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Multimodal Neuroanesthesia with an Ultrasound-Guided Dexamethasone-Adjuvanted Scalp Block for Endoscopic Transsphenoidal Resection of a Giant Sellar-Suprasellar Rathke Cleft Cyst: A Case Report

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ABSTRACT

Introduction: Sellar and suprasellar masses commonly present with visual failure and endocrinopathy, and the endoscopic endonasal transsphenoidal route is now the standard corridor for their resection. The procedure imposes intense but intermittent nociceptive surges upon a narrow, blood-intolerant surgical field, so the neuroanesthetic plan must reconcile profound analgesia, brain relaxation, hemodynamic stability, and rapid neurological emergence. We describe the perioperative management of a giant Rathke cleft cyst using a multimodal regimen anchored by a regional scalp block.

Case Presentation: A 45-year-old woman (body mass index 31.1 kg/m²) presented with one year of progressive bitemporal visual field narrowing. Magnetic resonance imaging showed a cystic sellar-suprasellar mass with a mural nodule compressing the optic chiasm, favoring a giant Rathke cleft cyst over a sellar abscess. She was American Society of Anesthesiologists physical status III with stage 1 hypertension. Under total intravenous anesthesia titrated to a bispectral index of 40 to 60, a bilateral scalp block using 0.375% ropivacaine with 10 mg dexamethasone in 20 mL was performed after induction to blunt the sympathetic response. Mean arterial pressure was held at 65 to 80 mmHg across the five-hour endoscopic transnasal resection with sphenoidectomy and nasoseptal flap reconstruction. Emergence was smooth without bucking. In intensive care the patient was Glasgow Coma Scale 15 with no deficit; urine output reached 2.35 mL/kg/hr and sodium fell mildly to 132 mEq/L, and both were managed conservatively without desmopressin.

Conclusion: Integrating a fully endoscopic transsphenoidal approach with a dexamethasone-adjuvanted scalp block and target-controlled total intravenous anesthesia delivered a bloodless field, immaculate hemodynamic control, and prompt neurological recovery. Structured intensive care surveillance for diabetes insipidus and dysnatremia secured a safe trajectory, offering a transferable framework for complex sellar tumor anesthesia.

1. Introduction

Space-occupying lesions of the sellar and suprasellar region constitute one of the most demanding intersections of neurosurgery and neuroanesthesia, because they combine a hazardous microanatomy with a fragile neuroendocrine axis and a surgical corridor that tolerates almost no bleeding. Rathke cleft cysts are benign, non-neoplastic, epithelium-lined cysts that arise from the embryological remnants of Rathke's pouch, and although the majority remain small and clinically silent throughout life, a minority enlarge progressively and compress the structures that surround the sella turcica.[1] Superior expansion of such a cyst characteristically encroaches upon the optic chiasm and produces the classical syndrome of bitemporal hemianopsia, while lateral and inferior growth may compress the normal pituitary gland and generate a spectrum of endocrine disturbances that ranges from isolated hormone deficiency to panhypopituitarism.[1,2]

Headache, visual deterioration, and hypopituitarism together account for most symptomatic presentations, and long-term cohorts confirm that these cysts, once symptomatic, rarely regress without intervention.[3,4]

The differential diagnosis of a cystic sellar mass is broad and clinically consequential, because it determines both the urgency and the technique of surgery. A giant Rathke cleft cyst must be distinguished from a cystic pituitary adenoma, a craniopharyngioma, and, importantly, an infective sellar abscess, since the last of these carries a distinct microbiological and inflammatory profile and a different postoperative course.[5,6] Radiological features, including the presence of a mural nodule and the signal characteristics of the cyst contents, guide but do not always resolve this distinction, and the definitive diagnosis frequently rests upon intraoperative inspection and histopathology.[6]

The surgical management of symptomatic sellar and suprasellar masses has been transformed over the past two decades by the ascendancy of the fully endoscopic endonasal transsphenoidal approach, which has largely superseded the microscopic technique and the open transcranial route for lesions confined to the sella and the suprasellar cistern. The endoscope offers panoramic visualization of the sphenoid sinus, the sella, and the suprasellar space, and permits maximal safe resection with reduced sinonasal morbidity and neurological injury.[7,8] Contemporary comparative series confirm that the endoscopic corridor achieves high rates of cyst evacuation with acceptable recurrence and favorable quality-of-life outcomes, and ongoing refinements, including exoscopic and endoscopic-assisted variants, continue to optimize the balance between radicality and the preservation of pituitary function.[2,7,9]

This surgical evolution has imposed a parallel and equally demanding evolution upon neuroanesthetic practice. The anesthesiologist confronted with an endoscopic transsphenoidal resection must simultaneously maintain adequate cerebral perfusion pressure, provide optimal brain relaxation to facilitate the descent of the suprasellar component into the surgical field, secure a bloodless corridor through tightly controlled hemodynamics, and enable a rapid and smooth emergence for immediate neurological assessment. These requirements are frequently in tension, and they are complicated by the intense, intermittent nociceptive stimuli generated during rigid nasal speculum insertion, septal mucosal dissection, and high-speed osseous drilling of the sphenoid rostrum. The sympathetic surges provoked by these maneuvers can produce acute hypertensive episodes that increase mucosal bleeding, obscure the endoscopic view, and threaten the delicate skull base repair.[10,11]

To intercept this nociceptive barrage and stabilize the hemodynamic environment, regional anesthesia has increasingly been integrated into neurosurgical practice. The scalp block, traditionally deployed in open craniotomy to blunt the hemodynamic response to head pinning and scalp incision, delivers comprehensive multimodal analgesia and sympathetic attenuation, and a substantial body of randomized evidence now documents its efficacy in reducing perioperative pain, opioid consumption, and hemodynamic lability in neurosurgical patients.[12-15] The pharmacological value of the block can be extended by adjuvants, of which perineural dexamethasone is among the best characterized, prolonging sensory blockade and contributing systemic antiemetic benefit.[16,17,18] The application of a dexamethasone-adjuvanted scalp block to an endoscopic transsphenoidal resection, where the primary nociceptive input arises from the trigeminal territory of the face and nasal cavity, represents a logical but comparatively underdescribed extension of these principles.

The novelty of this report lies in its detailed, step-by-step account and physiological analysis of a comprehensive multimodal neuroanesthetic regimen, built around an ultrasound-guided scalp block using ropivacaine adjuvanted with dexamethasone and combined with target-controlled total intravenous anesthesia, applied to an extended endoscopic transsphenoidal resection of a giant sellar-suprasellar Rathke cleft cyst. The aim of this report is to present the perioperative anesthetic and intensive care management of this case, to correlate the anesthetic strategy with the underlying pathophysiology of the

lesion and the surgical corridor, and to derive from the experience a structured, evidence-based framework for the anesthetic conduct of complex sellar tumor resection, with particular attention to the prevention of hemodynamic instability and to the early recognition of postoperative neuroendocrine derangement.

