

CASE REPORT

Dexamethasone-Adjuvanted Scalp Block and Total Intravenous Anesthesia for Endoscopic Transsphenoidal Resection of a Giant Sellar-Suprasellar Rathke Cleft Cyst: An Observational Analytic Case Study

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ABSTRACT

Background. Endoscopic transsphenoidal surgery for sellar-suprasellar lesions needs stable hemodynamics and a dry field. Case descriptions of a dexamethasone-adjuvanted scalp block with total intravenous anesthesia are scarce.

Objective. To describe the anesthetic management and the postoperative sodium and urine course of one patient and to discuss what the case suggests.

Methods. Single-patient case report (CARE guideline). A 45-year-old woman with hypertension had bitemporal visual field loss from a giant sellar-suprasellar Rathke cleft cyst. After induction, an ultrasound-guided bilateral scalp block used 20 mL of 0.375% ropivacaine with 10 mg of preservative-free dexamethasone. Propofol (bispectral index 40–60), fentanyl, and atracurium infusions maintained anesthesia during a 5-hour endoscopic resection, with a mean arterial pressure (MAP) target of 65–80 mmHg.

Results. MAP fell from 122 mmHg at baseline to 76–82 mmHg from induction to the end of resection and was 84 mmHg at emergence, without vasoactive rescue. Estimated blood loss was 60 mL, and the field was reported dry. Extubation was cough-free. Urine output was 2.35 mL/kg/h over the first 14 postoperative hours, and sodium fell to 132 mEq/L on day 1, 9.0% below the first pre-operative value of 145 mEq/L. This was managed with fluid restriction without desmopressin. By 48 hours urine output was 1.1–1.4 mL/kg/h and sodium 138 mEq/L. She went home on day 5 without complication.

Conclusion. Stable hemodynamics, low blood loss, and smooth emergence followed this combined technique, and early diuresis with mild hyponatremia settled with fluid restriction. One case cannot show efficacy, and controlled studies are needed.

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1. Introduction

Space-occupying lesions of the sellar and suprasellar compartments are difficult to manage, both surgically and anesthesiologically.¹ Rathke cleft cysts belong to this group. They are benign, non-neoplastic sellar masses that arise from persistent embryonic remnants of Rathke's pouch, an ectodermal diverticulum of the stomodeum that normally closes after the adenohypophysis forms.¹ Most cysts are small and stay asymptomatic, and postmortem studies are reported to show subclinical microcysts in a sizable share of the population. A subset keeps enlarging because of epithelial mucinous hypersecretion, intracystic proteinaceous accumulation, and secondary inflammatory exudation.^{1,2} Giant variants outgrow the sella. Beyond the bony sella turcica, the superior pole can breach the diaphragma sellae and rise into the suprasellar cistern, where it presses on the decussating fibers of the optic chiasm and lifts the infundibular stalk.³ The clinical result is insidious, progressive bitemporal visual field loss. Persistent headache is common, and some patients also

develop anterior or posterior hypopituitarism.^{3,4} Diagnosis can be harder because the imaging features overlap with those of cystic non-functioning pituitary macroadenomas, papillary craniopharyngiomas, and primary sellar abscesses, which call for different antimicrobial preparation and careful microdissection.^{5,6}

Many centers now use the fully endoscopic endonasal transsphenoidal corridor for symptomatic sellar and suprasellar disease, in place of microscopic sublabial or transnasal routes and open transcranial approaches, and comparative series report that these routes can treat Rathke cleft cysts effectively.^{3,7} The rigid endoscope gives a panoramic, high-definition view of the posterior nasal cavity, sphenoid rostrum, sellar floor, and suprasellar cistern. With it, surgeons can evacuate the cyst as completely as the anatomy allows, take a capsular biopsy, and decompress the visual apparatus. The route is generally considered to cause less sinonasal morbidity and a shorter convalescence, and limited secondary damage to the compressed pituitary parenchyma is expected. A single-center series and a longitudinal quality-of-life cohort report that vision commonly improves after

endoscopic endonasal surgery for these cysts.^{8,9} The route still demands a clear field. The endoscopic lens works in a channel only several millimeters wide, and bleeding impairs visibility during endoscopic nasal surgery.^{10,11} Even small amounts of continuous capillary oozing or venous bleeding from the engorged nasal and cavernous vasculature can foul the optics. The surgeon is then blinded. The rhythm of dissection breaks, and the risk of internal carotid artery laceration or inadvertent hypothalamic avulsion rises.

This setting places demands on neuroanesthetic management that a standard general anesthetic may not meet.^{10,12} The endoscopic corridor is intensely nociceptive. Transsphenoidal surgery is known to cause wide hemodynamic fluctuations from noxious stimulation at several stages.^{12,13} Rigid turbinectomy, posterior septectomy, mucosal elevation, and high-speed drilling of the sphenoid bone generate large somatosensory and sympathetic afferent volleys, carried mainly by the ophthalmic (V1) and maxillary (V2) divisions of the trigeminal nerve and by the sphenopalatine ganglion.¹⁴ Without adequate regional blockade, these insults can provoke sympathetic discharge, with catecholamine surges, hypertensive spikes, and reflex tachycardia.^{12,13} In hypertensive patients such surges may worsen engorgement of the mucosal capillary plexuses and bleeding within the corridor. Sympathetic hyperactivity may also raise cerebral blood flow and the cerebral metabolic rate of oxygen (CMRO₂), and with them intracranial pressure (ICP). Then the soft suprasellar cyst capsule might not descend into the sellar floor by itself. The surgeon would be pushed toward upward micro-curettage.

Regional techniques are used in multimodal neurosurgical care to improve intraoperative hemodynamic stability, lower pain scores, and reduce analgesic requirements.¹⁵ The scalp block was first developed for open awake or asleep craniotomy. Its purpose was to blunt the pressor response to Mayfield head-pinning and scalp incision. It intercepts the sensory branches of the trigeminal nerve (supraorbital, supratrochlear, and auriculotemporal nerves) and of the cervical plexus (greater and lesser occipital nerves). Randomized trials and a meta-analysis report that scalp block attenuates the heart rate and blood pressure response to pin insertion, skin incision, and dural incision.^{16–18} Perineural adjuvants can add to the block. Long-acting glucocorticoids such as preservative-free dexamethasone may bring two benefits at once.^{19,20} Perineural dexamethasone prolongs the analgesia of long-acting local anesthetics.²⁰ Proposed mechanisms are local vasoconstriction, glucocorticoid receptor-mediated suppression of inflammatory mediators, and modulation of potassium channels on unmyelinated nociceptive C-fibers. Gradual systemic absorption may also give antiemetic prophylaxis.²⁰ This matters. Postoperative nausea and vomiting (PONV) after endoscopic skull base surgery carries a risk of bleeding and cerebrospinal fluid (CSF) leakage,²¹ and retching also raises ICP, which may stress a delicate skull base reconstruction. Trials report longer analgesia with perineural dexamethasone, including in scalp block,^{19,20} and preclinical evidence supports the safety of low-dose, preservative-free preparations.²²

Combining a regional block with total intravenous anesthesia (TIVA) based on propofol, fentanyl, and titrated neuromuscular blockade is a logical pairing for this surgery. Volatile halogenated ethers such as sevoflurane or desflurane tend to cause cerebral vasodilation and uncouple cerebral blood flow from metabolic demand.

Propofol is thought to preserve cerebral autoregulation and to lower cerebral metabolism in proportion. That should give cerebral vasoconstriction, a smaller cerebral blood volume, and pharmacological brain relaxation. A network meta-analysis of randomized trials in intracranial surgery found a tendency toward more satisfactory brain relaxation with propofol than with sevoflurane, isoflurane, or desflurane, although the confidence intervals included no difference.²³ Whether such relaxation helps a suprasellar lesion descend into the sphenoid corridor under gravity remains a hypothesis. In endoscopic transsphenoidal surgery, propofol-based TIVA was followed by less emergence agitation than sevoflurane, although time to extubation was longer.²⁴ A smooth extubation without coughing or bucking on the endotracheal tube is also pursued in pituitary surgery because it is associated with steadier hemodynamics at extubation.²⁵ A sudden rise in ICP could disrupt a vascularized nasoseptal flap and induce high-output CSF rhinorrhea. A quiet emergence also allows early neurological assessment.²⁵ After surgery, disruption of the infundibular stalk predisposes patients to neuroendocrine fluctuations. These include central diabetes insipidus (DI), the syndrome of inappropriate antidiuretic hormone (ADH) secretion (SIADH), and delayed hyponatremia.^{26–28} These principles are widely discussed. Yet, to our knowledge, detailed case descriptions of the hemodynamic, brain relaxation, and sodium course under an ultrasound-guided, dexamethasone-adjuvanted scalp block during extended endoscopic transsphenoidal resection, together with the postoperative osmoregulatory course, are scarce. The objective of this report is to describe the anesthetic management and the postoperative sodium and urine course of one patient who underwent endoscopic transsphenoidal resection of a giant sellar-suprasellar Rathke cleft cyst under a dexamethasone-adjuvanted scalp block and TIVA, and to discuss what the case suggests.

2. Methods

2.1. Report design and reporting guideline

This article reports one patient. It is a single-patient case report prepared according to the CAse REport (CARE) guideline.²⁹ The report covers the whole perioperative period, from multidisciplinary optimization before surgery through induction, intraoperative microdissection, and emergence to surveillance in the neuro-intensive care unit (NICU). Physiological parameters were recorded serially against predetermined target ranges. No comparator was used, and no hypothesis was tested.

2.2. Setting

The patient was treated at Dr. Saiful Anwar Regional General Hospital, Malang, East Java, Indonesia. Care was provided by the Department of Anesthesiology and Intensive Care together with the Department of Neurosurgery, Faculty of Medicine, Universitas Brawijaya.

