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General Anesthesia and Scalp Block for Craniotomy due to Cerebral Abscess in Uncorrected Tetralogy of Fallot Patient: A Case Report and Literature Review

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Abstract

Introduction: Neurosurgery in Tetralogy of Fallot (ToF) poses complex anesthetic challenges due to risks of hypoxemia and hemodynamic instability. The aim of this case report was to describe the general anesthesia and scalp block management of a boy with uncorrected ToF who developed a cerebral abscess requiring craniotomy for abscess drainage.
Case: An 8-year-old boy with uncorrected ToF presented with headache, fever, and neurological deficits. Neuroimaging revealed a large frontal abscess necessitating urgent surgical intervention. Preoperative evaluation showed central cyanosis, digital clubbing, and oxygen saturation of 70% on room air, with echocardiography demonstrating a significant right-to-left shunt. The primary anesthetic objectives were to maintain systemic vascular resistance (SVR) and prevent increases in pulmonary vascular resistance (PVR). Anesthesia induction was achieved with ketamine 1 mg/kg and fentanyl 4 µg/kg to preserve SVR, followed by rocuronium 0.5 mg/kg for neuromuscular relaxation. A bilateral scalp block was administered with ropivacaine 0.25% (0.4 mL/kg per site) for regional analgesia. The surgery was completed without complications, and the patient was extubated safely after 24 hours in the pediatric intensive care unit.
Discussion: Anesthetic management of uncorrected ToF during neurosurgery focuses on maintaining SVR and avoiding increases in PVR to prevent worsening right-to-left shunting and hypoxemia. Ketamine-based induction and bilateral scalp block provided hemodynamic stability and effective analgesia, facilitating successful surgical and postoperative outcomes without major complications
Conclusion: Individualized anesthetic management that prioritizes SVR preservation, PVR control, and meticulous hemodynamic monitoring is essential for safe neurosurgical procedures in uncorrected ToF.

Keywords: Tetralogy of Fallot, cerebral abscess, craniotomy, pediatric anesthesia, neuroanesthesia

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Introduction

Cerebral abscesses are a recognized and potentially fatal complication in patients with cyanotic congenital heart disease, particularly in those with uncorrected Tetralogy of Fallot (ToF).[1] The incidence of brain abscess in uncorrected TOF ranges from 13% to 70%, with the primary pathophysiological mechanism being right-to-left intracardiac shunting.[2] This shunt bypasses the pulmonary capillary filtration system, facilitating hematogenous spread of bacteria to the cerebral circulation.[3] Additionally, chronic hypoxemia and resultant polycythemia increase blood viscosity, further compounding the risk of septic emboli lodging in cerebral vasculature.[4] Although uncommon, brain abscess remains a substantial cause of neurological morbidity and mortality, particularly among children and young adults in regions where timely corrective

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cardiac surgery is not universally available.[5,6] In patients with ToF, the most frequently implicated microorganisms are non-hemolytic bacteria, particularly Streptococcus milleri, which has a propensity to form localized abscesses.[2]

Anesthetic management of uncorrected ToF during non-cardiac surgery, such as craniotomy, presents unique challenges.[7] The pathophysiological right-to-left shunt is highly dynamic and sensitive to fluctuations in systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), preload, and oxygenation status.[8] Anesthetic management must therefore focus on maintaining normovolemia, preventing hypoxemia, and avoiding decreases in SVR or increases in PVR, as these changes can exacerbate right-to-left shunting and precipitate profound cyanosis or cardiovascular instability.[9] Non-cardiac procedures, particularly those requiring major neurosurgical intervention, carry an increased perioperative mortality in this patient population, largely attributable to the hemodynamic consequences of cyanosis and the narrow margin for physiologic compensation.

