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SILENT HEARTS, DEADLY BRAINS: BRAIN ABSCESS AS A LIFE-THREATENING COMPLICATION OF UNREPAIRED TETRALOGY OF FALLOT IN A 6-YEAR-OLD CHILD — A CASE REPORT

Cassandra Savira Alisa ¹, Yoga Alfian Noor ², Miftahul Afandi ³,
Mustaqim Apriyansa Rahmadhan ⁴, Hardito Puspo Yugo ⁵, Fransiska
Anggraeni Sihotang ⁶, Meliana Sulistio ⁷

¹ Department of General Medicine, RSUD Aji Muhammad Parikesit,
Tenggarong, Kalimantan Timur, Indonesia

^{2,3,6} Department of Cardiology, RSUD Aji Muhammad Parikesit,
Tenggarong, Kalimantan Timur, Indonesia

⁴ Department of Neurosurgery, RSUD Aji Muhammad Parikesit,
Tenggarong, Kalimantan Timur, Indonesia

⁵ Department of Neurology, RSUD Aji Muhammad Parikesit,
Tenggarong, Kalimantan Timur, Indonesia

⁷ Department of Pediatrics, RSUD Aji Muhammad Parikesit,
Tenggarong, Kalimantan Timur, Indonesia

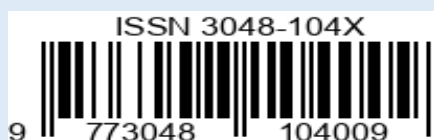
Correspondence: Cassandra Savira Alisa, dr. | Email:
Sandrasavira94@gmail.com

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ABSTRACT

Introduction: Brain abscess represents one of the most catastrophic intracranial complications of cyanotic congenital heart disease (CCHD), occurring predominantly in patients with uncorrected defects characterized by right-to-left intracardiac shunting. Tetralogy of Fallot (TOF), comprising ventricular septal defect (VSD), infundibular-valvar pulmonary stenosis, right ventricular hypertrophy, and overriding aorta, constitutes the most commonly implicated CCHD subtype, accounting for approximately 61% of all cardiogenic brain abscess

cases. The precise pathophysiological interplay between chronic systemic hypoxemia, polycythemia, relative iron deficiency, paradoxical embolism of septic microthrombi, and disruption of the blood-brain barrier remains an active area of investigation in pediatric neurocardiology.

Case Illustration: We report a 6-year-9-month-old male child (A.Z) born with very low birth weight (1,300 g) and neonatally diagnosed with TOF, who had not undergone corrective cardiac surgery due to economic constraints and lost follow-up for over 4 years. He presented to IGD RSUD Aji Muhammad Parikesit on June 8, 2026 with generalized tonic-clonic seizures, one month of intermittent fever, and SpO₂ 60% on room air. Echocardiography confirmed TOF with severe infundibular-valvar pulmonary stenosis and 1.4 cm malalignment VSD. Contrast-enhanced brain CT revealed a deep-seated cerebral abscess. On June 15, 2026, the patient underwent craniotomy with open evacuation of the deep-seated cerebral abscess, subdural drainage, and decompressive craniotomy with duraplasty, guided by neuronavigation. Post-operative course showed resolution of seizures, defervescence, and neurological stabilization. Pus and blood cultures yielded no pathogenic growth.

Discussion: This case underscores the well-established neurological vulnerability of children with uncorrected TOF. The cardinal pathomechanism involves paradoxical embolization of bacteremia-containing microthrombi through the right-to-left VSD shunt, bypassing pulmonary

capillary bactericidal filtration, facilitated by hypercoagulability of secondary polycythemia and relative iron deficiency. Sterile culture results, observed in up to 61% of cardiogenic brain abscess series, add diagnostic complexity. The ESCMID 2023 guidelines recommend empirical third-generation cephalosporin plus metronidazole combined with surgical aspiration or excision for 6–8 weeks.

Conclusion: Brain abscess in uncorrected TOF represents a neurosurgical and cardiac emergency demanding immediate multidisciplinary intervention. Timely corrective cardiac surgery remains the primary preventive strategy against cardiogenic brain abscess.