2.3. Patient information and case selection

A 45-year-old woman (body weight 70 kg, height 150 cm, body mass index [BMI] 31.1 kg/m², class I obesity) was referred to the neurosurgical service. Symptoms had begun 12 months earlier. She reported insidious, progressive bitemporal visual field loss and chronic bifrontal headache. She had hypertension and took no medication for it. Her blood pressure at the pre-operative assessment was

146/110 mmHg (mean arterial pressure [MAP] 122 mmHg), and her heart rate was 78 beats/min. Computed tomography (CT) and 3.0-T magnetic resonance imaging (MRI) of the brain and sella turcica led to the diagnosis of a giant Rathke cleft cyst. Pre-operative risk stratification placed her in American Society of Anesthesiologists (ASA) physical status class III because of the intracranial mass effect and the hypertension.

The case was chosen for reporting because it combined a cystic sellar-suprasellar lesion that compressed the optic

chiasm, in a hypertensive patient, with a dexamethasone-adjuvanted scalp block and TIVA. Detailed case descriptions of this combination seem scarce. to be examined.

2.4. Case timeline

The timeline in Table 1 follows the CARE format. Time is given relative to the day of surgery (day 0), with earlier days shown as negative numbers and postoperative days abbreviated as POD. Calendar dates are withheld.

Table 1. CARE timeline of the case, in relative time.

Time point	Clinical event	Investigation and result	Intervention
Month –12 to day –8	Progressive bitemporal visual field loss and bifrontal headache; referral to neurosurgery	Humphrey perimetry: severe bitemporal hemianopsia; visual acuity 6/18 in both eyes; bilateral optic disc pallor. CT and 3.0-T MRI: cystic sellar-suprasellar lesion of 28 × 22 × 24 mm with a 4-mm mural nodule	Pre-operative multidisciplinary assessment; hypertension unmedicated (146/110 mmHg, MAP 122 mmHg)
Day –8 and day –7	—	Day –8: Hb 13.5 g/dL, Hct 40.2%, leukocytes 7,080/μL, platelets 308,000/μL, albumin 3.98 g/dL, BUN 17.8 mg/dL, creatinine 0.72 mg/dL, random glucose 103 mg/dL. Day –7: Na 145, K 3.56, Cl 109 mEq/L; AST 16, ALT 5 U/L	—
Day 0, before surgery	ASA class III; airway examination reassuring	Hb 14.2 g/dL, Hct 44.0%, leukocytes 7,100/μL, platelets 301,000/μL, Na 137, K 4.27, Cl 101 mEq/L, albumin 4.50 g/dL	Arterial line; induction; ultrasound-guided bilateral scalp block (0.375% ropivacaine with dexamethasone 10 mg)
Day 0, operating room (5 h)	Endoscopic endonasal transsphenoidal resection; MAP 76–82 mmHg from induction to the end of resection; EBL 60 mL	End-of-surgery arterial blood gas: pH 7.39, PaCO ₂ 34.2 mmHg, PaO ₂ 168 mmHg (FiO ₂ 0.40), BE –1.2 mmol/L, lactate 1.1 mmol/L	Propofol, fentanyl, and atracurium infusions; 2,300 mL balanced crystalloid; extubation in the operating room (GCS 15); transfer to NICU
POD 0 (first 14 h)	Awake, CPOT 0–1, RASS 0, no PONV; polyuria	Urine 2,300 mL in 14 h (2.35 mL/kg/h)	NICU monitoring; hourly urine output
POD 1	Serum sodium fell; patient clinically stable	Na 132, K 3.79, Cl 104 mEq/L; Hb 13.3 g/dL, Hct 40.8%, leukocytes 8,820/μL, platelets 247,000/μL, albumin 3.67 g/dL, glucose 168 mg/dL	Fluid restriction to 1.5 L/day of isotonic crystalloid; hypotonic infusions avoided; desmopressin not given
POD 3 (about 48 h later)	Diuresis settled	Na 138 mEq/L; urine 1.1–1.4 mL/kg/h	No hypertonic saline
POD 5	Discharged home without neurological, endocrine, or rhinological complication	Not applicable	Discharge

Notes: ASA: American Society of Anesthesiologists; BE: base excess; BUN: blood urea nitrogen; CPOT: Critical-Care Pain Observation Tool; CT: computed tomography; EBL: estimated blood loss; GCS: Glasgow Coma Scale; Hb: hemoglobin; Hct: hematocrit; MAP: mean arterial pressure; MRI: magnetic resonance imaging; NICU: neuro-intensive care unit; PONV: postoperative nausea and vomiting; POD: postoperative day; RASS: Richmond Agitation-Sedation Scale. Day 0 is the day of surgery; negative numbers are days before surgery.

The sequence runs from the onset of visual symptoms about 12 months before surgery, through referral, imaging, and laboratory work-up, to the operation, the NICU stay, and discharge on POD 5.

2.5. Clinical findings and diagnostic assessment

Ophthalmic examination included Humphrey automated perimetry and visual acuity testing, which showed severe bitemporal visual field deficits, visual acuity of 6/18 in both eyes, and bilateral optic disc pallor. Non-contrast CT of the cranium and contrast-enhanced 3.0-T MRI of the brain and sella turcica were obtained. The cystic sellar-suprasellar lesion measured 28 mm craniocaudal, 22 mm anteroposterior, and 24 mm transverse. It was homogeneously hyperintense on T1-weighted sequences and heterogeneously high in signal on T2-weighted images, and its wall and contents did not enhance. A solid, non-enhancing mural nodule of 4 mm lay on the posterior-superior wall. These features are characteristic of a Rathke

cleft cyst. The treating team judged them to favor this diagnosis over pituitary macroadenoma and sellar abscess. MRI alone can fail to separate a Rathke cleft cyst from a cystic craniopharyngioma,⁶ so histopathology of the cyst was planned.

The airway examination was reassuring. It showed a Mallampati class II view, normal cervical spine mobility, a thyromental distance of 6.0 cm, an inter-incisor distance of three fingerbreadths (4.5 cm), and no signs of obstructive sleep apnea. Direct laryngoscopy was predicted to be straightforward. Access to the airway would be limited later by head fixation and transnasal instrumentation, so an easy intubation mattered.³⁰

Blood was sampled on day –8, day –7, on the morning of surgery (day 0), and on POD 1. On day –8 the panel covered hemoglobin, hematocrit, leukocyte and platelet counts, albumin, blood urea nitrogen, creatinine, and random glucose. On day –7 serum sodium, potassium, chloride, aspartate aminotransferase, and alanine aminotransferase

were measured. The hematological, electrolyte, and albumin panel was repeated on day 0 and on POD 1, and random glucose was added on POD 1. No pre-operative pituitary hormone testing is reported for this patient.

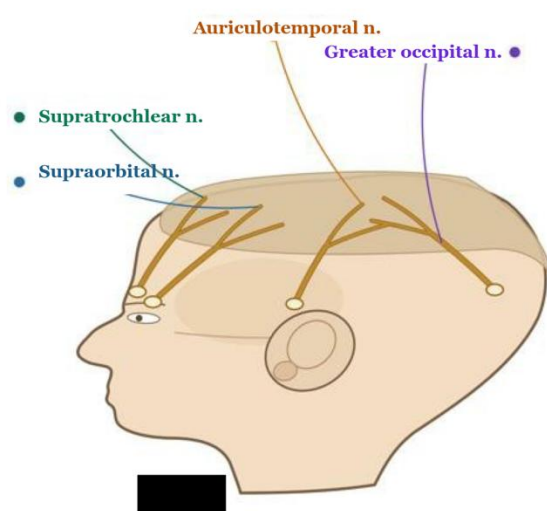
Three postoperative entities were defined for clinical reasoning in the NICU. Central DI was defined by urine output above 3.0 mL/kg/h for more than 2 consecutive hours, urine specific gravity below 1.005, urine osmolality below 300 mOsm/kg, and serum sodium above 145 mEq/L. Physiological post-stress diuresis was defined as brisk urinary excretion with preserved or mildly decreased serum sodium and intact urine concentrating capacity. Hyponatremia attributed to SIADH or cerebral salt wasting was defined by a serum sodium below 135 mEq/L. They are not validated diagnostic criteria. Comparable criteria and an algorithm for postoperative polyuria are described elsewhere.^{28,31} The team also used a urine output of 2.0 mL/kg/h as a screening threshold. No urine specific gravity, urine osmolality, urinary sodium, or serum osmolality is reported.

2.6. Therapeutic intervention

In the operating room, standard ASA monitoring was applied: five-lead electrocardiography (ECG), pulse oximetry (SpO₂), and non-invasive blood pressure (NIBP) measurement. A 20-gauge catheter was placed in the left radial artery after a negative modified Allen test and connected to a pressure transducer zeroed at the level of the external auditory meatus. This gave beat-to-beat MAP, systolic pressure, and pulse pressure variation. Preoxygenation used 100% oxygen through an anatomical facemask for 4 min and reached an end-tidal oxygen fraction above 0.90 (0.92 at induction).³⁰ Induction used midazolam 3 mg (0.043 mg/kg), fentanyl 150 mcg (2.14

mcg/kg), propofol 150 mg (2.14 mg/kg), and intravenous lidocaine 60 mg (0.86 mg/kg) to blunt airway pressor reflexes. Rocuronium 50 mg (0.71 mg/kg) provided neuromuscular blockade. Laryngoscopy with a Macintosh size 3 blade gave a Cormack-Lehane grade I view, and a 7.5-mm cuffed orotracheal tube was placed at the first pass. It was secured at 21 cm at the right corner of the mouth with waterproof tape, which left room for transnasal instrumentation.³⁰

Once the airway was secured, and before insertion of the rigid nasal speculum or head fixation, an ultrasound-guided bilateral scalp block was performed under aseptic conditions with a 10–15 MHz linear probe (SonoSite Edge II, FUJIFILM SonoSite, Bothell, WA, USA). Figure 1 illustrates the six terminal sensory nerves blocked on each side. They were the supraorbital and supratrochlear nerves at the supraorbital rim, the auriculotemporal nerve anterior to the tragus and posterior to the superficial temporal artery, and the greater and lesser occipital nerves along the superior nuchal line. The solution was 20 mL of 0.375% ropivacaine (15 mL of 0.5% ropivacaine diluted with 5 mL of normal saline; 75 mg in total, 1.07 mg/kg) with 10 mg (2.5 mL) of preservative-free dexamethasone. The procedure took 8 min. There was no vascular puncture or hematoma. The drugs and doses are listed in Table 2. The scalp block covers the forehead and scalp, including the pin and skin sites. It does not anesthetize the nasal mucosa, sphenoid, or sellar dura. Their supply comes from ethmoidal, maxillary, sphenopalatine, and meningeal branches, so general anesthesia and opioid had to cover nasal and sellar stimulation. The 10 mg of dexamethasone can also act as a systemic glucocorticoid, and this was kept in mind when postoperative glucose and leukocyte counts were read.