Despite the recognized risk, published literature on the neuroanesthetic management of uncorrected ToF undergoing craniotomy for cerebral abscess remains scarce.[4,5,7–15] Previous studies have described the use of either general anesthesia or scalp block alone in similar cases, yet details regarding intraoperative hemodynamic targets, anesthetic drug selection, and perioperative monitoring remain inconsistent and largely anecdotal.[4,5,7–15] In contrast, the present case report combines both general anesthesia and scalp block to optimize hemodynamic stability, analgesia, and cerebral protection during surgery. Therefore, the aim of this case report was to describe the perioperative neuroanesthetic management of an 8-year-old boy with uncorrected ToF who developed a cerebral abscess requiring craniotomy for abscess drainage. This case report was developed in accordance with the CAse REport (CARE) guideline and follows the consensus-based guidelines for anesthesia case reports as outlined by the Anesthesia Case Report (ACRE) recommendations.[16,17]

Case

Anamnesis/disease history

A 8-year-old Southeast Asian boy with a known history of uncorrected ToF was admitted to the Neurosurgical Department of Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia, with a diagnosis of cerebral abscess requiring urgent craniotomy and drainage. The child had never undergone corrective cardiac surgery and was not receiving chronic medication. Two days before admission the patient experienced a progressively worsening, pressure-like headache accompanied by nausea and non-projectile vomiting of food contents. No seizures or focal neurological deficits were reported. According to the patient's mother, the patient had recurrent cyanotic spells precipitated by exertion that resolved spontaneously when the patient performed a squatting position. There was no history of recent upper respiratory infection or other intercurrent illness.

Physical Examination

On admission the patient appeared alert but clearly cyanotic. Physical examination revealed an underweight child—height 121 cm, body weight 19 kg, body mass index 13 kg/m²—with vital signs showing a temperature of 36.5 °C, heart rate of 101 beats/min, respiratory rate of 26 breaths/min, blood pressure of 100/60 mmHg, and oxygen saturation of 80% while breathing room air. Head and neck evaluation demonstrated no meningeal signs; the pupils were equal and reactive to light and there was no lymphadenopathy. Airway assessment revealed adequate mouth opening and a Mallampati class I view; no loose teeth or other predictors of difficult intubation were evident. Respiratory examination found symmetrical chest expansion with vesicular breath sounds bilaterally and no rales or wheezing.

Cardiovascular examination showed central cyanosis of the lips and nail beds and a grade III/VI continuous murmur best heard at the left fourth intercostal space along the mid-clavicular line, with the first heart sound louder than the second; no gallop or pericardial rub was detected and peripheral pulses were strong and symmetrical. Abdominal and genitourinary evaluation

revealed a soft, non-tender abdomen with normal bowel sounds and no hepatosplenomegaly or genitourinary abnormalities. Neurological assessment demonstrated a Glasgow Coma Scale of E4V5M6, intact cranial nerves, and no focal motor deficits; the patient reported mild headache but showed no neck stiffness or overt signs of raised intracranial pressure other than the preceding episodes of vomiting.

Laboratory Examination

Laboratory investigations demonstrated elevated secondary erythrocytosis, with hemoglobin 21.3 g/dL and hematocrit 61%; red cell counts was 6.8 × 10^3/mm^3 (Table 1). Platelet count was 220 × 10^3/mm^3, white cell count 10.27 × 10^3/mm^3, and serum electrolytes were within normal limits (sodium 141 mmol/L, potassium 4.6 mmol/L, chloride 103 mmol/L). Serum albumin was 4.2 g/dL. (Table 1)

Supporting Examination

Electrocardiography revealed sinus rhythm with right axis deviation. Chest radiography showed normal cardiac silhouette and aortic contour, with no pulmonary or hilar abnormalities. Contrast-enhanced brain CT scan demonstrating

Table 1. Preoperative Laboratory, Radiological, and Cardiac Evaluation Findings of the Present Patient

Parameter	Preoperative
Hematology	
Hemoglobin (g/dL)	21.3
Hematocrit (%)	61
Red blood cell count (×10^3/mm^3)	6.8
White blood cell count (×10^3/mm^3)	10.27
Platelet count (×10^3/mm^3)	220
Serum biochemistry	
Sodium (mmol/L)	141
Potassium (mmol/L)	4.6
Chloride (mmol/L)	103
Albumin (g/dL)	4.2
Electrocardiography	Sinus rhythm with right axis deviation
Chest radiography	Normal cardiac silhouette and aortic contour; no pulmonary or hilar abnormalities
Non-contrast brain CT-scan	Multiple hypodense lesions within the right parietal lobe, the largest measuring 4.9 × 3.7 × 4.7 cm, accompanied by prominent surrounding vasogenic edema and features of meningoencephalitis.
Contrast-enhanced brain CT-scan	Multiple ring-enhancing lesions in the right parietal lobe with peripheral enhancement and central hypodensity, consistent with multiple cerebral abscesses. Extensive perifocal edema and associated meningoencephalitis are also evident.
Transthoracic echocardiography	Situs solitus with atrioventricular concordance and ventriculo-arterial discordance; right ventricular hypertrophy; moderate pulmonary stenosis; 9 mm ventricular septal defect with right-to-left shunt; overriding aorta (~50%); vegetation near aortic valve measuring 14 × 11 mm; normal left ventricular systolic and diastolic function
ASA physical status	IV