2. Case Presentation

Preoperative evaluation and patient optimization

A 45-year-old woman, weighing 70 kg with a height of 150 cm and a corresponding body mass index of 31.1 kg/m², was admitted to the neurosurgical service with a primary complaint of progressive visual field narrowing that had worsened insidiously over the preceding twelve months. Initial ophthalmological evaluation at a peripheral hospital had confirmed severe bitemporal visual field deficits, prompting referral for advanced neuroimaging. Cranial computed tomography identified a substantial intracranial mass, and subsequent high-resolution magnetic resonance imaging delineated a complex cystic mass with a solid mural nodule occupying the sellar and suprasellar regions and exerting significant mass effect, elevating and compressing the optic chiasm. The radiological differential diagnosis principally considered a giant Rathke cleft cyst versus a sellar abscess, a distinction of practical importance given the divergent implications of an infective lesion for perioperative management.[5] The baseline demographic, clinical, and airway characteristics of the patient are summarized in Table 1.

Table 1. Baseline demographic, clinical, and airway characteristics.

Parameter	Finding	Clinical relevance
Demographic and anthropometric profile		
Age / sex	45 years / female	Peak age range for symptomatic Rathke cleft cyst
Weight / height	70 kg / 150 cm	Basis for weight-based infusion dosing
Body mass index	31.1 kg/m²	Class I obesity; airway and positioning consideration
Presentation and diagnosis		
Chief complaint	Progressive bitemporal visual narrowing, 12 months	Optic chiasm compression
Imaging (MRI)	Cystic sellar-suprasellar mass, mural nodule, chiasm compression	Giant Rathke cleft cyst vs sellar abscess
Risk stratification and airway		
ASA physical status	III	Mass effect + stage 1 hypertension
Baseline blood pressure	**146/110 mmHg**	Stage 1 essential hypertension
Comorbidities	None (no DM, asthma, CVA, cardiac disease)	Otherwise unremarkable history
Mallampati / neck mobility	Class II / normal	Predicts uncomplicated intubation
Thyromental distance	6 cm	Reassuring airway geometry
Mouth opening	Three fingerbreadths	Adequate laryngoscopic access

Preoperative assessment in the anesthesia clinic assigned an American Society of Anesthesiologists physical status classification of III, attributed to the severe neurological mass effect and concurrent stage 1 essential hypertension, with a baseline blood pressure of 146/110 mmHg. The patient denied any history of diabetes mellitus, bronchial asthma, cerebrovascular accident, or cardiac disease. Airway examination revealed a Mallampati class II view with normal cervical spine mobility, a thyromental distance of 6 cm, and a mouth opening of three fingerbreadths, together predicting an uncomplicated

endotracheal intubation, an assessment of particular value given that the transnasal corridor precludes ready access to the airway once surgery is underway. As detailed in Table 2, the preoperative laboratory parameters were tracked across three time points during the admission and were reassuring, with preserved hematological indices, normal renal and hepatic function, and, importantly, a baseline serum sodium at the upper end of the reference range that provided a secure reference against which any postoperative neuroendocrine disturbance could be interpreted.

Table 2. Preoperative laboratory parameters across the admission.

Parameter	December 8	December 9	December 16	Reference
Hemoglobin (g/dL)	13.5	-	14.2	12.0-16.0
Leukocytes (/µL)	7,080	-	7,100	4,000-10,000
Hematocrit (%)	40.2	-	44.0	37-47
Platelets (/µL)	308,000	-	301,000	150,000-400,000
Sodium (mEq/L)	-	145	137	135-145
Potassium (mEq/L)	-	3.56	4.27	3.5-5.1
Chloride (mEq/L)	-	**109**	101	98-107
Albumin (g/dL)	3.98	-	4.50	3.5-5.2
AST / ALT (U/L)	-	16 / 5	-	<40 / <41
BUN / creatinine (mg/dL)	17.8 / 0.72	-	-	7-20 / 0.6-1.1
Random blood sugar (mg/dL)	103	-	-	<200

The preoperative optimization strategy therefore concentrated on the control of the patient's hypertension, on the anticipation of the neuroendocrine consequences of pituitary manipulation, and on the design of an anesthetic plan capable of delivering the competing objectives of brain relaxation, hemodynamic stability, and rapid emergence. Because the lesion abutted the optic apparatus and the pituitary stalk, the anesthetic and surgical teams jointly agreed in advance upon strict intraoperative blood pressure targets and upon a structured postoperative surveillance protocol for diabetes insipidus and dysnatremia, both of which are recognized sequelae of transsphenoidal surgery and both of which are most safely managed when anticipated rather than discovered.[19,20]

Intraoperative anesthetic management

The surgical plan, formulated jointly by the otorhinolaryngology and neurosurgery teams, comprised an endoscopic transnasal tumor excision, a partial septectomy, the elevation of a vascularized nasoseptal flap for skull base reconstruction, and an extensive sphenoidectomy. In the operating theater, standard American Society of Anesthesiologists monitors were applied, including continuous electrocardiography, non-invasive and subsequently invasive arterial blood pressure monitoring, and pulse oximetry, and an intravenous line was established with an 18-gauge cannula. Following preoxygenation, general anesthesia was induced by a carefully titrated intravenous sequence comprising midazolam 3 mg for anxiolysis, fentanyl 150 mcg for analgesia and reflex blunting, propofol 150 mg for hypnosis, and lidocaine 60 mg to attenuate the airway reflexes and the hemodynamic response to laryngoscopy. Neuromuscular blockade was achieved with rocuronium 50 mg, which allowed a smooth and atraumatic endotracheal intubation. The complete anesthetic and multimodal regimen, together with the pharmacological rationale for each component, is presented in Table 3.