Keywords: brain abscess; cerebral abscess; Tetralogy of Fallot; cyanotic congenital heart disease; right-to-left shunt; cardiogenic abscess; paradoxical embolism

INTRODUCTION

Background

Brain abscess (cerebral abscess) is defined as a focal, encapsulated suppurative infection within the brain parenchyma, evolving through sequential histopathological stages: early cerebritis, late cerebritis, early capsule formation, and late capsule formation. While multiple predisposing etiologies exist — including contiguous otorhinological spread, cranial trauma, post-neurosurgical infection, and hematogenous dissemination — the association between cyanotic congenital heart disease (CCHD) and brain abscess constitutes one of the most clinically consequential relationships in pediatric medicine.

Tetralogy of Fallot (TOF) is the most prevalent form of CCHD, occurring in approximately 0.28 per 1,000 live births and representing 5–7% of all congenital cardiac defects **(4)**. The four anatomical components — anterocephalad malalignment VSD, infundibular and/or valvar pulmonary stenosis (PS), right ventricular hypertrophy (RVH), and overriding aorta — collectively generate a hemodynamic substrate of right-to-left intracardiac shunting and systemic cyanosis, the severity of which is directly proportional to the degree of right ventricular outflow tract (RVOT) obstruction **(4, 12)**.

The epidemiological relationship between TOF and brain abscess has been established since the landmark anatomopathological series by Kagawa et al., which documented that TOF accounts for 61.2% of all cardiogenic brain abscess cases, with the characteristic age peak between 4–7 years — precisely the age of the present patient **(1)**. The pivotal series by Udayakumaran et al. (2017), reporting 26 pediatric cardiogenic brain abscess cases at a tertiary South Asian center, similarly confirmed that unresolved CCHD is the most common etiology of cerebral abscess in children, and that uncorrected or palliated CCHD confers persistent risk for occurrence, persistence, and recurrence **(2)**.

The natural history of unrepaired TOF carries grave extracardiac consequences. The 2018 AHA/ACC and 2020 ESC Guidelines for the Management of Congenital Heart Disease unambiguously recommend complete intracardiac repair — preferably in infancy — as the definitive

treatment **(12, 13)**. Long-term survival data from the Pediatric Cardiac Care Consortium (PCCC) multicenter registry, encompassing 3,283 patients, demonstrates 25-year transplant-free survival of 94.5% following surgical repair, reinforcing the transformative impact of corrective surgery on prognosis **(5)**. Conversely, survival beyond 20 years in unrepaired TOF is observed in fewer than 10% of patients **(2, 4)**.

In resource-limited settings — including many regions of Indonesia — a substantial proportion of children with TOF remain without corrective or palliative intervention, driven by socioeconomic barriers, limited access to tertiary cardiac surgical centers, and parental non-compliance with medical follow-up. This clinical reality renders cardiogenic brain abscess an ongoing, preventable public health problem in the developing world. **(19)**

Research Objectives

The primary objective of this case report is to provide a comprehensive clinicopathological description of deep-seated brain abscess as a life-threatening neurological complication in a child with uncorrected TOF, with emphasis on the pathophysiological mechanisms of cardiogenic abscess formation, its diagnostic approach, and the multidisciplinary surgical and medical management strategy employed at RSUD Aji Muhammad Parikesit, Tenggarong, Kalimantan Timur. Secondary objectives include a detailed literature review on epidemiology, microbiology, neuroimaging, surgical technique selection, antimicrobial regimen, and prognostic determinants of cardiogenic brain abscess **(1, 2, 3, 8)**.

Research Benefits

This report contributes to the clinical literature by documenting a contemporary case of cardiogenic brain abscess in a resource-constrained Indonesian setting, highlighting the diagnostic and therapeutic challenges encountered. It serves to reinforce clinical vigilance among emergency physicians, pediatricians, and cardiologists regarding the imperative of early recognition of intracranial septic complications in children with CCHD, and to advocate for timely corrective cardiac surgery as the cornerstone of neurological complication prevention.

Research Hypothesis

We postulate that in this patient, chronic systemic hypoxemia secondary to severe RVOT obstruction created a milieu of cerebral ischemic vulnerability, secondary polycythemia, and relative iron deficiency **(17, 18)**. A transient bacteremic episode — likely originating from an unidentified mucosal source — allowed septic microemboli to bypass pulmonary capillary bactericidal filtration via the right-to-left VSD shunt, seeding an ischemic cerebral locus and initiating abscess formation **(1, 14, 15)**.