Lateral view (face to left) — sensory nerves of the scalp

CV, cardiovascular; CNS, central nervous system; PONV, postoperative nausea and vomiting; TIVA, total intravenous anesthesia; BIS, bispectral index; CMRO₂, cerebral metabolic rate for oxygen; MAP, mean arterial pressure.

Figure 1. Schematic illustration of the ultrasound-guided bilateral scalp block. The drawing shows the infiltration sites around the supraorbital, supratrochlear, auriculotemporal, and greater occipital nerves on both sides, with 20 mL of 0.375% ropivacaine and 10 mg of preservative-free dexamethasone. It is an illustration and contains no patient image.

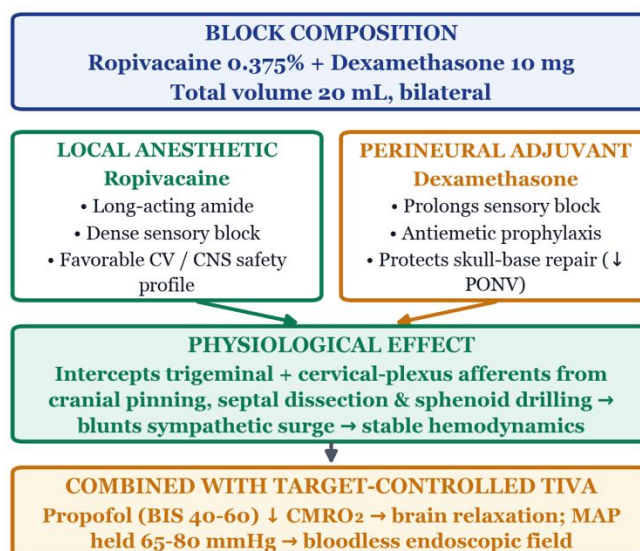


Table 2. Anesthetic management and doses.

Anesthetic phase	Agent	Dose / route	Intended effect
Pre-Induction & Sedation	Midazolam	3 mg IV bolus	GABA-A receptor potentiation; anxiolysis and retrograde amnesia without airway depression
Analgesia & Reflex Blunting	Fentanyl	150 mcg IV bolus	Mu-opioid agonism; blunts laryngoscopy and intubation pressor reflex surges
Hypnosis	Propofol	150 mg IV bolus	Depresses cerebral metabolic rate of oxygen (CMRO ₂); hypnotic
Airway Reflex Attenuation	Lidocaine	60 mg IV bolus	Membrane stabilization; suppresses tracheal irritability and sympathetic pressor response
Neuromuscular Relaxation	Rocuronium	50 mg IV bolus	Non-depolarizing blockade; facilitates smooth, atraumatic laryngoscopy and intubation
Ultrasound Regional Block	Ropivacaine 0.375%	20 mL total (75 mg); see Methods 2.6	Long-acting amide local anesthetic; scalp nerves blocked bilaterally (Figure 1)
Perineural Adjuvant	Preservative-free Dexamethasone	10 mg perineural	Proposed actions: prolongation of analgesia and systemic antiemetic effect
Maintenance Hypnosis	Propofol (infusion titrated)	50 – 200 mcg/kg/min	Reduces cerebral metabolic rate and cerebral blood flow; titrated to BIS 40–60
Maintenance Analgesia	Fentanyl infusion	50 mcg/hr	Continuous baseline systemic antinociception synergizing with regional sensory blockade
Maintenance Immobility	Atracurium infusion	6 – 12 mcg/kg/min	Immobility during microdissection near the cavernous carotid arteries
Invasive Monitoring	Radial Arterial Line	Continuous 20-G transducer	Beat-to-beat pressure monitoring for MAP titration toward 65–80 mmHg

Notes: BIS: bispectral index; GABA: gamma-aminobutyric acid; ICP: intracranial pressure; CMRO₂: cerebral metabolic rate of oxygen; IV: intravenous; MAP: mean arterial pressure. Maintenance propofol is given as an infusion in mcg/kg/min.

Anesthesia was maintained with a propofol infusion (50–200 mcg/kg/min) titrated to a bispectral index (BIS; Medtronic, Minneapolis, MN, USA) of 40–60, a fentanyl infusion at 50 mcg/h (0.71 mcg/kg/h), and an atracurium infusion at 6–12 mcg/kg/min titrated to two twitches on train-of-four (TOF) monitoring.³² Ventilation was volume-controlled. Tidal volume was 7 mL/kg of predicted body weight, and positive end-expiratory pressure (PEEP) was 5 cmH₂O. The respiratory rate was adjusted to keep end-tidal carbon dioxide (EtCO₂) at 32–35 mmHg, in line with the aim of normocapnia in patients at risk of cerebral injury.³³

The MAP target was 65–80 mmHg, a deliberate mild hypotension chosen to balance the quality of the surgical field against perfusion of the optic chiasm. Deliberate hypotension with propofol probably improves the surgical field in endoscopic sinonasal surgery, although the effect is small.³⁴

Fluids were given as a balanced crystalloid (Plasmalyte-A) under a goal-directed protocol, and 2,300 mL was infused over the 5 h, or about 6.6 mL/kg/h. Intraoperative urine output was 1,000 mL over 5 h, which is 200 mL/h or 2.86 mL/kg/h. The estimated maintenance and third-space requirement was 1,400 mL. It is an estimate, not a measured output. Together with 60 mL of estimated blood loss (EBL), it gives a total fluid loss of 2,460 mL, and the net balance is 2,300 – 2,460 = –160 mL.

Surgery was performed jointly by otorhinolaryngology and skull base neurosurgery teams, using a binostrial four-hand endoscopic endonasal transsphenoidal approach. A right vascularized Hadad-Bassagasteguy nasoseptal flap was pedicled on the posterior septal artery. A wide posterior septectomy and bilateral sphenoidotomies followed. The sellar floor was opened with a high-speed drill, the intercavernous sinuses were coagulated with bipolar cautery, and the dura was incised in a cruciate pattern. The cyst contents were evacuated and sent for microbiology and histopathology. The cyst wall was deliberately left attached to the adenohypophysis. The skull base was rebuilt in several layers with a collagen matrix inlay, the nasoseptal flap, and tissue bio-glue.

At the end of the operation, the infusions were stopped, and neostigmine 2.5 mg with atropine 1.0 mg antagonized the neuromuscular blockade. The trachea was extubated in the operating room once spontaneous breathing, eye opening on command, and a TOF ratio above 0.90 were confirmed.³² She was then taken to the NICU. Plans there were continuous hemodynamic monitoring, neurological charting, hourly diuresis measurement, and serial electrolyte sampling. Postoperative polyuria was to be assessed by excluding other causes, including perioperative intravenous fluids, before any desmopressin was given.^{28,31}

2.7. Outcome measures and follow-up

The surgical team recorded the Brain Relaxation Score at the cruciate incision of the sellar dura. The four-point scale gives 1 for perfectly relaxed or sunken, 2 for satisfactorily relaxed, 3 for firm or tense, and 4 for severely bulging or herniating, and it has been used in trials of propofol TIVA at dural opening.³⁵ The scale is made for the exposed brain. In this patient the surgical team recorded a score of 1.

After surgery, pain and sedation were scored with the Critical-Care Pain Observation Tool (CPOT) and the Richmond Agitation-Sedation Scale (RASS) over 24 h, and the Glasgow Coma Scale (GCS) was used for neurological status. PONV was noted as present or absent. Hourly urine output per kilogram of body weight and serum sodium were the main surveillance measures. Hypertensive spikes (more than 20% above baseline) and any need for rescue vasoactive drugs were recorded as events.

Values are reported as measured. Percent change from baseline is given as descriptive arithmetic only, using $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$, where T_0 is the baseline value and T_n is the later value. The baseline for laboratory values was the earliest pre-operative result. For serum sodium, the change from the day 0 pre-operative value is also given, because the sodium had already fallen from 145 to 137 mEq/L over the 7 days before surgery. Hemodynamic values are shown at baseline, after induction, hourly from 1 to 5 h, at emergence, and on POD 1. No inferential statistics were calculated. They have no meaning for one patient.

Follow-up covered the hospital stay until discharge. No causality scale was applied. The sequence of interventions and outcomes is described as it occurred, and any causal reading is offered only as interpretation in the Discussion.