Table 3. Multimodal neuroanesthetic regimen and pharmacological rationale.		
Phase / agent	**Dose**	**Rationale**
Induction		
Midazolam	3 mg IV	Anxiolysis, amnesia
Fentanyl	150 mcg IV	Analgesia, blunting of laryngoscopy response
Propofol	150 mg IV	Hypnosis; reduces $CMRO_2$ and ICP
Lidocaine	60 mg IV	Attenuates airway reflexes and pressor response
Rocuronium	50 mg IV	Neuromuscular blockade for atraumatic intubation
Regional block (post-induction)		
Ropivacaine 0.375%	20 mL total, bilateral	Long-acting sensory blockade of scalp nerves
Dexamethasone (perineural)	10 mg	Prolongs block duration; antiemetic prophylaxis
Nerve targets	Supraorbital, supratrochlear, auriculotemporal, greater occipital	Blunts sympathetic surge to pinning and dissection
Maintenance (target-controlled infusion)		
Propofol	50-200 mcg/kg/min	Brain relaxation; BIS 40-60
Fentanyl	50 mcg/hr	Continuous analgesia
Atracurium	6-12 mcg/kg/min	Immobility for microdissection near carotid

Immediately after induction, and before the application of the rigid nasal instrumentation, a bilateral scalp block was performed to provide intense regional analgesia and to mitigate the substantial sympathetic outflow associated with cranial fixation and septal dissection. As shown in Figure 1, which depicts the anatomical targets and the pharmacology of the block, the technique employed a mixture of 0.375% ropivacaine combined with 10 mg of preservative-free dexamethasone in a total volume of 20 mL, distributed to target the supraorbital, supratrochlear, auriculotemporal, and greater occipital nerves bilaterally. The selection of ropivacaine, a long-acting amide local anesthetic with a favorable cardiovascular and neurological safety profile, reflected the intention to secure a prolonged and dense sensory blockade, while the addition of perineural dexamethasone was chosen both to extend the duration of that blockade and to confer a systemic antiemetic effect of particular value in neurosurgery.[16-18]

Anesthesia was maintained using a balanced technique built upon continuous target-controlled infusions of fentanyl at 50 mcg/hr, atracurium at 6 to 12 mcg/kg/min, and propofol at 50 to 200 mcg/kg/min, each adjusted dynamically to maintain a bispectral index between 40 and 60. Propofol was selected as the primary hypnotic precisely because of its neurophysiological profile, since its dose-dependent reduction of the cerebral metabolic rate for oxygen produces, through preserved flow-metabolism coupling, a proportional fall in cerebral blood flow and cerebral blood volume, and thereby a desirable reduction in intracranial pressure and a state of brain relaxation that facilitates surgical exposure.[10,11] The surgical procedure spanned five continuous hours. Throughout this period, hemodynamic parameters were controlled tightly by means of continuous invasive arterial blood pressure monitoring, with the mean arterial pressure held deliberately between 65 and 80 mmHg in order to minimize mucosal oozing and preserve a pristine endoscopic view while simultaneously safeguarding cerebral perfusion. The intraoperative fluid strategy was restrictive and goal-directed, and a comprehensive summary of the intraoperative fluid balance and hemodynamic profile is presented in Table 4. The evolution of the perioperative hemodynamic and neuroendocrine variables across the successive phases of care is depicted in Figure 2.

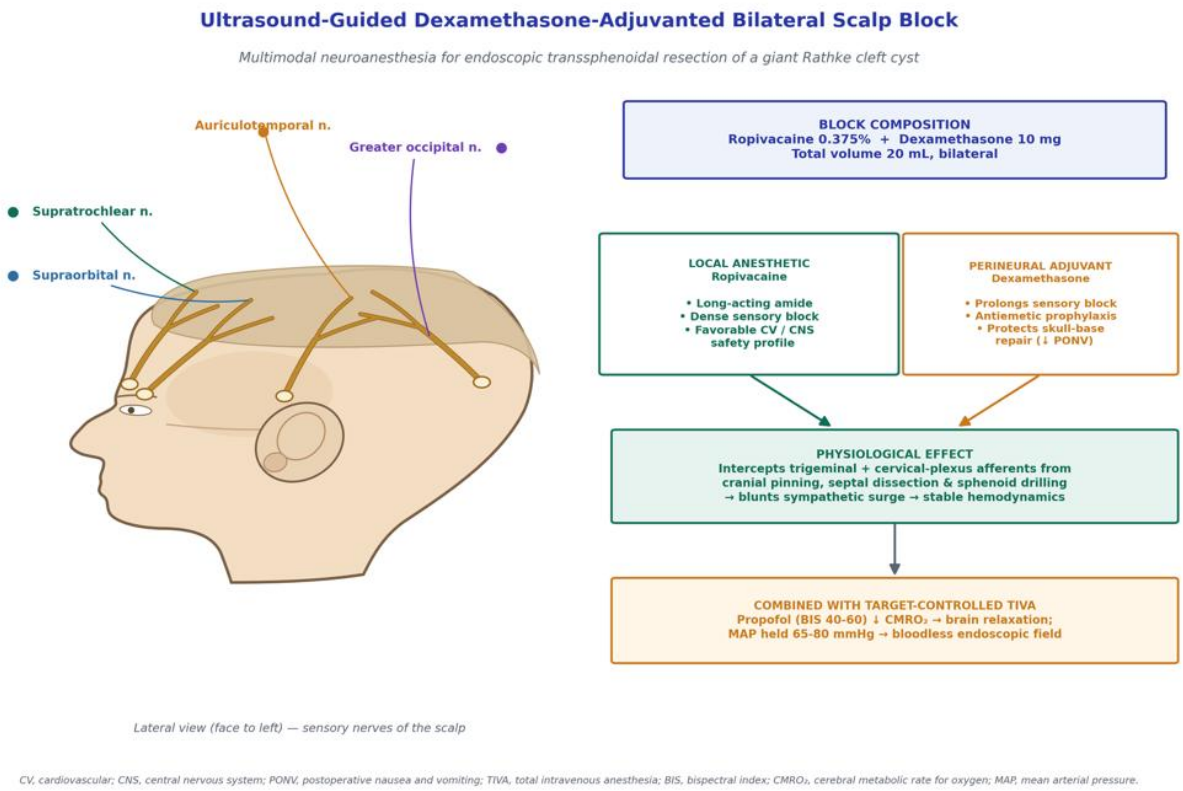

Figure 1. Anatomical targets and pharmacology of the ultrasound-guided bilateral scalp block. The block deposits 0.375% ropivacaine with 10 mg preservative-free dexamethasone (20 mL total) around the supraorbital, supratrochlear, auriculotemporal, and greater occipital nerves, intercepting trigeminal and cervical-plexus afferents to blunt the sympathetic response to cranial fixation and septal dissection. Perineural dexamethasone prolongs sensory blockade and provides systemic antiemetic prophylaxis. $CMRO_2$, cerebral metabolic rate for oxygen.

Table 4. Intraoperative fluid balance and hemodynamic profile.	
Parameter	**Recorded value**
Total surgical duration	5 hours
Total crystalloid input	2300 mL
Total colloid / blood input	0 mL
Urine output	1000 mL
Estimated blood loss	60 mL
Total maintenance + output	1400 mL
Net fluid balance	-280 mL
Average mean arterial pressure	75-85 mmHg
Average heart rate	60-80 beats/min

Postoperative intensive care management

Following meticulous dissection and complete extirpation of the mass, the patient emerged smoothly from anesthesia without coughing or bucking, so that no sudden rise in intracranial pressure occurred that might have compromised the fragile skull base repair. She was transferred to the intensive care unit for continuous monitoring. On admission she was fully awake, breathing spontaneously through a non-rebreather mask at 10 liters per minute with a peripheral oxygen saturation of 100%, and her neurological status was excellent, with a Glasgow Coma Scale score of 15, comprising eye-opening 4, motor 6, and verbal 5, and with no lateralizing motor deficit, indicating functional preservation of the surrounding neural parenchyma. The immediate postoperative laboratory parameters are documented in Table 5, and the specific therapeutic targets and neuroendocrine surveillance protocol applied in the intensive care unit are summarized in Table 6.