Research Gap

The existing literature on cardiogenic brain abscess in children with CCHD is predominantly composed of retrospective series from tertiary cardiac surgical centers in developed nations or high-volume South Asian hospitals **(1, 2, 3)**. Contemporary case reports from Indonesian centers — where the epidemiological burden of unrepaired CCHD remains disproportionately elevated — are conspicuously scarce. Furthermore, the impact of very low birth weight (VLBW, <1,500 g) as an additive neurodevelopmental risk factor in patients who subsequently develop cardiogenic brain abscess has received minimal scholarly attention. The perioperative cardiac anesthetic challenges — particularly the risk of tetralogy spell during neurosurgical procedures under general anesthesia — are also insufficiently documented in the published literature **(19)**.

Novelty

To the best of the authors' knowledge, this case represents the first documented report from East Kalimantan, Indonesia of cardiogenic brain abscess complicating uncorrected TOF in a child born with VLBW who had been lost to cardiac follow-up for over four years. The concurrent documentation of echocardiographic, electrocardiographic, laboratory, and operative findings in a single case, together with the multidisciplinary perioperative management strategy — including specific measures to prevent intraoperative tetralogy spell, propranolol continuation perioperatively, and the neurosurgical technique of deep-seated abscess evacuation with decompressive craniectomy — constitutes a valuable contribution to the regional and national literature.

CASE ILLUSTRATION

Patient Identity

Patient Name	A.Z
Date of Birth / Age	6 years 9 months 23 days
Sex	Male
Address	Jonggon A, Tenggara, Kutai Kartanegara
Insurance	BPJS Kesehatan
Date of Admission	June 8, 2026

Anamnesis

Chief Complaint

Generalized tonic-clonic convulsive seizure since 2 hours prior to admission.

History of Present Illness

The patient, a 6-year-9-month-old male, was brought to the Emergency Department (IGD) of RSUD Aji Muhammad Parikesit on June 8, 2026 with a chief complaint of generalized tonic-clonic seizure onset 2 hours prior to admission. The seizure episode lasted approximately 5 minutes and was characterized by bilateral upper and lower limb tonic-clonic movements. The patient was post-ictal upon arrival. Several days prior, the family reported a single brief self-limiting seizure episode of approximately 1 minute for which no medical attention was sought.

In the weeks preceding the acute presentation, the patient had been experiencing intermittent (remittent) fever for approximately 1 month's duration, with temperatures fluctuating but not reaching sustained pyrexia. Concurrent headache (cephalgia) had been reported, but its precise character could not be fully elicited from the child. There was no antecedent history of upper respiratory tract infection, odontogenic sepsis, otitis media, sinusitis, skin infection, or cranial trauma. No prior history of meningitis, encephalitis, or neurosurgical procedure was reported.

The patient's chronic medical history is notable for significant exercise intolerance and exertional dyspnea, such that he was not enrolled in school and largely confined to the household.

Frequent episodes of dyspnea and fatigue with minimal exertion had been present throughout the patient's life.

Birth and Neonatal History

The patient was born via spontaneous vertex delivery at a local hospital with a birth weight of 1,300 grams (Very Low Birth Weight, VLBW, <1,500 g). He was the first child of a mother aged approximately 40 years — a recognized risk factor for de novo fetal cardiac anomalies. In the immediate neonatal period, episodic central cyanosis and low SpO₂ were observed, prompting echocardiographic evaluation, which demonstrated findings consistent with Tetralogy of Fallot (TOF).

Cardiac Follow-up History

Following the neonatal TOF diagnosis, the patient was followed regularly in pediatric cardiology for the first two years of life. The family was counseled regarding the necessity of corrective cardiac surgery and scheduled follow-up. However, after approximately 2 years of age, the family ceased all follow-up attendance, citing perceived clinical improvement and economic constraints. The patient had therefore not received any cardiological evaluation, palliation, or corrective surgery for approximately 4 years and 9 months prior to the current admission.