2.8. Ethics and consent

The protocol was reviewed and approved by CMHC Research Center Ethics Committee, Indonesia (protocol number 2026/8942). Care followed the Declaration of Helsinki and the guidelines of the Council for International Organizations of Medical Sciences (CIOMS).^{36,37}

Before surgery, the patient and her legal proxy received a detailed explanation of the anesthetic plan, invasive arterial catheterization, the ultrasound-guided nerve block, the operative risks, and data collection. Both gave written informed consent for treatment, anesthesia, and data collection. Separate written consent covered publication of anonymized clinical details, laboratory values, and illustrations. Figure 1 is a schematic illustration and not a photograph of the patient.

3. Results

3.1. Presentation and baseline findings

The patient was a 45-year-old woman admitted electively to the neurosurgical service. She reported 12 months of progressive visual decline with bilateral visual field constriction, together with persistent, dull bifrontal headaches that responded poorly to conventional analgesics. Neuro-ophthalmological assessment showed severe bitemporal hemianopsia and a visual acuity of 6/18 in both eyes, which was reduced. Funduscopy showed bilateral optic disc pallor. The sequence of events from presentation to discharge is summarized in Table 1.

She had no history of diabetes mellitus, bronchial asthma, cerebrovascular accident, or ischemic heart disease. Her weight was 70 kg and her height 150 cm, giving a BMI of 31.1 kg/m², which corresponds to class I obesity. The baseline profile is listed in Table 3. Preoperative assessment placed her at ASA physical status III because of the intracranial mass and unmedicated hypertension. Admission blood pressure was 146/110 mmHg (MAP, 122 mmHg) and heart rate was 78 beats/min. The airway examination was reassuring: Mallampati class II, thyromental distance 6.0 cm, mouth opening of three fingerbreadths (4.5 cm), and full neck mobility.

Table 3. Baseline clinical profile and airway assessment.

Clinical domain	Baseline finding	Diagnostic / analytical significance
Age and Sex	45 years / Female	Typical age and sex for a symptomatic sellar Rathke cleft cyst
Weight and Height	70 kg / 150 cm	Basis for weight-adjusted pharmacokinetic infusion calculations
Body Mass Index (BMI)	31.1 kg/m ²	Class I obesity; requires consideration for head-up positioning and functional residual capacity
Chief Complaint	Progressive bitemporal visual field loss, 12 months	Chiasmatic compression syndrome attributed to superior cyst expansion
Magnetic Resonance Imaging (MRI)	Cystic sellar-suprasellar mass with solid mural nodule; chiasm compression	Features favoring a giant Rathke cleft cyst over sellar abscess; optic apparatus displacement
ASA Physical Status	Class III	Significant neurological mass effect and concurrent unmedicated hypertension
Baseline Blood Pressure	146/110 mmHg (MAP ~122 mmHg)	Hypertension; baseline reference for targeted deliberate hypotension
Resting Heart Rate	78 beats/min	Normocardia; preserved baseline chronotropic autonomic balance
Comorbidities	No diabetes mellitus, asthma, stroke, or CAD	Isolated endocrine-mechanical neurosurgical pathology without organ failure
Mallampati Airway Class	Class II	Good pharyngeal visualization; predicts uncomplicated direct laryngoscopy
Thyromental Distance	6.0 cm	Preserved mandibular-laryngeal anatomical distance
Mouth Opening & Neck Mobility	Three fingerbreadths (4.5 cm); full cervical mobility	Favorable geometry for rapid atraumatic orotracheal intubation prior to nasal corridor prep

Notes: ASA: American Society of Anesthesiologists; BMI: body mass index; CAD: coronary artery disease; MAP: mean arterial pressure; MRI: magnetic resonance imaging.

Preoperative laboratory values are given in Table 4. On day –8, hemoglobin was 13.5 g/dL, hematocrit 40.2%, leukocyte count 7,080/μL, platelet count 308,000/μL, and albumin 3.98 g/dL. Blood urea nitrogen was 17.8 mg/dL, serum creatinine 0.72 mg/dL, and random blood glucose 103 mg/dL. On day –7, serum sodium was 145 mEq/L, potassium 3.56 mEq/L, and chloride 109 mEq/L, with aspartate aminotransferase 16 U/L and alanine

aminotransferase 5 U/L. On day 0, before surgery, the sodium was 137 mEq/L, potassium 4.27 mEq/L, chloride 101 mEq/L, and albumin 4.50 g/dL. Hemoglobin was 14.2 g/dL, hematocrit 44.0%, leukocytes 7,100/μL, and platelets 301,000/μL. Sodium had therefore fallen by 8 mEq/L between day –7 and day 0, and the day 0 value was at the lower part of the reference interval.

Table 4. Hematological, biochemical, and hepatorenal values before and after surgery, by relative day.

Biological Parameter	Day –8	Day –7	Day 0 (preop)	POD 1	Change (Δ%)	Reference Interval
Hemoglobin (g/dL)	13.5	—	14.2	13.3	–1.5%	12.0 – 16.0
Hematocrit (%)	40.2	—	44.0	40.8	+1.5%	37.0 – 47.0
Leukocyte Count (/μL)	7,080	—	7,100	8,820	+24.6%	4,000 – 10,000
Platelet Count (/μL)	308,000	—	301,000	247,000	–19.8%	150,000 – 400,000
Serum Sodium (mEq/L)	—	145	137	132	–9.0% (–3.6% from Day 0)	135 – 145
Serum Potassium (mEq/L)	—	3.56	4.27	3.79	+6.5%	3.5 – 5.1
Serum Chloride (mEq/L)	—	109	101	104	–4.6%	98 – 107
Serum Albumin (g/dL)	3.98	—	4.50	3.67	–7.8%	3.5 – 5.2
AST / ALT (U/L)	—	16 / 5	—	—	Single value	<40 / <41
BUN / Creatinine (mg/dL)	17.8 / 0.72	—	—	—	Single value	7.0 – 20.0 / 0.6 – 1.1
Random Blood Sugar (mg/dL)	103	—	—	168	+63.1%	<200

Notes: ALT: alanine aminotransferase; AST: aspartate aminotransferase; BUN: blood urea nitrogen; POD: postoperative day. Day 0 is the day of surgery. Change (%) is calculated from the earliest value to the POD 1 value: $[(\text{POD 1 value} - \text{earliest value}) / \text{earliest value}] \times 100\%$. For sodium, the earliest value is day –7 (145 mEq/L); from day 0 (137 mEq/L) the change is –3.6%.

3.2. Diagnostic findings

CT and 3.0-Tesla MRI of the brain and sella showed a large, well-circumscribed cystic lesion of the sellar and suprasellar space, which the authors describe as a giant Rathke cleft cyst. It measured 28 mm craniocaudally, 22 mm anteroposteriorly, and 24 mm transversely. The lesion expanded the bony sella. The sellar floor was thinned and remodeled, the dorsum sellae was eroded, and the diaphragma sellae was breached superiorly, so that the lesion filled the suprasellar cistern. The optic chiasm was elevated, stretched, and flattened, the infundibulum was displaced to the left, and the normal pituitary gland was displaced laterally.

The signal was homogeneously hyperintense on T1-weighted and heterogeneously high on T2-weighted sequences. There was no enhancement of the wall or contents after intravenous gadolinium. A 4-mm solid, non-enhancing mural nodule lay on the posterior-superior wall of the cyst. These findings were characteristic of a Rathke cleft cyst, rather than pathognomonic, and the radiological differential included cystic pituitary adenoma and sellar abscess. Figure 2 is a schematic of the lesion, its relation to the optic chiasm, and the endoscopic transsphenoidal corridor.

PATHOLOGICAL SELLAR MORPHOLOGY (MRI)

- Cystic sellar-suprasellar lesion (~28 × 22 × 24 mm)
- High proteinaceous mucinous content (hyperintense T1)
- Solid mural nodule (hallmark of Rathke cleft cyst)
- Superior extension lifting and stretching the optic chiasm
- Direct microvascular ischemia of decussating nasal retinal fibers
- Clinical manifestation: Progressive bitemporal hemianopsia
- Differential: Giant Rathke cleft cyst vs. Sellar abscess / Adenoma
- Lateral compression against normal pituitary gland & stalk
- Baseline endocrinopathy risk: Stalk elongation & subclinical DI

ENDOSCOPIC SURGICAL TRAJECTORY

1. Transnasal approach through right/left sphenoid ethmoidal recess
2. Harvest of vascularized Hadad-Bassagasteguy nasoseptal flap
3. Wide anterior sphenoidotomy and posterior septectomy
4. High-speed osseous drilling of sellar floor (rostrum)
5. Cruciate dural incision exposing fibrous cyst capsule
6. Evacuation of mucoid contents -> Immediate chiasm descent
7. Preservation of pituitary gland and arachnoid barrier
8. Multi-layer reconstruction with nasoseptal flap & tissue glue
9. Critical neuroanesthetic demands: Absolute immobility & zero coughing

NEUROANESTHETIC MANDATE: MAP 65–80 mmHg avoids mucosal bleeding; Propofol-induced brain relaxation facilitates spontaneous descent of suprasellar cyst capsule into the sphenoid corridor without hypothalamic traction.

Figure 2. Schematic of the pre-operative sellar-suprasellar morphology and the endoscopic transsphenoidal corridor. The drawing shows the cystic lesion, elevation and stretching of the optic chiasm, lateral displacement of the normal pituitary gland, and the corridor through the sphenoid sinus into the sella turcica. It is a schematic and not a copy of the patient's images.

3.3. Treatment course and outcome

On the day of surgery (day 0), preoxygenation was continued to an end-tidal oxygen fraction of 0.92. Induction used intravenous midazolam 3 mg, fentanyl 150 mcg, propofol 150 mg, and lidocaine 60 mg, followed by rocuronium 50 mg; the doses are listed in Table 2. Laryngoscopy with a Macintosh size 3 blade gave a Cormack-Lehane grade I view. A 7.5-mm cuffed

endotracheal tube was placed on the first pass and secured at 21 cm at the right oral angle. After the airway was secured, bilateral ultrasound-guided scalp block was performed with a total of 20 mL of 0.375% ropivacaine and 10 mg of preservative-free dexamethasone. The sites were the supraorbital and supratrochlear nerves along the supraorbital ridge, the auriculotemporal nerves anterior to the auricle, and the greater occipital nerves along the superior nuchal line. The block took 8 minutes. There was

no vascular puncture, hematoma, or hemodynamic instability.