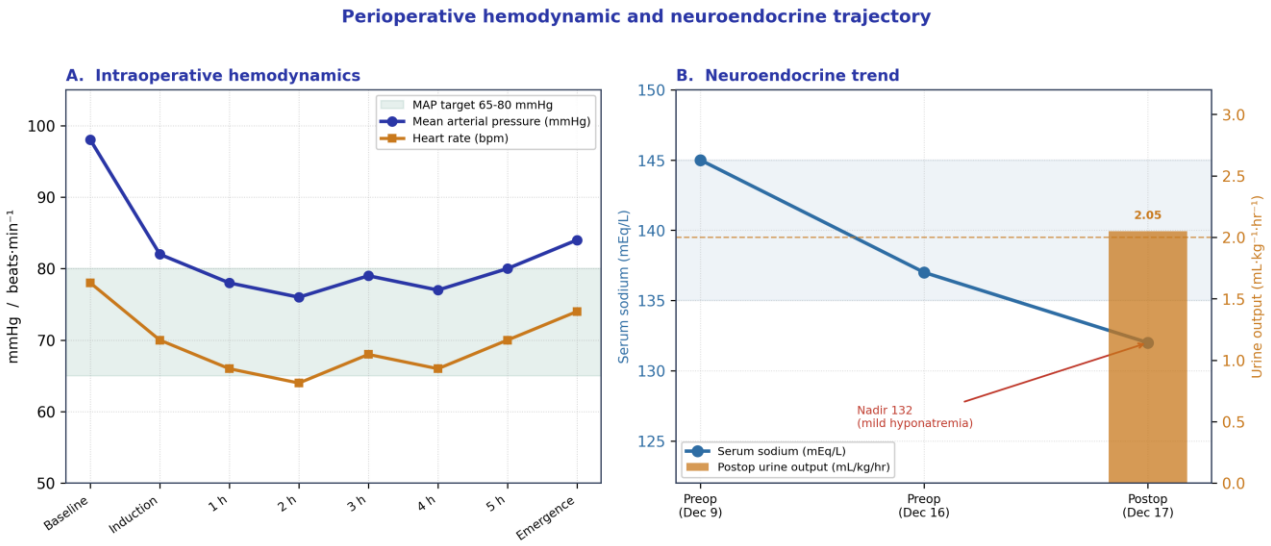

Figure 2. Perioperative hemodynamic and neuroendocrine trends across the phases of care. (A) Mean arterial pressure was maintained within the target window of 65 to 80 mmHg throughout the five-hour endoscopic resection while heart rate remained stable. (B) Serum sodium fell from a preoperative peak of 145 mEq/L to a postoperative nadir of 132 mEq/L, and urine output rose to 2.35 mL/kg/hr in the early postoperative period, prompting diabetes insipidus surveillance that was managed conservatively. MAP, mean arterial pressure; POD, postoperative period.

Table 5. Immediate postoperative laboratory parameters (December 17).

Parameter	Value	Reference	Interpretation
Sodium (mEq/L)	**132**	135-145	**Mild dilutional hyponatremia**
Potassium (mEq/L)	3.79	3.5-5.1	Normal
Chloride (mEq/L)	104	98-107	Normal
Hemoglobin (g/dL)	13.30	12.0-16.0	Normal
Leukocytes (/µL)	8,820	4,000-10,000	Normal postoperative range
Hematocrit (%)	40.80	37-47	Normal
Platelets (/µL)	247,000	150,000-400,000	Normal
Albumin (g/dL)	3.67	3.5-5.2	Normal
Random blood sugar (mg/dL)	168	<200	Mild stress hyperglycemia

The primary focus of intensive care shifted to precise hemodynamic regulation and to vigilant monitoring for neuroendocrine complications, most notably central diabetes insipidus, which is a recognized consequence of mechanical manipulation of the pituitary stalk. Strict hourly fluid balance charting was instituted. Over the first fourteen hours after surgery the patient produced 2300 mL of urine, corresponding to a relatively high output of 2.35 mL/kg/hr, a value that mandated close scrutiny for evolving diabetes insipidus but that, in the presence of a preserved and only mildly reduced serum sodium and appropriate urine characteristics, was interpreted as a physiological postoperative diuresis rather than as established vasopressin deficiency.[19,20] As detailed in Table 6, the critical care team maintained explicit therapeutic targets of an oxygen saturation greater than 94%, a mean arterial pressure greater than 65 mmHg, a Critical-Care Pain Observation Tool score of 0 to 2, and a Richmond Agitation-Sedation Scale score between 0 and minus 2, and the patient met these targets throughout. The single laboratory abnormality of note was a mild dilutional hyponatremia, with a serum sodium of 132 mEq/L, which was managed conservatively with judicious fluid balance and did not require hypertonic correction.[21,22]

Table 6. Intensive care targets and neuroendocrine surveillance protocol.		
Domain	**Target / action**	**Rationale**
Physiological targets		
Oxygen saturation	> 94%	Adequate oxygenation
Mean arterial pressure	> 65 mmHg	Cerebral perfusion; protect skull base repair
CPOT (pain)	0-2	Adequate analgesia, avoid pain-driven hypertension
RASS (sedation)	0 to -2	Calm, rousable for neurological assessment
Neuroendocrine surveillance		
Hourly urine output	Chart every hour	Detect diabetes insipidus (output 2.35 mL/kg/hr observed)
Serial serum sodium	Repeated measurement	Detect hyper- or hyponatremia (nadir 132 mEq/L)
Fluid balance	Strict input-output charting	Distinguish physiological diuresis from DI
Desmopressin	Withheld	Sodium preserved/low; DI not confirmed

The combination of a smooth emergence, an intact neurological examination, and a stable, well-monitored neuroendocrine course permitted a rapid and safe recovery. Exogenous desmopressin was not required, the mild hyponatremia resolved with conservative measures, and the patient's trajectory confirmed the value of the anticipatory, target-driven surveillance strategy that had been agreed upon preoperatively. The overall perioperative course, from presentation through preoperative optimization, induction and regional blockade, the five-hour endoscopic resection, and intensive care surveillance to recovery, is represented as an integrated management algorithm in Figure 3.