Physical Examination

Vital Signs at Admission — Emergency Department (June 8, 2026)

Parameter	Finding
Glasgow Coma Scale (GCS)	E4V5M6 = 15 (Alert)
Blood Pressure (BP)	100/60 mmHg
Heart Rate (HR)	125 bpm, regular
Respiratory Rate (RR)	35 breaths/minute (tachypnea)
Temperature (T)	39.0°C (febrile)

Parameter	Finding
SpO2 — Room Air	60% (severe hypoxemia)
SpO2 — Nasal Cannula 5 LPM	78–80%
SpO2 — Simple Mask 8 LPM	89%

Physical Examination by System

Head/Neck: Conjunctivae not anemic; sclerae not icteric; no nuchal rigidity. Central cyanosis on lips and mucous membranes (+).

Thorax: Intercostal and subcostal retractions present (+). Vesicular breath sounds bilaterally without crackles or wheezing. Prominent grade III-IV/VI systolic murmur consistent with RVOT obstruction and VSD. No gallop rhythm.

Abdomen: Flat, non-distended. Normoactive bowel sounds. No organomegaly, no tenderness.

Extremities: Warm acral perfusion, no pitting edema. Bilateral digital clubbing (hippocratic fingers and toes) (+) consistent with chronic hypoxemia.



Figure 1. Clinical Presentation

Investigations

Hematological Profile — Sequential Laboratory Results

Three serial complete blood count (CBC) examinations were performed: at admission (June 8, 2026), on Day 4 (June 12, 2026), and on the pre-operative day (June 15, 2026).

Table 1. Complete Blood Count — June 8, 2026 (Admission)

Parameter	Result	Reference Range	Unit
Hemoglobin	17.1 *	10.7–14.7	g/dL
Erythrocytes	6.1 *	3.7–5.7	/uL ($\times 10^6$)
Hematocrit	48 *	31–43	%
MCV	78.5	72–88	fL
MCH	28.2	22–34	pg
MCHC	35.9	32–36	g/dL
Leukocytes	32,850 *	5,000–14,500	/uL
Neutrophils	91.3 *	50–70	%
Lymphocytes	3.0 *	25–40	%
Platelets	466,000	181,000–521,000	/uL
Sodium	130 *	132–145	mmol/L
Potassium	3.9	3.1–5.1	mmol/L
Chloride	100	96–111	mmol/L
Random Blood Glucose	99	60–150	mg/dL

Table 2. Complete Blood Count — June 12, 2026 (Day 4)

Parameter	Result	Reference Range	Unit
Hemoglobin	15.4 *	10.7–14.7	g/dL
Erythrocytes	5.5	3.7–5.7	/uL ($\times 10^6$)
Hematocrit	42	31–43	%
Leukocytes	18,540 *	5,000–14,500	/uL
Neutrophils	83.3 *	50–70	%
Lymphocytes	11.4 *	25–40	%
Platelets	358,000	181,000–521,000	/uL

Table 3. Complete Blood Count — June 15, 2026 (Pre-operative Day)

Parameter	Result	Reference Range	Unit
Hemoglobin	14.3	10.7–14.7	g/dL
Erythrocytes	5.1	3.7–5.7	/uL ($\times 10^6$)
Hematocrit	39	31–43	%
Leukocytes	18,260 *	5,000–14,500	/uL
Neutrophils	91.5 *	50–70	%
Lymphocytes	6.6 *	25–40	%
Platelets	370,000	181,000–521,000	/uL

Table 4. Additional Laboratory Results — June 9, 2026

Parameter	Result	Reference Range	Unit
Ureum	12 *	19–47	mg/dL

Parameter	Result	Reference Range	Unit
Creatinine	0.4 *	0.44–0.65	mg/dL
Sodium	132	132–145	mmol/L
Potassium	3.4	3.1–5.1	mmol/L
Chloride	105	96–111	mmol/L
Malaria DDR	Negative	Negative	—
Tubex Typhoid	2 (Negative)	≥4: Positive	—
Urine — Protein	Negative	Negative	—
Urine — Leukocytes	0–1	0–5 /LPB	/LPB

Microbiology Results

Culture of intraoperative abscess pus specimen (June 19, 2026): No pathogenic microorganism growth identified. Blood culture (pre-operative): No pathogenic microorganism growth identified. These sterile culture results are consistent with the published literature documenting sterile abscess cultures in 50–61% of cardiogenic brain abscess cases, attributable to prior empirical antibiotic administration and the fastidious nature of anaerobic and streptococcal organisms (1). Recommendation from the infectious disease specialist (dr. Morfino, Sp.MK): Antibiotic regimen may be transitioned to ampicillin-sulbactam combined with gentamicin, or continued until completion of the 14-day course.