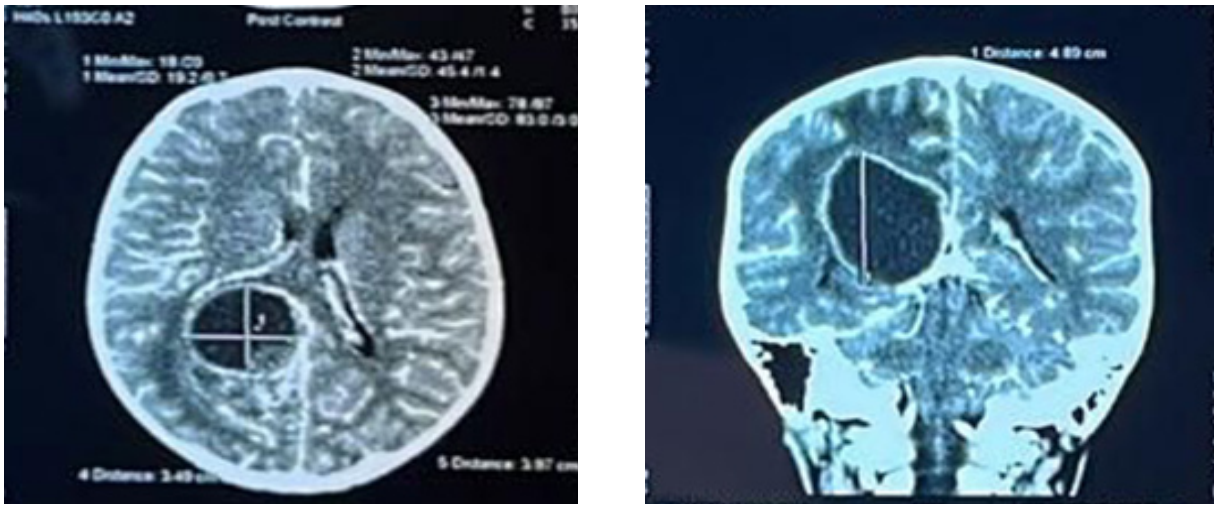

Figure 1. Contrast-Enhanced Brain CT Demonstrating Multiple Ring-Enhancing Abscesses in the Right Parietal Lobe with Extensive Perifocal Edema and Associated Meningoencephalitis.

multiple ring-enhancing lesions in the right parietal lobe with peripheral enhancement and central hypodensity, consistent with multiple cerebral abscesses (Figure 1). Transthoracic echocardiography performed revealed situs solitus with atrioventricular concordance and ventriculo-arterial discordance, right ventricular hypertrophy, moderate pulmonary stenosis, a 9 mm ventricular septal defect with right-to-left shunt, and an overriding aorta of approximately 50%. Notably, a vegetation measuring 14 × 11 mm was visualized near the aortic valve. Left ventricular systolic and diastolic function remained normal. Based on these findings, the patient was classified as American Society of Anesthesiologists (ASA) physical status IV