Hemodynamic values are shown in Table 5 and Figure 3A. MAP was 122 mmHg at baseline and 82 mmHg after induction. It then ranged from 76 to 82 mmHg until the end of resection: 78 mmHg at 1 hour, 76 mmHg at 2 hours (the lowest value, 38% below baseline), 79 mmHg at 3 hours, 77 mmHg at 4 hours, and 80 mmHg at 5 hours. At

emergence it was 84 mmHg. The target window was 65 to 80 mmHg, so the post-induction and emergence values were above the upper limit of this window. Heart rate was 78 beats/min at baseline and 70, 66, 64, 68, 66, and 70 beats/min after induction and at hours 1 to 5, and 74 beats/min at emergence. No hypertensive crisis or reflex tachycardia was recorded during head positioning or cranial pin fixation. No vasodilator, adrenergic antagonist, or vasopressor was given.

Table 5. Intraoperative parameters.

Physiological / operative parameter	Observed value	Target range	Clinical relevance
Total Surgical Duration	5.0 hours (300 min)	Not stated	Adequate window for complete cyst fenestration and nasoseptal flap repair
Total Crystalloid Administration	2,300 mL (Balanced crystalloid)	Goal-directed (target not stated)	Normovolemia maintained
Total Colloid / Blood Products	0 mL	No transfusion needed	No transfusion was required
Intraoperative Urine Output	1,000 mL (200 mL/hr; 2.86 mL/kg/hr)	>0.5 mL/kg/hr	Urine output above 0.5 mL/kg/hr during controlled hypotension
Estimated Surgical Blood Loss (EBL)	60 mL	<150 mL	Dry endoscopic field reported by the surgical team
Calculated Maintenance & Third Space	1,400 mL	Estimated requirement	Estimate of requirement, not a measured loss
Net Intraoperative Fluid Balance	-160 mL	Neutral to slightly negative	Arithmetic net of 2,300 mL input and 2,460 mL estimated output
Intraoperative Mean Arterial Pressure	76 – 82 mmHg (nadir 76 mmHg; 84 mmHg at emergence)	65 – 80 mmHg	Recorded values; chiasm perfusion was not measured
Intraoperative Heart Rate	64 – 74 beats/min	50 – 90 beats/min	No reflex tachycardia or bradycardia recorded
Bispectral Index (BIS) Score	40 – 60	40 – 60	Within target; postoperative awareness interview not reported

Notes: BIS: bispectral index; EBL: estimated blood loss; MAP: mean arterial pressure. Net balance = crystalloid 2,300 mL minus (urine 1,000 mL + estimated maintenance and third-space requirement 1,400 mL + EBL 60 mL) = -160 mL. The maintenance requirement is an estimate, not a measured output.

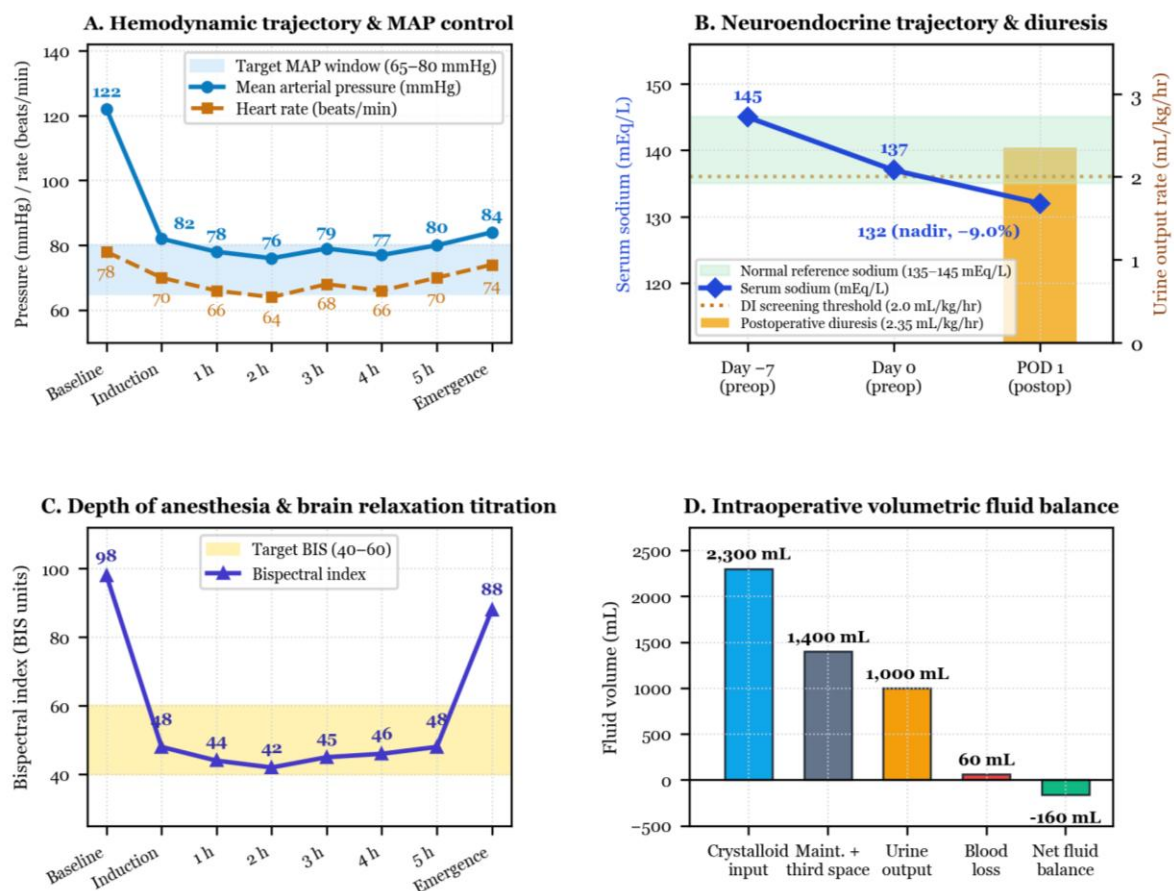


Figure 3. Perioperative parameters. (A) Mean arterial pressure (MAP, mmHg) at baseline, after induction, hourly from 1 to 5 hours, and at emergence, with the target window of 65 to 80 mmHg (recorded values 76 to 82 mmHg from induction to the end of resection; 84 mmHg at emergence), and heart rate (HR, beats/min). (B) Serum sodium (mEq/L) on day -7, day 0, and postoperative day 1 (132 mEq/L on postoperative day 1) and postoperative urine output (2.35 mL/kg/h over 14 hours). (C) Bispectral index (BIS) at the same time points. (D) Fluid balance: crystalloid 2,300 mL against urine 1,000 mL, estimated maintenance and third-space requirement 1,400 mL, and estimated blood loss 60 mL, giving a net balance of -160 mL.

Anesthesia was maintained with a propofol infusion of 50 to 200 mcg/kg/min, a fentanyl infusion of 50 mcg/h, and an atracurium infusion of 6 to 12 mcg/kg/min. The bispectral index (BIS) was 98 at baseline, 48 after induction, 44, 42, 45, 46, and 48 at hours 1 to 5, and 88 at emergence (Figure 3C). After endoscopic sphenoidotomy and craniotomy opening of the sellar dura, the surgical team recorded a Brain Relaxation Score of 1. The suprasellar cyst capsule descended into the sphenoid corridor without upward traction or blind curettage near the hypothalamic floor. The surgical team described a dry field, and endoscopic visualization through 0-degree and 30-degree rigid endoscopes was not interrupted. EBL over the operation was 60 mL, and no blood product or colloid was transfused.

The operation lasted 5 hours. The otorhinolaryngology and skull base neurosurgery teams used a binostril, four-hand endoscopic endonasal transsphenoidal approach. A right vascularized Hadad-Bassagasteguy nasoseptal flap, pedicled on the posterior septal artery, was harvested and stored in the nasopharynx. Wide posterior septectomy and bilateral sphenoidotomies exposed the sphenoid rostrum, sella turcica, clival recess, and both optic-carotid recesses. The sellar floor was opened with a high-speed drill and the intercavernous sinuses were coagulated with bipolar cautery. A craniotomy dural incision showed a thickened, milky-white cyst capsule. Opening the capsule released about 8 mL of thick, yellowish, mucinous fluid with cellular debris and cholesterol crystals. After drainage, the stretched and thinned normal pituitary gland lay displaced laterally and the superior cyst wall collapsed downward. Micro-curettage and irrigation followed, and the adherent cyst wall was left attached to the normal adenohypophysis. The skull base was reconstructed in layers with a collagen matrix inlay, the nasoseptal flap over the bony defect, and tissue bio-glue.

Intraoperative crystalloid was 2,300 mL of balanced solution (Plasmalyte-A) over 5 hours (Table 5; Figure 3D). Urine output was 1,000 mL, which is 200 mL/h or 2.86 mL/kg/h. The estimated maintenance and third-space

requirement was 1,400 mL, an estimate and not a measured output, and EBL was 60 mL. These three losses sum to 2,460 mL, so the net fluid balance was –160 mL. Arterial blood gas analysis at the end of surgery showed pH 7.39, PaCO₂ 34.2 mmHg, PaO₂ 168 mmHg on an inspired oxygen fraction of 0.40, base excess –1.2 mmol/L, and lactate 1.1 mmol/L.