Electrocardiography (ECG) — June 14, 2026

The 12-lead ECG was performed at RSUD Aji Muhammad Parikesit with a heart rate of approximately 94 bpm. Findings demonstrated right ventricular hypertrophy (RVH): right-axis deviation, prominent R-waves in right precordial leads (V1–V2), and right heart strain pattern — consistent with the echocardiographic evidence of severe RVH in TOF. No acute ischemic changes or significant arrhythmia were identified.

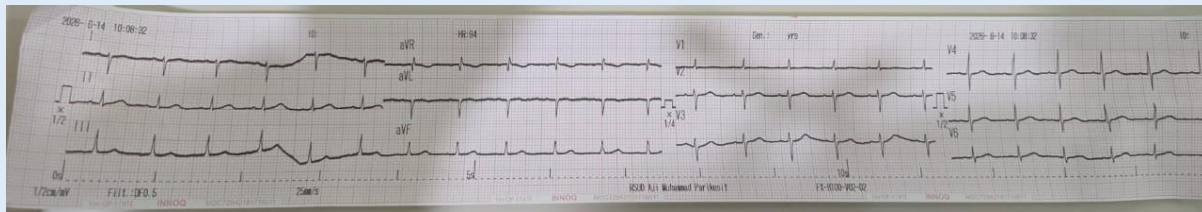


Figure 2. Electrocardiography (ECG) — June 14, 2026

Chest X-Ray (PA) — June 11, 2026

Frontal chest radiograph demonstrated the characteristic boot-shaped (coeur en sabot) cardiac silhouette of Tetralogy of Fallot: RVH elevating the cardiac apex superiorly, with concavity of the left heart border due to hypoplastic main pulmonary artery. The aorta appeared prominent with rightward shift consistent with overriding aorta. Pulmonary vascularity appeared diminished bilaterally, consistent with reduced pulmonary blood flow secondary to severe RVOT obstruction. No focal consolidation, pleural effusion, or pneumothorax was identified.



Figure 3. Chest X-Ray (PA) — June 11, 2026

Echocardiography — June 9, 2026

Transthoracic echocardiography (TTE) performed by dr. Yoga Alfian Noor, Sp.JP, FIHA documented: Situs Solitus; AV-VA concordance; pulmonary venous drainage normal; RVH prominent; severe infundibular-valvar pulmonary stenosis (MPA Vmax 1.82 m/s; MPA maxPG 13.28

mmHg); VSD malalignment type, diameter 1.4 cm; overriding aorta with normal leftward arcus; no PDA; no CoArct; LV systolic function normal (EF Teich 62%, FS 32%). Conclusion: Consistent with Tetralogy of Fallot (TOF).

Contrast-Enhanced CT Brain — June 10, 2026

CECT of the brain was performed under anesthesiologic support with premedication (intravenous metoclopramide 2 mg and dexamethasone 2 mg). The CECT demonstrated a ring-enhancing lesion in the deep cerebral parenchyma, consistent with an encapsulated brain abscess: central hypodense purulent core surrounded by a hyperdense enhancing capsule, with surrounding perilesional cerebral edema and mass effect. The deep-seated anatomical localization informed the operative planning, including neuronavigation guidance.