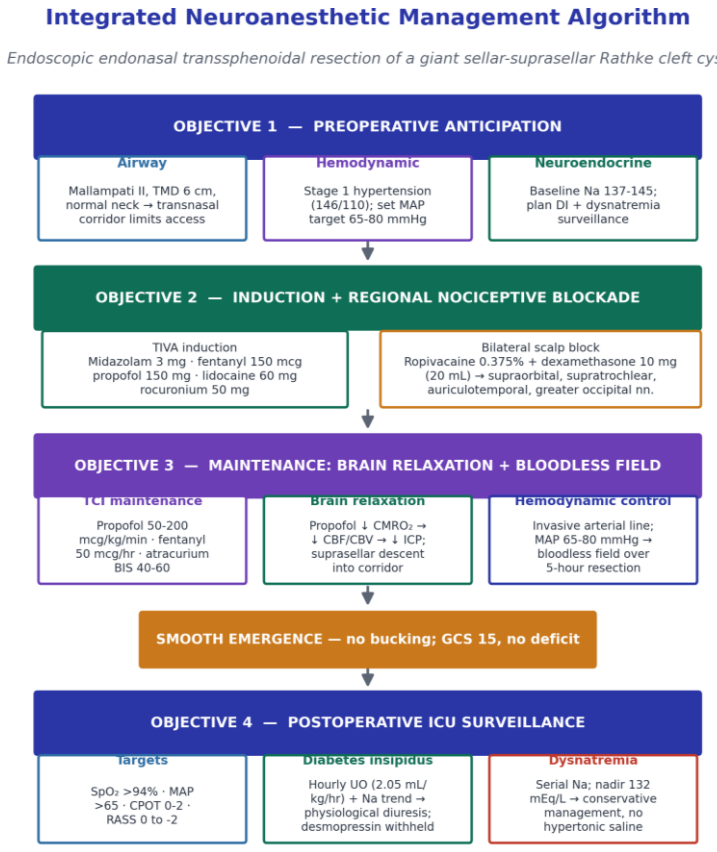

Figure 3. Integrated neuroanesthetic management algorithm for endoscopic transsphenoidal resection of a giant sellar-suprasellar mass, organized around four sequential objectives: preoperative anticipation of airway and neuroendocrine risk; suppression of trigeminal nociception with a dexamethasone-adjuvanted scalp block at induction; simultaneous brain relaxation and bloodless-field control through target-controlled total intravenous anesthesia with tight arterial pressure regulation; and structured postoperative intensive care surveillance for diabetes insipidus and dysnatremia. TIVA, total intravenous anesthesia; MAP, mean arterial pressure; BIS, bispectral index; DI, diabetes insipidus; ICU, intensive care unit.

3. Discussion

The comprehensive management of a giant suprasellar lesion demands an integrated understanding of the pathophysiology of the cyst, the physiology of the surgical corridor, and the pharmacology of the anesthetic agents deployed. This case illustrates how a multimodal neuroanesthetic strategy, anchored by a dexamethasone-adjuvanted scalp block and target-controlled total intravenous anesthesia, can reconcile the competing demands of brain relaxation, a bloodless field, hemodynamic stability, and rapid neurological emergence, and how a structured postoperative surveillance protocol can secure a safe course through the neuroendocrine hazards that characterize transsphenoidal surgery. The discussion that follows situates each element of the management within the contemporary evidence and derives a set of transferable principles.

Pathophysiology of the Rathke cleft cyst and the origin of the presenting syndrome

Rathke cleft cysts are non-neoplastic, benign lesions that originate from the embryological remnants of the craniopharyngeal duct. During early embryogenesis an upward invagination of the oral ectoderm forms Rathke's pouch, which normally pinches off to form the anterior pituitary gland, and failure of the cleft to obliterate completely leaves a potential space that can accumulate mucinous or serous fluid lined by a single layer of cuboidal or columnar ciliated epithelium.[1] As demonstrated in this case, continuous fluid accumulation drives gradual cystic expansion, and the fluid within such cysts frequently contains high concentrations of mucopolysaccharides, cholesterol crystals, and cellular debris. Microscopic leakage of this material into the surrounding sellar microenvironment can initiate a localized inflammatory response, and squamous metaplasia and the nuclear translocation of beta-catenin within the cyst epithelium have been associated with a more aggressive phenotype and a higher risk of recurrence, providing a molecular link between the inflammatory milieu and the biological behavior of the lesion.[1,6]

The pathophysiology of the visual field defect observed in this patient is directly attributable to the anatomical relationship between the sella turcica and the visual pathways. As the cyst expands superiorly out of the sella and into the suprasellar cistern, it encounters the optic chiasm, which contains the decussating fibers arising from the nasal retinae of both eyes and responsible for transmitting information from the temporal visual fields. Mechanical compression and the resultant localized microvascular ischemia of these crossing fibers manifest clinically as bitemporal hemianopsia, the hallmark that brought this patient to attention.[1] Long-term cohorts of Rathke cleft cysts confirm that visual compromise and headache are among the most frequent indications for surgery and that timely decompression is associated with meaningful recovery of visual function, a consideration that lends urgency to the operative and anesthetic planning.[2,3] The pre-existing differential of a sellar abscess, entertained on imaging in this case, further underscores the importance of preparedness for an inflammatory or infective intraoperative finding, since pituitary abscesses share radiological features with cystic lesions yet demand distinct antimicrobial and surveillance strategies.[5]

The decision to proceed to surgery, and thereby to expose the patient to the perioperative hazards described in this report, rests upon a careful weighing of the natural history of the lesion against the risks of intervention. Not every Rathke cleft cyst warrants resection, and the accumulated experience of large cohorts has clarified which features justify operation. A twenty-year retrospective cohort of pediatric Rathke cleft cysts, for example, established that surgical intervention is principally indicated for progressive visual deterioration, symptomatic mass effect, and endocrine compromise, rather than for the incidental discovery of a small and asymptomatic cyst, and it emphasized that the goals of surgery are decompression of the visual apparatus and preservation of pituitary function rather than the aggressive pursuit of complete cyst wall excision.[23] The same principles govern adult practice, where the balance between radicality and the avoidance of recurrence on the one hand, and the preservation of the fragile normal pituitary tissue on the other, defines the surgical strategy and, in turn, the anesthetic demands of the procedure.[2,4] In the

present patient, the severe and progressive bitemporal visual loss, together with the demonstrable compression of the optic chiasm by a giant cyst, provided an unambiguous indication for decompression, and the anesthetic plan was constructed to serve that surgical objective while protecting the neuroendocrine axis whose disturbance defines the postoperative course.