Anesthesia Management

Following multidisciplinary discussion between the neurosurgical, pediatric cardiology, and anesthesia teams, a tailored neuroanesthetic plan was developed to address the conflicting goals of maintaining stable cerebral perfusion while avoiding any increase in right-to-left shunting. On the day of surgery the patient was transferred to the operating room and positioned supine. Standard ASA monitoring was instituted, including electrocardiography, pulse oximetry, capnography, and invasive arterial blood pressure monitoring via a radial arterial line. Two peripheral intravenous catheters (22-gauge) were placed in the left hand and right foot, and a central venous catheter was inserted in the left femoral vein under ultrasound guidance. Preoxygenation with 100% oxygen for three minutes was performed. Premedication consisted of fentanyl 4 µg/kg and midazolam 0.05 mg/kg intravenously. Anesthetic induction was achieved with ketamine 1 mg/kg intravenously to preserve SVR, followed by rocuronium 0.5 mg/kg to facilitate intubation. The airway was secured with a 5.5 mm cuffed endotracheal tube. Mechanical ventilation was initiated with a tidal volume of 170 mL, respiratory rate of 16 breaths/min, positive end-expiratory pressure of 4 cm H_2O, and an inspired oxygen fraction of 50%, using a total fresh-gas flow of 2 L/min with an oxygen-to-air ratio of 2:1. Target parameters included a pulse oximetry saturation of approximately 87%—representing the patient's baseline—and an end-tidal carbon dioxide of 31 mmHg.

To provide regional analgesia and minimize hemodynamic fluctuation, a bilateral scalp block was performed after induction using ropivacaine 0.25% (total volume adjusted to 0.4 mL/kg per injection site). Maintenance of anesthesia was achieved with sevoflurane supplemented by intermittent fentanyl boluses. Tranexamic acid 10 mg/kg intravenously was administered as antifibrinolytic prophylaxis, while ondansetron 0.1 mg/kg and dexamethasone 0.1 mg/kg were

Table 2. Intraoperative Hemodynamic Monitoring during the 180-Minute Surgical Procedure

Time (min)	HR (beats/min)	SpO_2 (%)	NIBP (mmHg)	MAP (mmHg)	RR (breaths/min)
0	85	97	146/86	103	14
30	68	100	107/72	82	14
60	60	100	99/71	81	15
90	52	92	94/63	73	15
120	56	86	89/61	70	15
150	63	86	94/58	70	15
180	67	86	97/57	70	15

Abbreviations: HR, heart rate; SpO_2, peripheral oxygen saturation; NIBP, noninvasive blood pressure; MAP, mean arterial pressure; RR, respiratory rate

given for postoperative nausea and vomiting prevention. In intraoperative monitor, the patient maintained relatively stable hemodynamics throughout the procedure, with heart rate ranging from 49–71 beats/min, invasive arterial blood pressure ranging from 85–97/64–73 mmHg (mean arterial pressure 73–83 mmHg), respiratory rate consistently 15 breaths/min, and oxygen saturation maintained between 86–88% (Table 2). No episodes of significant hypotension, arrhythmia, cyanotic spells, or hemodynamic instability were observed. Throughout the procedure, careful attention was paid to avoiding hypotension and hypercapnia to maintain cerebral perfusion and minimize intracranial pressure elevation. (Table 2)

Post-surgical Management

Craniotomy and drainage of the right parietal abscess proceeded uneventfully over approximately 180 minutes. Estimated blood loss was 150 mL and was replaced with 400 mL of 0.9% sodium chloride crystalloid solution. Hemodynamic parameters remained stable with no arrhythmias and no desaturation episodes beyond baseline cyanosis. Intraoperative urine output remained within the normal range (0.7 mL/kg/hour). At the end of the procedure neuromuscular blockade was reversed with neostigmine 0.04 mg/kg intravenously combined with atropine 0.02 mg/kg. The patient was transferred to the Pediatric Intensive Care Unit (PICU) while still intubated for elective overnight ventilation. Extubating was successfully performed 24 hours postoperatively after ensuring stable hemodynamic and neurological conditions. Hemodynamic parameters remained stable, with heart rate ranging from 55–68 beats/min and blood pressure maintained between 89–100/57–63 mmHg, corresponding to a mean arterial pressure ≥65 mmHg. Oxygen saturation remained at 86–87%. No episodes of hypotension, arrhythmia, cyanotic spells, respiratory compromise, or neurological deterioration were observed during the postoperative period. The patient was subsequently transferred to the general pediatric ward on postoperative day two for continuation of antibiotic therapy.