Diagnosis

- Seizure secondary to Brain Abscess (Cerebral Abscess) — Deep-Seated
- Uncorrected Tetralogy of Fallot (TOF) — Severe Infundibular-Valvar Pulmonary Stenosis
- Secondary Polycythemia with Chronic Systemic Hypoxemia
- Epilepsy (under evaluation)

Multidisciplinary Management

Medical Management

Upon presentation, immediate ABC stabilization was initiated. Supplemental oxygen was commenced targeting SpO₂ >80% per cardiology recommendation (dr. Yoga, Sp.JP). Dual intravenous access was established. Propranolol 3x2 mg oral was commenced to prevent infundibular spasm and tetralogy spell. Empirical antibiotic therapy: Ceftriaxone 2x600 mg IV (third-generation cephalosporin) plus Metronidazole 4x100 mg IV (anaerobic coverage). Dexamethasone 2x5 mg IV as adjunctive corticosteroid for perilesional cerebral edema. Paracetamol 4x150 mg IV as antipyretic. Omeprazole 2x10 mg IV as gastroprotection.

Pre-operative Assessment and Anesthetic Risk Stratification

Given the significant perioperative risk in severe uncorrected CCHD, a formal anesthesiology consultation (dr. Yudistira, Sp.AN) was obtained. The family received a detailed informed consent discussion regarding the risks of general anesthesia in TOF, including tetralogy spell, hemodynamic collapse, and fatal outcome. Key pre-operative instructions included: dual peripheral IV access

(minimum 24G, upper extremity); NPO 6 hours; propranolol continuation on the morning of surgery with minimal water (maximum 2 sips, ~20 mL); Asering 5% at 45 mL/hour by infusion pump during NPO period; avoidance of beta-agonist bronchodilators; mandatory PICU backup with mechanical ventilator ready pre-operatively. Knee-chest position and rescue morphine 1 mg IV were designated for intraoperative tetralogy spell management.

Operative Procedure — June 15, 2026

Procedure (dr. Mustaqim Apriyansa Rahmadhan, Sp.BS): Craniotomy with Open Evacuation of Deep-Seated Cerebral Abscess + Subdural Drainage + Decompressive Craniotomy with Durotomy and Duraplasty, guided by neuronavigation. Intraoperatively, a well-encapsulated purulent collection was identified and completely evacuated. A subdural drain was placed for continued egress of residual pus. Decompressive craniectomy with duraplasty was performed to relieve mass effect and reduce intracranial pressure. Operative pus was submitted for microbiological culture. The procedure was completed without significant intraoperative cardiac or anesthetic adverse events. The patient was transferred directly to PICU post-operatively.

Post-operative Follow-up

Table 5. Daily Patient Follow-up (Clinically Significant Days)

Date	Subjective	Objective (Key)	Assessment	Plan/Advice
June 9	Fever(-), Seizure(-), SpO2 70-80% with O2	HR 80, T 39°C, SpO2 80% NK 5LPM	Obs Convulsion + TOF	ECHO, CT scan kontras, Propranolol 3x2mg, Target SpO2 75-85%
June 10	Dyspnea+, Fatigue+, Seizure-	HR 98, T 36.2°C, SpO2 95% NK 5LPM	Obs Convulsion + TOF	CT scan kepala kontras + anesthesi pendampingan, EEG

Date	Subjective	Objective (Key)	Assessment	Plan/Advice
June 11	Dyspnea+, Fever intermittent	HR 98, T 36.2°C, SpO2 95% NK 5LPM	Obs Convulsion + TOF + Epilepsi	CT scan kontras, Cek DL/Tubex/DDR/CRP
June 12	Dyspnea(+) reduced, Good contact	HR 98, T 36.2°C, SpO2 89% NK 5LPM	Obs Convulsion + TOF	Raber bedah saraf; Pro operasi open drainase
June 13	Dyspnea+, No seizure	HR 98, T 36.2°C, SpO2 91% NK 5LPM	Obs Convulsion + TOF	ACC SpJP operasi; Pro op drainase + neuronavigasi
June 14	Headache+, Dyspnea-, Vomiting x1	HR 98, T 36.2°C, SpO2 92% NK 5LPM	Obs Convulsion + TOF	ACC operasi; PICU+ventilator ready; Instruksi anestesi periop
June 15	Seizure-, Headache-, Fever-	HR 98, T 36.2°C, SpO2 92% NK 5LPM	Brain Abscess + TOF	OPERASI: Craniotomi evakuasi abses + subdural drainage + dekompresi + duraplasty
June 16– 18	Seizure-, Dyspnea-, Fever-, Vomiting+	Stable, maintained SpO2	Post-op Day 1–3	Ceftriaxone, Metronidazole, Dexametasone, Propranolol,