The endoscopic transsphenoidal corridor and its anesthetic implications

The excision of sellar and suprasellar masses through the endoscopic endonasal transsphenoidal approach has revolutionized skull base surgery by exploiting the natural anatomical corridor of the nasal cavity and sphenoid sinus, thereby avoiding brain retraction and reducing the incidence of postoperative cerebral edema, cognitive deficit, and prolonged hospitalization.[7,8] Comparative series demonstrate that the endoscopic route achieves high rates of cyst evacuation and symptomatic relief with acceptable recurrence, and that the choice between endoscopic, endoscopic-assisted, and exoscopic variants influences radicality and the preservation of pituitary function.[4,7,9] Multicenter data further show that quality-of-life outcomes after endoscopic transsphenoidal resection of Rathke cleft cysts are generally favorable, provided that pituitary function and the skull base repair are protected.[2] From the anesthetic perspective, however, the very features that make the corridor attractive to the surgeon impose specific physiological demands. The insertion of rigid endoscopes, the mechanical fracture of the nasal turbinates, and the high-speed osseous drilling of the sphenoid rostrum generate intense, localized nociceptive signals transmitted through the maxillary and ophthalmic divisions of the trigeminal nerve, and the narrowness of the corridor means that even minimal venous oozing can obscure the endoscopic lens and halt the operation.

These two features, an intense trigeminal nociceptive input and an absolute intolerance of bleeding, define the central anesthetic problem of endoscopic transsphenoidal surgery. Uncontrolled nociception provokes sympathetic surges that raise arterial pressure and mucosal bleeding, degrade the surgical view, and increase the risk of hemorrhage and of disruption of the skull base repair. The anesthetic strategy must therefore suppress the nociceptive input at its source while maintaining a mean arterial pressure low enough to preserve a bloodless field yet high enough to protect cerebral perfusion, and it must accomplish this without sacrificing the rapid emergence required for immediate neurological assessment. In this case these objectives were met by combining a regional scalp block, which intercepted the afferent nociceptive barrage, with target-controlled total intravenous anesthesia, which delivered titratable hypnosis, analgesia, and brain relaxation, and with invasive arterial monitoring, which permitted the mean arterial pressure to be held continuously within the narrow window of 65 to 80 mmHg.

The scalp block as multimodal analgesia in transsphenoidal surgery

The scalp block intercepts the terminal branches of the trigeminal and cervical plexus nerves that innervate the scalp and, in part, the deep structures of the anterior cranial base, and although it was developed for open craniotomy its physiological rationale extends naturally to procedures in which the trigeminal territory is stimulated. A substantial and growing body of randomized evidence documents that scalp blockade reduces postoperative pain and opioid consumption and attenuates the hemodynamic response to noxious surgical stimulation in neurosurgical patients.[12,13,14] A randomized comparison of scalp block, local wound infiltration, and systemic analgesia found that regional blockade provided superior post-craniotomy pain relief, and a pediatric randomized trial similarly demonstrated improved analgesia and reduced perioperative opioid requirement with scalp nerve block.[12,13] Opioid-sparing multimodal regimens that incorporate regional blockade have been shown in placebo-controlled trials to reduce post-craniotomy pain, and related regional techniques such as the sphenopalatine ganglion block, which targets nociceptive afferents of direct relevance to the transnasal corridor, have likewise been validated in randomized trials of craniotomy pain.[14,15] Adjunctive non-pharmacological measures have even been combined with scalp blockade in randomized studies

of tumor resection, reflecting the broad interest in maximizing the analgesic yield of regional techniques.[24]

In this patient the scalp block served three simultaneous purposes. First, it blunted the sympathetic surge associated with cranial fixation and with the initial phases of septal dissection, contributing materially to the hemodynamic stability that preserved the surgical view. Second, it reduced the intraoperative requirement for systemic opioid and hypnotic agents, thereby supporting the rapid emergence that permitted immediate neurological assessment. Third, by providing sustained postoperative analgesia it diminished the likelihood of pain-driven hypertension and agitation in the early recovery period, a consideration of particular importance when a fragile skull base repair must be protected from surges in intracranial pressure. The convergence of these benefits illustrates why regional blockade has become an increasingly attractive component of the neuroanesthetic armamentarium, and why its extension from open craniotomy to endoscopic skull base surgery is both logical and clinically valuable.

The technical conduct of the block merits comment, because its efficacy depends upon the accurate deposition of local anesthetic around the correct nerves. The four paired nerves targeted in this case, namely the supraorbital and supratrochlear nerves anteriorly, the auriculotemporal nerve laterally, and the greater occipital nerve posteriorly, together provide the sensory innervation of the frontal, temporal, and occipital scalp and contribute to the afferent pathways stimulated during cranial fixation, and their reliable blockade requires an appreciation of their emergence points and their relationship to palpable bony and vascular landmarks. The use of ultrasound guidance, where available, permits direct visualization of the target and of the spread of the injectate, reduces the risk of intravascular injection given the proximity of the superficial temporal and occipital arteries, and may improve the consistency of the block, and randomized comparisons of adjuvants for scalp block have increasingly been conducted under image guidance for precisely these reasons.[16,24] The volume and concentration selected in this case, twenty milliliters of 0.375% ropivacaine, delivered a total dose of 75 mg, which for a 70 kg patient corresponds to approximately 1.1 mg/kg and lies comfortably below the generally accepted threshold for ropivacaine systemic toxicity, so that the block provided an adequate mass of local anesthetic to achieve dense blockade of multiple nerves while preserving a wide margin of safety against local anesthetic systemic toxicity, a margin that becomes especially important when the block is combined, as it was here, with the systemic agents of a total intravenous technique.

Dexamethasone as a perineural adjuvant: mechanism and evidence

The addition of 10 mg of preservative-free dexamethasone to the ropivacaine mixture was a deliberate pharmacological choice with a dual rationale. Pharmacologically, perineural dexamethasone prolongs the duration of sensory blockade, an effect attributed to local vasoconstriction that slows the systemic absorption of the local anesthetic and to a direct modulation of potassium channel activity on nociceptive fibers that reduces their excitability. A randomized controlled trial in awake craniotomy directly compared dexmedetomidine and dexamethasone as adjuvants to ropivacaine for scalp nerve block and confirmed the clinical utility of dexamethasone in this precise setting, and randomized trials across a range of other regional techniques consistently demonstrate that perineural dexamethasone extends analgesic duration when combined with ropivacaine.[16,17,18] A randomized trial employing a deliberately low dose of perineural dexamethasone likewise showed prolongation of blockade, indicating that the adjuvant effect is achievable without recourse to high doses.[25]

Systemically, the fraction of dexamethasone absorbed from the perineural site exerts a potent prophylactic antiemetic effect, a benefit of considerable importance in neurosurgery, where postoperative nausea and vomiting can precipitate dangerous rises in intracranial pressure and threaten the integrity of a delicate dural or skull base repair. The choice of dexamethasone therefore addressed two distinct perioperative hazards with a single agent, simultaneously prolonging the sensory blockade that

stabilized the intraoperative course and reducing the emetic risk that could have jeopardized the postoperative repair. This economy of purpose exemplifies the rational construction of a multimodal regimen, in which each component is selected to serve more than one objective and in which the interactions between components are anticipated rather than incidental. It should be acknowledged, however, that the magnitude and duration of the adjuvant effect vary with dose, injection site, and the specific block performed, and that the evidence base, though consistent in direction, derives predominantly from regional techniques other than the scalp block, so that the extrapolation to transsphenoidal surgery, while reasonable, remains to be confirmed by dedicated trials.[17,18,25]