After reconstruction and nasal dressing placement, the propofol, atracurium, and fentanyl infusions were stopped, and neuromuscular blockade was reversed with neostigmine 2.5 mg and atropine 1.0 mg. The train-of-four ratio was above 0.90. The patient opened her eyes within 9 minutes of stopping the infusions. She was extubated in the operating room without coughing, straining, or bucking. Her GCS score was 15 (E4M6V5), pupillary light reflexes were intact, and no lateralizing motor deficit was present. She was then transferred to the NICU.

On NICU admission the patient was awake and alert. She breathed spontaneously through a non-rebreather mask at 10 L/min with an SpO₂ of 100% and was soon weaned to room air. The CPOT score was 0 to 1 and the RASS score was 0 throughout the first 24 hours. No postoperative nausea or vomiting (PONV) occurred.

Hourly urine output and serial electrolytes were monitored (Table 6; Figure 3B). In the first 14 hours the patient produced 2,300 mL of clear urine, a rate of 2.35 mL/kg/h. This exceeded the screening threshold of 2.0 mL/kg/h used by the team and was below the 3.0 mL/kg/h used in the authors' definition of DI. On postoperative day (POD) 1, serum sodium was 132 mEq/L, down from 145 mEq/L on day –7 (–9.0%) and from 137 mEq/L on day 0 (a fall of 5 mEq/L, –3.6%). Potassium was 3.79 mEq/L, chloride 104 mEq/L, hematocrit 40.8%, hemoglobin 13.3 g/dL, albumin 3.67 g/dL, and platelets 247,000/μL (Table 4). The leukocyte count was 8,820/μL, within the reference interval but 24.6% above the earliest value, and random glucose was 168 mg/dL. These changes are of the kind expected after dexamethasone 10 mg and surgery. Urine specific gravity, urine and serum osmolality, and urine sodium were not reported.

Table 6. Postoperative neuro-intensive care surveillance.

Surveillance domain	Target	Observed outcome	Clinical interpretation
Neurological Integrity	GCS 15 (E4M6V5); no cranial nerve deficit	GCS 15 achieved immediately post-extubation	Rapid emergence after propofol TIVA
Ventilatory & Oxygenation Status	SpO ₂ > 94% on room air or minimal FiO ₂	SpO ₂ 100% on NRB 10 L/min, weaned to ambient	Emergence without coughing or bucking
Hemodynamic Stability	MAP > 65 mmHg; avoid hypertensive surges (>140 mmHg)	MAP 70 – 85 mmHg without vasopressors	Aim: protect the vascularized nasoseptal flap repair
Objective Pain & Sedation Control	CPOT score 0 – 2; RASS score 0 to –2	CPOT 0 – 1; RASS 0 throughout ICU stay	No pain behavior over 24 h; block duration was not tested
Hourly Diuresis Surveillance	Hourly urine volume < 3.0 mL/kg/hr	2,300 mL / 14 h (2.35 mL/kg/hr)	Interpreted as physiological post-stress diuresis; urine studies not reported
Serial Serum Sodium Surveillance	Serum sodium 135 – 145 mEq/L	Nadir 132 mEq/L on POD 1	Mild hyponatremia; desmopressin withheld

Notes: CPOT: Critical-Care Pain Observation Tool; DI: diabetes insipidus; GCS: Glasgow Coma Scale; NRB: non-rebreather mask; POD: postoperative day; RASS: Richmond Agitation-Sedation Scale; SpO₂: oxygen saturation.

The NICU team used the following reasoning. Central DI produces hypotonic polyuria with a rising sodium above 145 mEq/L, whereas this patient had a falling sodium. They therefore regarded the high urine output as a transient diuresis with mild hyponatremia and not as central DI. Desmopressin was withheld, and hypertonic saline was not given. Management was fluid restriction to 1.5 L/day of

isotonic crystalloid, with hypotonic infusions avoided. Over the next 48 hours urine output fell to 1.1 to 1.4 mL/kg/h and serum sodium rose to 138 mEq/L. The patient was discharged home on POD 5 with no neurological, endocrine, or rhinological complication. Her visual fields were reported as expanded. The management steps are summarized in Figure 4.

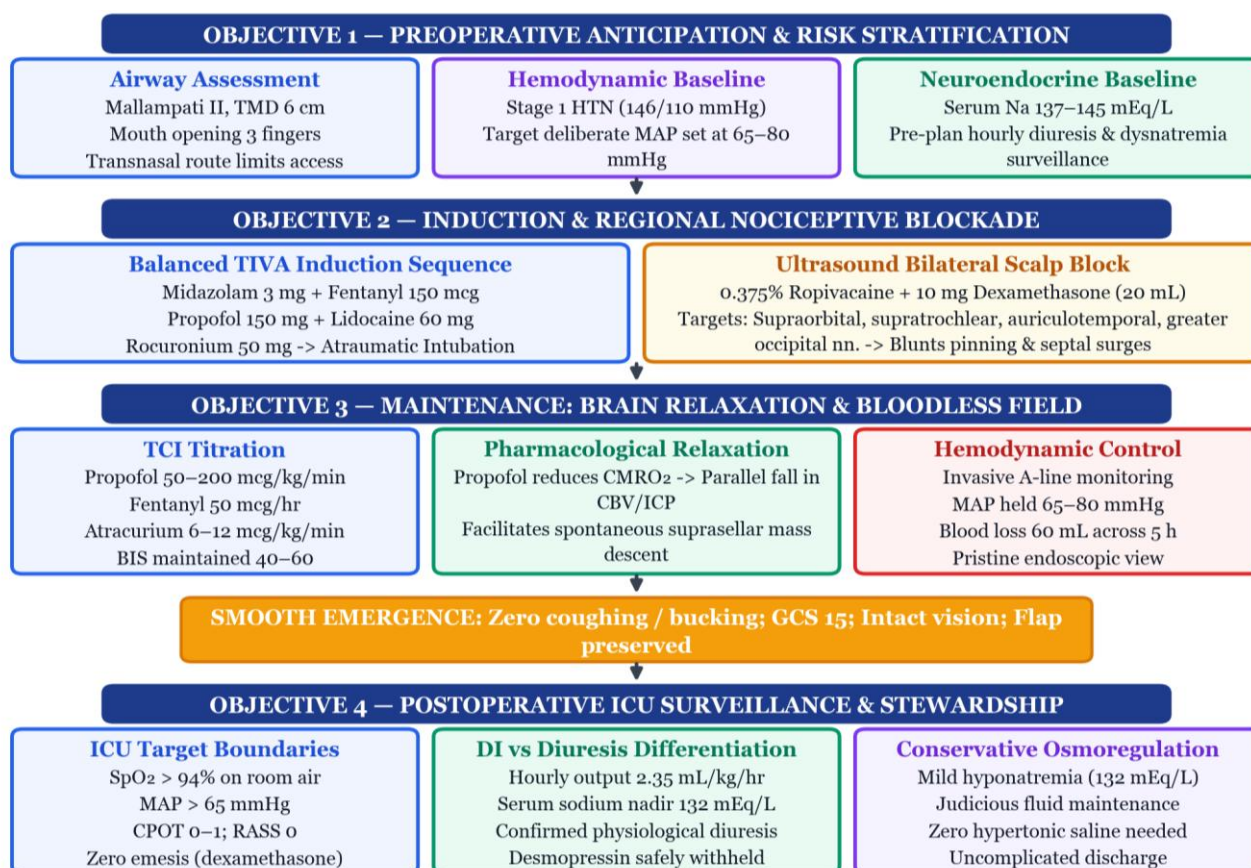


Figure 4. Management algorithm for the perioperative course. The four steps are: (1) preoperative assessment and risk stratification; (2) induction and ultrasound-guided dexamethasone-adjuvanted scalp block; (3) maintenance with total intravenous anesthesia and a MAP target of 65 to 80 mmHg; and (4) postoperative neuro-intensive care surveillance of urine output and serum sodium.

4. Discussion

4.1. Principal lesson of the case

In this patient, an ultrasound-guided scalp block with dexamethasone and propofol-based TIVA were followed by stable hemodynamics, a dry field and a smooth emergence. Early polyuria with a falling serum sodium was managed without desmopressin. One patient cannot show that the technique caused any of these outcomes. That is the main limit of this report.

Anesthesia for endoscopic transsphenoidal surgery asks for three things that pull in different directions. The first is control of the response to intense, brief stimuli. Rigid sinonasal instrumentation, septal mucosal fracture and high-speed sphenoid drilling are examples, and transsphenoidal surgery is associated with wide hemodynamic fluctuations.^{12,13} The second is a dry field in a narrow endoscopic corridor, which favors a deliberate, modest reduction of blood pressure.^{10,34} Here the target was a MAP of 65 to 80 mmHg. It had to be weighed against chronic untreated hypertension and a stretched optic chiasm. The third is a quiet emergence without coughing, so that the nasoseptal flap is not stressed.^{24,25} It also needs a way to separate the polyuria that follows sellar surgery from true central DI.^{28,31} In this woman, one regimen served all three aims. It combined a bilateral scalp block with dexamethasone placed after intubation, propofol infusion for maintenance, and structured monitoring of urine and sodium afterward. Whether this regimen served those aims better than other regimens is a question that this report cannot answer.

4.2. Comparison with the published literature

Published reports on Rathke cleft cysts concentrate on surgical, visual and endocrine outcomes and say little about anesthetic management. In a single-institution series of 38 patients, headache improved in 24 of the 25 who had it. The four visual field defects recovered. About half of the cysts extended into the suprasellar space.³ A multicenter cohort combined with a systematic review likewise found that vision commonly improves after endoscopic surgery, whereas endocrine deficits are likely to persist.⁸ That series concluded that complete aspiration with wide fenestration is usually sufficient.³ A pediatric cohort found fewer endocrine abnormalities but more recurrences after fenestration with partial wall resection.³⁸ Leaving the wall attached to the gland in this patient follows that conservative approach, although the case provides no recurrence data. None of these reports describes an anesthetic technique. This case therefore cannot be compared with them on intraoperative variables.