Date	Subjective	Objective (Key)	Assessment	Plan/Advice
				Ondansetron lanjut; Pro aff drain H5
June 19– 21	Seizure-, Fever- , Stable	Improving	Post-op Day 4–6	Kultur: no growth; SpMK: pertimbangkan Ampicillin-Sulbactam + Gentamicin; terapi s/d H14

Final Diagnoses

- Brain Abscess (Cerebral Abscess), Deep-Seated — Post-Craniotomy Evacuation
- Uncorrected Tetralogy of Fallot (TOF) with Severe Infundibular-Valvar Pulmonary Stenosis
- Secondary Polycythemia with Chronic Systemic Cyanosis
- Recurrent Convulsive Seizures — resolved post-operatively

DISCUSSION

Definition and Epidemiology of Brain Abscess in Cyanotic Congenital Heart Disease

Brain abscess (cerebral abscess) is defined as a focal, encapsulated collection of purulent exudate within the brain parenchyma, initiated by hematogenous or direct microbial seeding, evolving through stereotyped histopathological stages: early cerebritis, late cerebritis, early capsule formation, and late capsule formation. In immunocompetent children without anatomical predisposition, brain abscess occurs at an incidence of approximately 0.4–0.9 per 100,000 per year. In the context of CCHD with right-to-left shunting, this risk escalates dramatically, constituting the most feared neurological complication of this cardiac category (3, 8).

The seminal series by Kagawa et al. (1983) documented that among 62 cases of brain abscess associated with CCHD, TOF accounted for 38 cases (61.2%), with a characteristic age distribution

peaking sharply at 4–7 years — precisely concordant with the age of the present patient (6 years 9 months) **(1)**. Udayakumaran et al. (2017), in their retrospective analysis of 26 pediatric cardiogenic brain abscess cases managed surgically, confirmed a mean patient age of 7.19 years, 10 of 26 patients (38%) requiring re-aspiration after initial surgery, and identified uncorrected or palliated CCHD as the dominant etiological factor, with cardiogenic origin being the most common cause of cerebral abscess in children **(2)**.

The global burden of unrepaired TOF remains disproportionately concentrated in resource-limited settings. A systematic review and meta-analysis by Geleta et al. (2025) documented a pooled TOF prevalence of 10% (95% CI: 9–12%) among children with congenital heart defects in Sub-Saharan Africa — substantially exceeding global estimates from high-income nations — driven by limited access to pediatric cardiac surgery. Analogous challenges prevail in much of Indonesia, particularly East Kalimantan **(19)**.

Pathophysiology of Cardiogenic Brain Abscess in TOF

The pathogenesis of brain abscess in TOF is multifactorial, involving the synergistic interplay of several well-characterized mechanisms that collectively create a cerebrovascular environment uniquely susceptible to bacterial colonization **(1, 14, 15)**.

Right-to-Left Intracardiac Shunting and Paradoxical Embolism

The foundational mechanism involves paradoxical passage of bacteremia-containing venous blood through the right-to-left VSD shunt of TOF, entirely bypassing pulmonary capillary bactericidal filtration. Under normal physiological conditions, pulmonary capillary endothelial cells, alveolar macrophages, and the physical sieve of the microvascular bed efficiently trap and eliminate blood-borne bacteria from venous blood before it enters the systemic circulation. In TOF with right-to-left shunting, this immunological filtration step is abolished, allowing circulating microorganisms direct access to the cerebral arterial circulation, where they impact in terminal arteriolar beds, producing focal ischemia permissive for bacterial proliferation **(1, 2)**.

The predilection for the MCA territory — particularly the frontal, temporal, and parietal lobes (76% of abscess sites in the Kagawa series) — reflects the hemodynamic distribution of MCA flow. The deep-seated location in the present patient is consistent with deep perforating artery embolization

(1). Kawamata et al. (2001) demonstrated analogous paradoxical embolism pathomechanics in the setting of patent foramen ovale (PFO), documenting that even modest right-to-left atrial shunts could facilitate bacteremic cerebral seeding from dental sources without additional risk factors (15). Boother et al. (2017), in a large prospective series of 445 patients with pulmonary AVMs — another right-to-left shunt model — identified low oxygen saturation as an independent multivariate predictor of cerebral abscess, consistent with this patient's profound hypoxemia (SpO₂ 60% room air) (14).