Discussion

The anesthetic management of these patients during neurosurgical intervention presents a dual challenge: maintaining optimal cerebral perfusion pressure (CPP) and controlling intracranial pressure (ICP), while simultaneously preserving cardiovascular stability and minimizing intracardiac shunting. The literature uniformly emphasizes the importance of avoiding reductions in SVR, which can exacerbate right-to-left shunting and result in severe hypoxemia.[4,5,7–10,13,14] Ketamine is frequently recommended for induction due to its ability to maintain or elevate SVR and heart rate, making it a hemodynamically favorable agent in TOF patients.[4,8–10]

TOF consists of four major anatomical abnormalities: ventricular septal defect, right ventricular outflow tract obstruction (RVOTO), overriding aorta, and right ventricular hypertrophy, with the severity of RVOTO determining the degree of right-to-left shunting and systemic hypoxemia. Anesthetic management

aims to maintain heart rate within a normal-to-slightly low range while avoiding tachycardia, which may worsen RVOTO, shorten diastolic filling time, increase myocardial oxygen demand, and aggravate right-to-left shunting, as well as profound bradycardia that may reduce cardiac output.[9,14] Therefore, the primary anesthetic goals include maintaining adequate preload and systemic vascular resistance, avoiding increases in pulmonary vascular resistance, preventing excessive sympathetic stimulation, and preserving sinus rhythm and myocardial oxygen balance.[4,7] These objectives were achieved through careful anesthetic titration, adequate analgesia, maintenance of intravascular volume, avoidance of hypoxia and hypercarbia, and continuous hemodynamic monitoring throughout the procedure.

Various anesthetic strategies have been employed in this context, tailored to the clinical status of the patient, available resources, and surgical urgency. Previous studies have reported successful use of regional scalp blocks with sedation in pediatric ToF patients undergoing abscess drainage.[5,9,11,12] These techniques demonstrated minimal cardiovascular disturbance and are especially beneficial in resource-limited settings where invasive monitoring and advanced airway equipment may not be available.[5,11] In such scenarios, the avoidance of general anesthesia and airway manipulation reduces the risk of cyanotic spells and hemodynamic instability.[11,12] However, these regional techniques are often only appropriate for cooperative, stable patients undergoing superficial or minor procedures. In more complex or emergency neurosurgical cases, general anesthesia with controlled ventilation becomes necessary. Previous reports have documented the successful use of general anesthesia in patients with uncorrected Tetralogy of Fallot (ToF) when managed with meticulous monitoring and anesthetic strategies tailored to the underlying pathophysiology.[5,15] These included strategies such as gradual induction, careful titration of anesthetic depth, and meticulous fluid and vasopressor management.[15] The emphasis in these cases is on anticipating hemodynamic changes and proactively countering deleterious shifts in SVR or PVR.[5,15] Few studies have addressed the concurrent use of GA and regional scalp block in uncorrected ToF patients undergoing neurosurgical procedures.[4,7,10,13,14] Our case contributes to this limited but growing body of evidence by demonstrating the feasibility and benefits of a hybrid approach. The scalp block provided excellent analgesia for painful stimuli such as pin fixation and skin incision, reducing the need for volatile anesthetics and systemic opioids, both of which can affect SVR. Meanwhile, GA allowed for airway protection, controlled ventilation, and meticulous anesthetic depth management. This combination enhanced intraoperative stability and may serve as a useful model for similar high-risk patients requiring emergent neurosurgical intervention.

Scalp block was performed as part of a multimodal analgesic approach to attenuate nociceptive stimulation during cranial pin fixation, skin incision, and other painful neurosurgical stimuli.[18] In patients with TOF, adequate suppression of sympathetic responses is particularly important because excessive catecholamine release may increase myocardial oxygen consumption, induce tachycardia, worsen RVOTO, and aggravate right-to-left shunting.[4,8,13] The primary target of the scalp block is the sensory innervation of the scalp, including the supraorbital, supratrochlear, zygomaticotemporal, auriculotemporal, greater occipital, and lesser occipital nerves. Blocking these nerves reduces perioperative pain transmission and blunts hemodynamic responses to surgical stimulation.[18,19] In this patient, scalp block contributed to maintaining hemodynamic stability, reducing perioperative opioid requirements, and minimizing sympathetic stimulation, which are essential anesthetic goals in TOF physiology.