For the anesthetic aspects, the closest evidence comes from other settings. Randomized trials of anesthetic adjuncts in transsphenoidal surgery describe blood loss and wide hemodynamic fluctuations as expected features of the operation.^{12,13} A pterygopalatine fossa block has been used to improve the surgical field in endoscopic transsphenoidal pituitary surgery.¹⁰ Scalp block has been studied mainly in craniotomy. A meta-analysis of five randomized trials in adult craniotomy reported that scalp block and sphenopalatine ganglion block both maintained intraoperative hemodynamic stability.¹⁷ In a randomized trial, a bilateral scalp nerve block whose injectate contained dexamethasone gave steadier arterial pressures

than intravenous morphine.¹⁸ Evidence on scalp block for endonasal transsphenoidal surgery itself is thin.

Evidence on dexamethasone as an adjuvant is stronger for block duration than for other effects. A randomized trial of scalp nerve block with bupivacaine reported longer analgesia with dexamethasone (median 660 versus 420 minutes).¹⁹ A pooled analysis of trials across block sites estimated that 4 mg of perineural dexamethasone extends analgesia from about 11 to 16.5 hours. It also halved the rate of postoperative nausea and vomiting.²⁰ In awake craniotomy, a trial that compared dexmedetomidine with dexamethasone as scalp block adjuvants favored dexmedetomidine for rescue analgesia.³⁹ The 10-mg dose used here is higher than the 4 mg in the pooled estimate. The cited literature shows no added benefit at that dose in scalp block.

The literature on postoperative water and sodium disorders is richer. In a prospective multicenter series of 449 patients, suprasellar extension was among the

predictors of DI after endoscopic transsphenoidal surgery,⁴⁰ and stalk stretch on imaging predicted DI after macroadenoma resection.⁴¹ Delayed symptomatic hyponatremia is a known complication of transsphenoidal surgery.²⁶ A review of salt and water disorders after pituitary surgery names SIADH (there called syndrome of inappropriate antidiuresis) as the usual cause of hyponatremia. It describes cerebral salt wasting as very rare and a triphasic course as possible.²⁸ Fluid restriction was followed by fewer cases of hyponatremia in before-and-after comparisons, although one of them found no clear fall in readmissions.^{42,43}

4.3. Pathophysiological or mechanistic interpretation

Figure 5 sets out the mechanistic cascade proposed here. Each link below is labeled as observed in this patient or inferred from physiology and the literature, because the inferred links were not measured.

Mechanistic Divergence of Hemodynamic Autoregulation, Brain Relaxation, and Neuroendocrine Osmoregulation in Endoscopic Skull Base Surgery



Figure 5. Conceptual schematic of nociceptive stimulation during endoscopic transsphenoidal surgery and the anesthetic approach used in this case. The four tiers are: (1) surgical stimuli and sellar changes; (2) mechanisms; (3) operative effects and physiological targets; and (4) clinical endpoints. The left column is hypothetical and does not represent an observed comparator; the right column shows what was observed in this patient.

Intracranial compliance and perfusion pressure. Observed: baseline blood pressure was 146/110 mmHg (MAP 122 mmHg). MAP ranged from 76 to 82 mmHg from induction to the end of resection and was 84 mmHg at emergence. The lowest value, 76 mmHg, was 38% below baseline. Inferred: the Monro-Kellie doctrine states that brain, blood and cerebrospinal fluid volumes adjust reciprocally within the fixed cranium, and that when this buffering fails, intracranial pressure rises.^{44,45} An expanding sellar-suprasellar cyst can use up part of this buffer, and CPP (MAP minus ICP) then depends more on arterial pressure. Cerebral autoregulation may be disrupted in untreated chronic hypertension,⁴⁶ and we assumed that the lower limit of autoregulation in this patient was above normal. On that reasoning, an excessive fall in pressure could threaten the brain and the chiasm, and a pressure surge could raise capillary pressure and mucosal bleeding. Neither ICP, cerebral oximetry nor flow was monitored. The absence of ischemia rests on clinical findings alone (GCS 15, no lateralizing deficit).

Nociceptive input and what the scalp block covers. Observed: the block was placed after the airway was secured and before the nasal speculum and head fixation. Heart rate stayed between 64 and 74 beats/min and MAP remained stable. Inferred: the block anesthetizes the territories of six nerves on each side. The supraorbital and supratrochlear nerves are branches of the frontal nerve from the ophthalmic division (V1) of the trigeminal nerve, the auriculotemporal nerve comes from the mandibular division (V3), and the greater and lesser occipital nerves arise from the C2 and C2-C3 roots. Together they supply the forehead and scalp, including the pin sites and the skin. The nasal septum, turbinates, sphenoid mucosa and sellar dura are supplied by ethmoidal branches of V1, sphenopalatine branches of the maxillary division (V2) and meningeal branches. The scalp block does not reach them. Stimulation from the speculum, septectomy, drilling and dural incision was therefore covered by general anesthesia and opioid (fentanyl 150 mcg at induction and 50 mcg/h), not by the block. The stable heart rate and pressure under fentanyl and propofol cannot be attributed to the block alone. Without a comparison, it is also unknown how much the block blunted the response to head fixation, as reported in craniotomy.^{17,18}

Ultrasound guidance and local anesthetic dose. Real-time ultrasound with a 10-15 MHz linear transducer allowed the supraorbital notch, the superficial temporal artery and the superior nuchal line to be identified. The local anesthetic could then be placed around the nerves and not into the superficial temporal or occipital arteries. Intravascular injection is the main route to local anesthetic systemic toxicity (LAST). No vascular puncture or hematoma was recorded. The ropivacaine dose of 75 mg (1.07 mg/kg) was below the 3.0 mg/kg reference limit used by the authors. Sensory testing was not performed. The quality and duration of the block are unknown.

Dexamethasone. Observed: 10 mg of preservative-free dexamethasone was added to the ropivacaine. No postoperative nausea or vomiting occurred, and CPOT scores were 0 to 1. On postoperative day 1 the glucose was 168 mg/dL and the leukocyte count 8,820/ μ L. Inferred: perineural dexamethasone prolongs analgesia in trials and pooled analyses.^{19,20} Three mechanisms have been proposed: local vasoconstriction that slows clearance of the local anesthetic, a direct effect on nociceptive C fibers through glucocorticoid receptors and potassium channels, and an anti-inflammatory action with reduced nuclear

factor-kappa B signaling and lower release of cytokines such as tumor necrosis factor-alpha and interleukin-1 beta. These mechanisms come mainly from experimental work. They were not tested here. Preclinical evidence supports the safety of low-dose preservative-free perineural dexamethasone, but it is at high risk of bias,²² and the dose used here was 10 mg. A 10-mg dose also acts as a systemic glucocorticoid, and the glucose and leukocyte values are expected effects of steroid and surgical stress. The absence of nausea and vomiting matters because PONV after endoscopic skull base surgery is common and carries a risk of postoperative bleeding and CSF leak.²¹ Propofol-based TIVA is itself an antiemetic strategy,⁴⁷ so the absence of PONV in one patient says nothing about the efficacy of dexamethasone.

Propofol and volatile agents. Observed: propofol was infused at 50-200 mcg/kg/min to keep the BIS at 40 to 60, blood loss was 60 mL, and the field was dry. Inferred: volatile anesthetics dilate cerebral vessels in a concentration-dependent way. They can increase cerebral blood volume (CBV) and ICP. Propofol lowers CMRO₂, cerebral blood flow (CBF) and CBV in parallel. A network meta-analysis of 11 randomized trials found a tendency toward more satisfactory brain relaxation with propofol than with sevoflurane, isoflurane or desflurane, although the confidence intervals included no difference.²³ A role for volatile agents in mucosal bleeding is also proposed. This patient did not receive a volatile agent. The comparison is indirect.

Brain relaxation and collapse of the cyst wall. Observed: the surgical team recorded a Brain Relaxation Score of 1. After the dura was incised and about 8 mL of thick mucinous fluid was drained, the cyst wall collapsed. Inferred: drainage of the cyst is the most direct explanation for the collapse of the wall and for the view of its remaining part. Propofol-related reduction in brain volume may have made more room, but its contribution cannot be separated from that of drainage. Brain relaxation matters in this operation. A tense suprasellar capsule pressed against the diaphragma sellae can tempt the surgeon to curette upward or to apply forceful suction. That risks injury to the chiasm and the stalk. Postoperative imaging was not reported, so decompression of the chiasm is supported only by the visual field improvement that the patient reported.

Deliberate hypotension and chiasm perfusion. Observed: the target was a MAP of 65 to 80 mmHg and the recorded values were 76 to 82 mmHg. EBL was 60 mL. The patient reported wider visual fields after emergence, but no perimetry was done. Inferred: the nasal mucosa and sphenoid receive blood from the external carotid system (sphenopalatine and descending palatine arteries) and the internal carotid system (ethmoidal arteries). Oozing from the cut surfaces is therefore hard to avoid, and it obscures the endoscopic view. Controlled hypotension is used to limit bleeding in pituitary surgery.⁴⁸ Propofol-based deliberate hypotension probably improves the surgical field in endoscopic sinonasal surgery. The effect in trials was small, and the target pressures in normotensive patients were lower than here.³⁴ The other side of the balance is perfusion. The fall of 38% from baseline took MAP to a level that would be modest in a normotensive patient. In chronic hypertension and with a stretched, flattened chiasm the margin is narrower. A MAP below 60 mmHg is generally avoided in such patients. The visual field report on awakening fits preserved perfusion, yet it is

subjective. No direct measure of chiasm perfusion links it to the pressure record.

