerebral perfusion pressures are illustrative estimates. No repeat imaging was done after medical therapy, so its effect on the hydrocephalus and the herniation is inferred. Follow-up lasted one year, and the visual loss was permanent. The results may not apply to other patients or to settings without invasive monitoring.

4.7. Recommendations for future work

Point-of-care ultrasound measurement of the transorbital optic nerve sheath diameter (ONSD) offers a promising non-invasive way to follow real-time intracranial pressure changes at the bedside in pregnant patients with skull base neoplasms. It could serve as an adjunct to invasive arterial waveform analytics. Testing whether the approach described here improves outcomes would need a multicenter case series or registry with prespecified measurement of intracranial pressure surrogates and follow-up imaging.

5. Conclusion

This case report shows that a physiology-based general anesthetic regimen, delivered with invasive hemodynamic monitoring, can be used to deliver a healthy infant by emergency cesarean section when severe non-communicating hydrocephalus and posterior fossa mass effect contraindicate neuraxial anesthesia. The regimen used slow propofol-atracurium induction, continuous remifentanyl infusion, low-dose sevoflurane and strict mechanical normocapnia. A slow, controlled oxytocin infusion and smooth awake extubation completed it. Where cerebrospinal fluid diversion is declined, dexamethasone and acetazolamide, with multidisciplinary coordination, were followed by a stable course in this patient. Mother and infant were well at one year. These observations come from one case. They do not establish efficacy.

6. Declarations

Ethical approval and consent to participate. This case report was approved by the Central Medical and Health Committee Ethics Committee, Indonesia, under reference number 2025/145. The report was prepared in accordance with the Declaration of Helsinki (1975, revised 2013). Written informed consent was obtained from the patient and her legal surrogate.

Consent for publication. Written informed consent for publication of this case, including anonymized clinical details, physiological trajectories and diagnostic imaging findings, was obtained from the patient and her legal surrogate. A copy of the written consent is available for review by the Editor-in-Chief on request.

Availability of data and materials. All clinical data, hemodynamic time-series datasets and laboratory values generated or analyzed during this study are contained within this article and its tables and figures.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript. The authors reviewed and edited all output and take full responsibility for the integrity, originality and accuracy of the work.

Author contribution statement. R.A.R. conceptualized the report, performed the intraoperative neuroanesthetic management, gathered the clinical data, drafted the manuscript and designed the figures (Conceptualization; Investigation; Data curation; Visualization; Writing – original draft). I. and B.H.L. supervised the clinical management, provided expert neuroanesthetic and obstetric consultation, and critically revised the manuscript (Supervision; Resources; Writing – review & editing). All authors read and approved the final

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