Preoperative optimization focuses on preventing tet spells, which may be triggered by stress, dehydration, pain, or excessive sympathetic stimulation. Hypovolemia must be avoided, as it can aggravate RVOT obstruction, particularly in the presence of infundibular stenosis.[4] Hemodynamic management aims to maintain SVR higher than PVR to minimize right-to-

left shunting.[8] Conditions that increase PVR—hypoxia, hypercarbia, or acidosis—must be strictly prevented.[9] Premedication may include low-dose midazolam and opioids administered slowly to reduce sympathetic activation without causing respiratory depression or significant reduction in cardiac output. Invasive hemodynamic monitoring with an arterial line and central venous catheter should be established early.[10]

Intraoperative anesthetic strategy is directed toward maintaining SVR above PVR.8 Induction is performed slowly using agents that support or increase SVR. Intravenous ketamine 2 mg/kg is preferred because it raises SVR and thereby limits right-to-left shunting, combined with fentanyl 5–10 μg/kg for analgesia and hemodynamic stability. In cases with hemodynamic instability or hypovolemia, etomidate 0.2–0.3 mg/kg provides an alternative with minimal cardiovascular depression. Rocuronium 1 mg/kg is administered to facilitate intubation, with cricoid pressure as part of rapid sequence induction to reduce the risk of aspiration. Any decrease in SVR should be treated promptly with an α-adrenergic agonist such as phenylephrine.[9] Maintenance of anesthesia utilized sevoflurane at 1.5% end-tidal concentration in a 50% inspired oxygen fraction, targeting SpO_2 around 87% (baseline) and end-tidal CO_2 of 31 mmHg. Ventilation is controlled to maintain normocapnia or mild hypocapnia (arterial $PaCO_2$ 30–35 mmHg), which helps lower PVR and simultaneously reduces ICP. Positioning with slight head elevation and administration of mannitol as needed help maintain stable ICP. All intravenous lines must be carefully de-aired to avoid paradoxical air embolism through the ventricular septal defect.

Perioperative hemodynamic stability is crucial. Sudden hypoxemia, hypercarbia, acidosis, or hypotension can precipitate a hypercyanotic (tet) spell.[4] Management includes increasing anesthetic depth, administering 100% oxygen, gentle hyperventilation, restoring intravascular volume, and giving phenylephrine to raise SVR. Sympathetic stimulation during scalp incision or craniotomy should be blunted by scalp block and multimodal analgesia, including intravenous paracetamol and an α_2-agonist such as dexmedetomidine.[10,11] Atropine or esmolol should be available for tachycardia or RVOT spasm.[15] Several intraoperative complications require vigilant prevention and management. Aspiration risk is high because general anesthesia decreases lower esophageal sphincter tone and abolishes airway reflexes, while emergency neurosurgical procedures often proceed without optimal fasting.[12,13] This raises the likelihood of gastric content aspiration, potentially causing chemical pneumonitis.[12,13] Strategies include rapid sequence induction with endotracheal intubation, low positive end-expiratory pressure (PEEP), tidal volumes of 6–8 mL/kg, and maintaining $PaCO_2$ at 30–35 mmHg to limit PVR and preserve cerebral perfusion.[7,14]

Desaturation may result from ventilation–perfusion mismatch, changes in patient position, intraoperative bleeding, or alterations in right-to-left shunt dynamics.[7] Venous air embolism, a recognized neurosurgical risk, can cause abrupt decreases in end-tidal CO_2 and oxygen saturation.[5] Controlled ventilation through an endotracheal tube and continuous monitoring of oxygen saturation and cerebral oximetry are essential.[10] Furthermore, hypothermia is common in pediatric neurosurgery due to large body surface area relative to mass, exposure during craniotomy, cold intravenous fluids, and inhalation of cold anesthetic gases. Inhaled and intravenous anesthetics impair thermoregulatory vasoconstriction, leading to increased blood loss (approximately 22% for every 1°C drop in core temperature) and impaired coagulation. Active warming methods—forced-air warming, warmed intravenous fluids, thermal insulation, and maintaining the operating room temperature ≥23 °C—are critical. Core temperature should be monitored every 15 minutes using esophageal or nasopharyngeal probes.