Emergence and the skull base repair. Observed: infusions were stopped and neostigmine with atropine was given. The eyes opened within 9 minutes, and the patient was extubated in the operating room without coughing or bucking (GCS 15). No CSF leak or rhinological complication was recorded before discharge. Inferred: coughing and straining raise venous and intracranial pressure and could stress the repair. The rationale for a quiet emergence is therefore sound. No pressure was measured, however. Smooth emergence, with less agitation but slower awakening, has been reported with propofol-based TIVA in a randomized comparison with sevoflurane in endoscopic transsphenoidal surgery.²⁴ The pedicled nasoseptal flap has been associated with a lower rate of CSF leak after endonasal pituitary surgery.⁴⁹ The short context-sensitive half-time of propofol may have contributed to the rapid awakening. The scalp block could have eased discomfort at the pin sites. It does not cover the trachea or the nasal packing, so the cough-free emergence cannot be credited to the block.

Stalk physiology. Antidiuretic hormone is made in the supraoptic and paraventricular nuclei of the hypothalamus, travels along axons through the infundibulum, and is stored and released from the posterior pituitary. Traction, edema or loss of blood supply at the stalk can interrupt this transport and lower circulating hormone, which causes central DI.²⁸ Stalk stretch on imaging has been linked to postoperative DI,⁴¹ and suprasellar extension was a predictor in a large series.⁴⁰ In this patient the infundibulum was displaced to the left and the cyst reached into the suprasellar space. The wall was left attached to the gland. The stalk was therefore at some risk. Observed: no polyuria of the DI pattern was documented, but urine concentration was never measured.

Polyuria and hyponatremia: the reasoning used. Observed: urine output was 2,300 mL over the first 14 hours in the NICU (2.35 mL/kg/h), and on postoperative day 1 serum sodium was 132 mEq/L. That is a fall of 13 mEq/L (9.0%) from the day -7 value of 145 mEq/L. It is a fall of 5 mEq/L (3.6%) from the pre-operative value of 137 mEq/L, which itself had dropped 8 mEq/L over 7 days. The hematocrit (40.8%), albumin (3.67 g/dL) and potassium (3.79 mEq/L) were normal, and the intraoperative balance was -160 mL after 2,300 mL of crystalloid. Urine specific gravity, urine and serum osmolality and urine sodium were not reported. The clinical reasoning was as follows. In established central DI, water loss exceeds solute loss and the sodium tends to rise,²⁸ so a falling sodium argued against DI at that time. That reasoning is not proof, because a first DI phase can be followed by a phase of antidiuresis in a triphasic course.²⁸

Several explanations for polyuria with hyponatremia after sellar surgery cannot be separated without urine data. They include transient SIADH from nonosmotic ADH release provoked by pain, stress, general anesthesia or pituitary surgery,⁵⁰ the early phase of a triphasic response,²⁸ and cerebral salt wasting, although that is very rare after pituitary surgery.²⁸ Osmotic diuresis from steroid-related hyperglycemia (glucose 168 mg/dL) and a glucocorticoid effect on water excretion are also possible. So is the fluid load of 2,300 mL of crystalloid, since other causes of increased urine output, including perioperative intravenous fluids, should be excluded before polyuria is called DI.²⁸ The normal hematocrit, albumin and potassium and the neutral balance argue against marked

hypovolemia, but they do not distinguish the other candidates. The team withheld desmopressin. They restricted fluids to 1.5 L/day of isotonic crystalloid, avoiding hypotonic infusions. Over 48 hours urine output fell to 1.1–1.4 mL/kg/h and sodium rose to 138 mEq/L without hypertonic saline. This course fits transient water excess, for which fluid restriction is first-line.^{28,50} Spontaneous resolution would give the same picture. One patient cannot decide between the alternatives.

4.4. Implications for practice

A clinician managing a similar patient could draw several points from this case, within the limits of a single report. Hemodynamic goals can be set against the patient's own baseline pressure as well as against population values. The reason for accepting a given fall should be recorded with whatever perfusion monitoring is available. A scalp block can be placed under ultrasound after the airway is secured and before head fixation, with the total local anesthetic dose calculated against body weight. Dexamethasone may be added, but this case does not show that it improves the outcome, and the 10-mg dose has less support than lower doses.^{20,22}

The postoperative lesson is better supported. After sellar surgery, a protocol could record hourly urine output and check serum sodium at set intervals. It could also obtain urine osmolality and sodium whenever urine output exceeds a stated threshold. Polyuria could then be classified before treatment. Desmopressin should not be given reflexively for polyuria, particularly when the sodium is falling. Several causes of polyuria do not need it, and an algorithm exists to avoid unnecessary treatment.^{28,31} If the picture points to water excess, fluid restriction is a reasonable first step,^{28,42,43} with continued monitoring in case a polyuric phase follows. These points apply to a woman with a cystic sellar lesion, normal pre-operative renal function and a wall left attached to the gland. They may not transfer to patients with adenomas, stalk transection or other comorbidity.

4.5. Strengths

The record of this case is detailed. Arterial pressure, heart rate and BIS are available at hourly points, and the doses, weights, fluid volumes, blood loss, arterial blood gas values and the laboratory trajectory are all stated. The postoperative course includes the urine output, the sodium values, the clinical reasoning and the management that followed. The report also states which data are missing. The reader can see where the reasoning rests on inference. Consent for treatment and for publication was obtained separately, ethics approval is documented, and the report follows the CARE guideline.

4.6. Limitations

This is a single case. It has no comparator and cannot establish efficacy, safety or superiority of the scalp block, of dexamethasone or of propofol TIVA over other regimens. Several concurrent factors could account for the hemodynamic findings, including fentanyl, propofol, positioning, the 5-hour duration of surgery, the surgeon's technique and the patient's own baseline. The block was given together with dexamethasone, so the effects of the two cannot be separated. The sodium of 145 mEq/L on day -7 fell to 137 mEq/L before surgery. Several factors may have influenced the postoperative sodium, including 2,300 mL of crystalloid, dexamethasone, hyperglycemia and surgical stress.

Important measurements are missing. Urine osmolality, specific gravity and sodium were not obtained, so the cause of polyuria and hyponatremia is not resolved. Intracranial pressure, cerebral oximetry and flow were not monitored, so cerebral and chiasm perfusion cannot be assessed. No sensory testing was done to show the extent or duration of the block. No postoperative perimetry was reported, and the visual improvement rests on the patient's report. No endocrine panel was reported, and no pre-operative baseline either. Follow-up ended at discharge on postoperative day 5, so recurrence of the cyst and later endocrine function are unknown.

4.7. Recommendations for future work

The question raised by this case needs a comparative design. A randomized or at least comparative series of scalp block with and without dexamethasone in endoscopic transsphenoidal surgery should use predefined outcomes. These are the hemodynamic response at head fixation, speculum placement, drilling and dural incision, blood loss, a rated surgical field, opioid use, emergence quality, PONV, duration of analgesia and glycemia. Dexmedetomidine as an alternative adjuvant should be among the comparators.³⁹

For the postoperative course, a prospective protocol should record hourly urine output with urine osmolality, urine sodium and serum sodium. Copeptin could be added where available.^{51,52} Follow-up should include formal perimetry, a full endocrine panel and imaging over a period long enough to detect recurrence,³ because the recurrence of cystic lesions has been reported after years.

5. Conclusion

This report described the anesthetic management and the sodium and urine course of one 45-year-old woman who underwent endoscopic transsphenoidal resection of a sellar-suprasellar Rathke cleft cyst. Under an ultrasound-guided bilateral scalp block with dexamethasone and propofol-based TIVA, MAP ranged from 76 to 82 mmHg and blood loss was 60 mL. Emergence was free of coughing. She went home on postoperative day 5 without neurological, endocrine or rhinological complication. On the first postoperative day she had polyuria of 2.35 mL/kg/h and a serum sodium of 132 mEq/L. This was managed with fluid restriction and without desmopressin, and the sodium returned to 138 mEq/L within 48 hours. The case suggests that this combination was tolerated by a similar patient and that polyuria with a falling sodium should be classified with urine osmolality and sodium before desmopressin is considered. This is one patient. There was no comparator and no urine osmolality. The case cannot show that the block or the dexamethasone produced these findings, and the cause of the polyuria remains unresolved.

6. Declarations

Ethical approval and consent to participate. This case report was approved by the CMHC Research Center Ethics Committee, Indonesia, under protocol number 2026/8942. The report follows the CARE guideline and the Declaration of Helsinki. Written informed consent was obtained from the patient and her legal proxy before surgery for treatment, anesthesia and data collection.

Consent for publication. Separate written informed consent was obtained from the patient for publication of

this case, including anonymized clinical details, laboratory values and illustrations. Figure 1 is a schematic illustration and contains no patient image or identifier.

Availability of data and materials. All quantitative data and observations generated during this case are contained within the article and its tables and figures. Further anonymized details are available from the corresponding author on reasonable request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. A.A.F. conceived the report, performed the ultrasound-guided scalp block and administered the anesthesia (Conceptualization, Investigation), collected the clinical data (Data curation), and drafted the manuscript (Writing – original draft). B.H.L. contributed to the anesthetic design and supervised intraoperative hemodynamic management and critical care monitoring (Methodology, Supervision), and revised the manuscript (Writing – review & editing). D.A.S. took part in pre-operative risk stratification and in the design of the neuroendocrine surveillance protocol (Investigation, Validation), prepared the descriptive analysis of the recorded values (Formal analysis), and reviewed the manuscript critically (Writing – review & editing). All authors approved the final manuscript and agree to be accountable for all aspects of the work.

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