Total intravenous anesthesia, brain relaxation, and hemodynamic control

The selection of propofol as the primary hypnotic for the maintenance of anesthesia was grounded in neurophysiological principle. Propofol acts principally by potentiating the inhibitory neurotransmitter gamma-aminobutyric acid at the gamma-aminobutyric acid type A receptor complex, and it produces a dose-dependent reduction in the cerebral metabolic rate for oxygen. Through preserved flow-metabolism coupling this metabolic suppression yields a proportional decrease in cerebral blood flow and cerebral blood volume, and hence a reduction in intracranial pressure and a state of brain relaxation. Randomized comparisons of total intravenous anesthesia with volatile-based techniques in patients undergoing craniotomy for supratentorial tumors have examined precisely this property, and total intravenous anesthesia has been associated with favorable brain relaxation and intracranial pressure profiles in this setting.[10,11] In the present case, the pharmacological brain relaxation afforded by propofol was instrumental in facilitating the descent of the suprasellar cystic component into the sphenoid surgical corridor once the inferior sellar floor had been opened, thereby permitting complete macroscopic resection without hazardous blind dissection upward toward the hypothalamus.

The maintenance of hemodynamic stability was the second pillar of the intraoperative strategy, and it was pursued through the coordinated use of target-controlled infusions and continuous invasive arterial monitoring. The surgical corridor of endoscopic transsphenoidal surgery is exceptionally narrow, and even minimal venous oozing from the highly vascular nasal mucosa can obscure the camera lens and interrupt the operation, so that a mean arterial pressure titrated carefully between 65 and 80 mmHg was necessary to secure a pristine field while safeguarding cerebral perfusion pressure. The controlled reduction of arterial pressure within this window, achieved by titration of the propofol and fentanyl infusions rather than by the reactive administration of vasoactive boluses, produced a stable and predictable hemodynamic course, and it did so without compromising the rapid emergence that followed the discontinuation of the infusions. The absolute immobility of the surgical field, essential during the most delicate phases of capsular dissection near the cavernous sinus and the internal carotid arteries, was guaranteed by the continuous infusion of atracurium titrated to neuromuscular monitoring, which eliminated the risk of diaphragmatic bucking or sudden movement. The coordinated deployment of these techniques exemplifies the manner in which total intravenous anesthesia, regional blockade, and invasive monitoring can be combined to satisfy the exacting and partly contradictory demands of endoscopic skull base surgery.

The quality of emergence deserves particular emphasis, because it is at the transition from anesthesia to wakefulness that many of the gains of a stable intraoperative course can be lost. In endoscopic transsphenoidal surgery the newly reconstructed skull base, sealed in this case with a vascularized nasoseptal flap, is vulnerable to any sudden rise in intracranial pressure, and coughing, straining, or bucking during emergence can generate precisely such rises and threaten a cerebrospinal fluid leak. The pharmacokinetic profile of a propofol-based total intravenous technique, with its rapid and predictable offset, is well suited to a controlled emergence, and the residual analgesia provided by the scalp block reduces the nociceptive drive that would otherwise provoke coughing and hypertension as the systemic agents are withdrawn. In this patient the combination

produced an emergence that was smooth and free of bucking, allowing the endotracheal tube to be removed without a surge in intracranial pressure and permitting an immediate and reliable neurological examination that confirmed the preservation of consciousness and the absence of any new deficit. The value of a calm emergence extends into the early recovery period, where the sustained analgesia and antiemetic prophylaxis of the regional block continue to guard against the pain-driven hypertension and the vomiting that are among the most common precipitants of postoperative complications after skull base surgery.[14,16]

Neuroendocrine surveillance: diabetes insipidus and dysnatremia

The postoperative phase of transsphenoidal surgery is dominated by the risk of neuroendocrine derangement, and the intensive care management of this patient was organized explicitly around that risk. Mechanical manipulation, thermal injury from electrocautery, or localized edema surrounding the pituitary stalk can disrupt the axonal transport of antidiuretic hormone from its sites of synthesis in the supraoptic and paraventricular nuclei of the hypothalamus to its storage site in the posterior pituitary gland, producing an acute deficiency of vasopressin that manifests clinically as central diabetes insipidus, with the excretion of copious dilute urine, rapid intravascular volume depletion, and secondary hypernatremia.[19,20] Contemporary studies have sought to identify the predictors of this complication, and both the degree of pituitary stalk stretch and the geometry of the resection cavity have been shown to correlate with the risk of postoperative diabetes insipidus, while multicenter work has produced structured scoring systems, such as the SALT score, to stratify that risk.[19,20] In this patient the high urine output of 2.35 mL/kg/hr triggered the diabetes insipidus surveillance protocol, but the concurrent finding of a preserved and indeed mildly reduced serum sodium, rather than the rising sodium that characterizes true vasopressin deficiency, indicated that the diuresis was physiological, and exogenous desmopressin was therefore appropriately withheld.

The mirror image of diabetes insipidus is postoperative hyponatremia, which may arise through the syndrome of inappropriate antidiuresis or through cerebral salt wasting and which, in its delayed form, is a recognized cause of readmission after transsphenoidal surgery. Recent studies have characterized both the early and the delayed patterns of hyponatremia after endoscopic transsphenoidal resection and have developed predictive models to identify patients at risk, underscoring the value of serial sodium monitoring in the postoperative period.[21,22] The mild dilutional hyponatremia of 132 mEq/L observed in this patient fell within the range that can be managed conservatively through careful fluid balance, and it resolved without the need for hypertonic saline. The juxtaposition of a brisk urine output with a mildly low serum sodium in the same patient illustrates the delicate and sometimes paradoxical fluid and electrolyte physiology that follows pituitary surgery, and it demonstrates why the interpretation of any single variable, whether urine output or serum sodium, must always be undertaken in the context of the others.[19,21] The structured protocol of hourly fluid balance, serial sodium measurement, and explicit hemodynamic and sedation targets, summarized in Table 6, provided exactly this integrated context and enabled the team to distinguish physiological postoperative diuresis from evolving diabetes insipidus and to manage the mild hyponatremia without overcorrection.