Hypoxia may develop when oxygen delivery is insufficient to meet tissue demand, exacerbated by right-to-left shunting and polycythemia. Low FiO_2, elevated PVR from hypercarbia or acidosis, or airway obstruction can reduce cerebral perfusion.[13] Preventive strategies

include delivering FiO_2 of 50–100%, maintaining normocapnia or mild hypocapnia, and correcting acidosis promptly.[5] Additionally, cardiac arrest is a potential catastrophic event in children with congenital heart disease and intracranial infection, triggered by hypovolemia, massive hemorrhage, anaphylaxis, air embolism, or acute myocardial dysfunction. Paradoxical embolism is a specific risk in ToF due to intracardiac shunting.[3] Continuous invasive monitoring and immediate availability of vasopressors such as phenylephrine and epinephrine are essential. Advanced Cardiac Life Support (ACLS) protocols must be implemented without delay if cardiac arrest occurs.

Postoperative pain after craniotomy can increase sympathetic activity, elevate SVR, and precipitate a tet spell.[9] Acute pain triggers catecholamine release, raising right ventricular contractility and exacerbating right-to-left shunting.[15] Multimodal analgesia with continuous opioid infusion and intravenous paracetamol is recommended to maintain hemodynamic stability without the renal adverse effects of nonsteroidal anti-inflammatory drugs.[6] Postoperative care requires close monitoring in the intensive care unit. Extubation should be delayed until both hemodynamic and neurological stability are achieved; mechanical ventilation may be continued as necessary.[7] Continuous monitoring of heart rate, blood pressure, oxygen saturation, and respiratory function is mandatory.[13] Adequate analgesia and oxygen supplementation help prevent sympathetic surges and recurrent tet spells.[5] Fluid management must be carefully balanced to maintain adequate cerebral perfusion while avoiding fluid overload.[10] Red blood cell transfusion may be considered to optimize oxygen-carrying capacity despite high baseline hematocrit.[4] Anticonvulsants should be continued to prevent seizures, and corticosteroids administered as indicated to reduce cerebral edema.[20]

This case report has several important limitations. First, as a single-patient observation, the findings cannot be generalized to all pediatric patients with uncorrected ToF undergoing craniotomy for cerebral abscess. Hemodynamic responses to anesthetic drugs and ventilatory strategies vary widely depending on the degree of right ventricular outflow tract obstruction, baseline oxygen saturation, and presence of comorbidities; therefore, the described approach may not be applicable to all clinical scenarios. Second, detailed long-term outcomes, including neurologic recovery and late cardiac events, were not available, limiting assessment of the sustained safety of the chosen anesthetic strategy. Third, the absence of invasive measurements of pulmonary vascular resistance or advanced cardiac imaging during the perioperative period prevented a more precise correlation between hemodynamic targets and clinical outcomes. Finally, because this case occurred in an emergency setting, not all recommended preoperative investigations could be performed, which may have influenced perioperative decision-making.

Future research should move beyond single cases by conducting multicenter prospective studies or case series that evaluate standardized neuroanesthesia protocols for patients with uncorrected ToF. Comparative studies assessing different anesthetic induction agents, intraoperative hemodynamic targets, and ventilatory strategies could clarify the most effective approaches to minimize right-to-left shunting and maintain cerebral perfusion. Additionally, incorporation of advanced monitoring—such as continuous cerebral oximetry, transesophageal echocardiography, and real-time PVR measurement—would provide more robust physiologic data to guide individualized anesthetic management. Long-term follow-up of similar patients is also needed to determine the impact of specific anesthetic techniques on postoperative neurological outcomes, cardiac function, and risk of recurrent cerebral abscess.

Conclusion

Anesthetic management of craniotomy for cerebral abscess drainage in a child with uncorrected ToF presents significant challenges. Optimal preoperative preparation, vigilant intraoperative monitoring, and the use of

anesthetic agents that preserve systemic vascular resistance while avoiding elevations in pulmonary vascular resistance were critical to prevent right-to-left shunt worsening and perioperative hypoxemia. The present case report emphasizes the importance of individualized anesthetic strategies and meticulous perioperative planning for emergency neurosurgical procedures in cyanotic congenital heart disease.

Ethics approval
Written informed consent was obtained from the patient's parent or legal guardian prior to the surgical procedure, including authorization for the intervention as well as approval for the collection, analysis, and dissemination of clinical data in an anonymized manner for research and academic purposes.

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Competing Interests
All the authors declare that there are no conflicts of interest.

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Underlying data
Derived data supporting the findings of this study are available from the corresponding author on request.

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