A further conceptual point clarifies why the equivocal high-urine-output state after pituitary surgery is so treacherous and why its interpretation demands the integration of several variables. Damage to the pituitary stalk may produce not a single, stable pattern of vasopressin deficiency but a classical triphasic response, in which an initial phase of diabetes insipidus, caused by axonal shock and the cessation of vasopressin release, is followed by a transient phase of inappropriate antidiuresis, caused by the uncontrolled release of stored hormone from degenerating neurons, and then by a final, sometimes permanent, phase of diabetes insipidus once the hormone stores are exhausted. Each phase carries its own electrolyte signature, hypernatremia during the diabetes insipidus phases and hyponatremia during

the interposed antidiuretic phase, and a management decision that is correct in one phase may be dangerous in the next, so that the reflexive administration of desmopressin in response to a high urine output risks precipitating profound hyponatremia if the patient has entered the antidiuretic phase. In this patient the coexistence of a brisk urine output with a mildly reduced rather than elevated serum sodium was reassuring against established vasopressin deficiency and consistent with a physiological diuresis or an early antidiuretic tendency, and the conservative decision to withhold desmopressin and to manage by careful fluid balance and serial sodium measurement was the safest response to an inherently ambiguous picture.[19,20,21] This reasoning illustrates the general principle that in the postoperative neuroendocrine surveillance of the transsphenoidal patient no variable should be interpreted in isolation and that the pattern, rather than any single value, must guide therapy.

An integrated framework for the neuroanesthesia of sellar tumor resection

The management of this case suggests a structured framework for the anesthetic conduct of endoscopic transsphenoidal surgery that can be organized around four sequential objectives. The first objective, established preoperatively, is the anticipation of both the airway and the neuroendocrine consequences of the lesion, since the transnasal corridor restricts intraoperative airway access and since the risk of diabetes insipidus and dysnatremia must be planned for before rather than after it manifests.[19,22] The second objective, achieved at induction, is the suppression of the trigeminal nociceptive input by means of a regional scalp block, adjuvanted with dexamethasone to prolong its effect and to provide antiemetic prophylaxis, thereby stabilizing the hemodynamic environment from the outset.[13,16] The third objective, sustained through maintenance, is the simultaneous provision of brain relaxation and a bloodless field by means of target-controlled total intravenous anesthesia and tightly regulated arterial pressure, delivered without prejudice to a rapid emergence.[10,11] The fourth objective, pursued postoperatively, is the structured surveillance of neuroendocrine and hemodynamic status in an intensive care setting, with explicit targets and serial measurement, so that derangements are detected early and managed conservatively wherever possible.[19,21]

The value of articulating this framework lies in its capacity to convert a set of individually familiar techniques into a coherent and reproducible strategy. Each element of the plan, considered in isolation, is well supported by evidence, but it is their integration, and in particular the manner in which the scalp block, the total intravenous anesthesia, and the invasive monitoring reinforce one another, that produces the smooth and stable course observed in this patient. The framework also clarifies where the principal uncertainties lie, since the extension of the dexamethasone-adjuvanted scalp block from open craniotomy to transsphenoidal surgery, though physiologically reasoned and supported by adjacent evidence, has not yet been subjected to dedicated randomized evaluation, and the optimal arterial pressure target and the optimal management of the equivocal high-urine-output state remain matters of clinical judgement informed by, but not fully resolved by, the current literature.[15,20]

Limitations

This report is subject to the limitations inherent in the description of a single patient. As a case report it can establish feasibility and generate hypotheses, but it cannot demonstrate that the described strategy is superior to alternatives, nor can it quantify the magnitude of the benefit attributable to any individual component of the multimodal regimen. The favorable course observed here should not be interpreted as proof that a dexamethasone-adjuvanted scalp block is necessary or sufficient for a stable anesthetic in transsphenoidal surgery, since the same patient received target-controlled total intravenous anesthesia, invasive hemodynamic monitoring, and structured postoperative surveillance, and the contributions of these elements cannot be disentangled within a single case. The diagnosis of a Rathke cleft cyst, favored on imaging and consistent with the operative findings, rests ultimately on histopathological confirmation, and the differential of a sellar abscess entertained preoperatively is a reminder that the anesthetic implications of an

inflammatory lesion may differ. Several specific data were not captured in a manner that would strengthen the report: the analgesic efficacy of the scalp block was not quantified objectively, for example through a documented attenuation of the pressor response to cranial pinning or a measured reduction in intraoperative opioid requirement against a comparator, so that its contribution is inferred from the stable course rather than demonstrated directly; a formal preoperative anterior pituitary hormone panel, which would ideally accompany the assessment of any sellar mass, is not reported here; and the absence of long-term follow-up on visual recovery, pituitary function, and cyst recurrence limits the conclusions that can be drawn about the ultimate outcome. These limitations notwithstanding, the structured framework described here, organized around anticipation, regional analgesia, titratable brain relaxation, and neuroendocrine surveillance, offers a rational and transferable approach that can be adapted to the circumstances of comparable patients.

4. Conclusion

The surgical management of a giant sellar and suprasellar Rathke cleft cyst is a formidable undertaking that demands precise and seamless collaboration between the surgical and anesthetic teams. This case demonstrates that the integration of a fully endoscopic endonasal transsphenoidal approach with a carefully constructed multimodal neuroanesthetic strategy yields a favorable outcome across every phase of care. The dexamethasone-adjuvanted regional scalp block attenuated the sympathetic responses provoked by cranial instrumentation and septal dissection, contributing to immaculate intraoperative hemodynamic stability and a bloodless surgical corridor, while the target-controlled total intravenous anesthesia delivered the brain relaxation necessary for surgical exposure together with a rapid and smooth emergence that permitted immediate neurological assessment. The structured intensive care surveillance for diabetes insipidus and dysnatremia, organized around explicit targets and serial measurement, enabled the team to distinguish a physiological postoperative diuresis from evolving vasopressin deficiency and to manage a mild dilutional hyponatremia conservatively, without recourse to desmopressin or hypertonic saline. Tailoring advanced pharmacological and regional anesthetic techniques to the specific physiological demands of endoscopic skull base surgery is therefore paramount for minimizing morbidity, preserving neurological function, and accelerating recovery, and the integrated, anticipatory framework commended here, rather than any single technique in isolation, is what ultimately safeguards the patient undergoing resection of a complex sellar tumor.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent for the anesthetic and surgical procedures and for the anonymized publication of clinical details was obtained from the patient. No identifying patient information is included in this report.

Consent for publication

Written informed consent for the publication of this anonymized case report and any accompanying data was obtained from the patient.

Availability of data and materials

The clinical data supporting the findings of this report are contained within the article. Further anonymized details are available from the corresponding author on reasonable request.

Conflict of interests

The authors declare that they have no competing interests.

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Authors' contributions

All authors contributed to the perioperative management of the patient, the acquisition and interpretation of the clinical data, and the drafting and critical revision of the manuscript. All authors have reviewed and approved the final version of the manuscript.

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