Shovlin et al. (2007), in a cohort of 323 consecutive patients with pulmonary AVMs and hereditary hemorrhagic telangiectasia, documented that primary determinants of brain abscess risk were independent of PAVM severity, with male sex (hazard ratio 3.61; 95% CI 1.58–8.25) and bacteremia from dental sources as dominant risk factors — reinforcing the importance of bacterial seeding in the right-to-left shunt context (16).

Secondary Polycythemia and Hypercoagulability

Chronic systemic hypoxemia in TOF stimulates renal erythropoietin (EPO) production via hypoxia-inducible factor (HIF)-2 α -mediated transcriptional activation, driving compensatory secondary erythrocytosis (polycythemia). The present patient manifested marked polycythemia: Hb 17.1 g/dL, Ht 48%, erythrocytes 6.1×10^6 /uL. Leachman et al. (1965) documented that Hb >18 g/dL in TOF patients undergoing surgical correction was associated with surgical mortality of 31% versus 10% in those with Hb <18 g/dL, underscoring the systemic impact of polycythemia (17).

Secondary polycythemia produces a hypercoagulable state through increased blood viscosity, platelet activation by endothelial shear stress, and impaired fibrinolysis. This milieu predisposes to cerebral microvascular thrombosis, creating ischemic foci that provide the optimal condition for bacterial colonization. The elevated hematocrit simultaneously reduces cerebral blood flow velocity, impairing local immune surveillance (17, 6).

Relative Iron Deficiency

A frequently underappreciated pathomechanism is relative iron deficiency in the context of polycythemia. In TOF, the compensatory erythroid drive generates iron demands exceeding dietary supply, resulting in low transferrin saturation and microcytosis — hallmarks of iron-deficient erythropoiesis even in the setting of elevated hemoglobin. Absolute or relative iron deficiency has

been identified as an independent risk factor for cerebral abscess in right-to-left shunting conditions, as iron-deficient erythrocytes exacerbate cerebral ischemia **(14)**. Ontoseno (2008), working at Dr. Soetomo Hospital Surabaya, documented that iron therapy significantly reduced hematocrit in TOF patients with relative iron deficiency (transferrin saturation <11.5%), demonstrating that coexistent relative iron deficiency amplifies ineffective erythropoiesis **(18)**. In the present patient, MCV 78.5 fL alongside marked polycythemia is suggestive of coexistent relative iron deficiency.

Cerebral Microvascular Injury and Blood-Brain Barrier Disruption

Chronic cerebral hypoperfusion secondary to reduced pulmonary blood flow in TOF produces regional cerebral ischemia with consequent impairment of blood-brain barrier (BBB) integrity. Ischemic endothelial cells express surface adhesion molecules (ICAM-1, VCAM-1) facilitating bacterial adherence and penetration. Neurosonographic studies have documented measurable delays in cerebral cortical development in fetuses with CCHD (including TOF), suggesting that neurological vulnerability begins in utero and continues postnatally, creating a chronically injured cerebrovascular substrate more susceptible to bacterial seeding **(1, 2)**.

Predisposing Factors in the Present Case

The patient exhibited a constellation of synergistic predisposing factors: (1) Uncorrected TOF with large malalignment VSD (1.4 cm) providing a high-flow right-to-left shunt; (2) Severe RVOT obstruction driving SpO₂ 60% on room air; (3) Secondary polycythemia (Hb 17.1 g/dL, Ht 48%); (4) Age 6–7 years — within the highest-risk period for cardiogenic brain abscess (4–7 years) **(1)**; (5) Male sex — identified as an independent brain abscess risk factor in right-to-left shunting conditions (HR 3.61 per Shovlin et al., 2007) **(16)**; (6) 4+ years of non-compliance with cardiac follow-up without corrective surgery; (7) VLBW (1,300 g) as a potential neurodevelopmental vulnerability; (8) Advanced maternal age (~40 years) as a de novo fetal cardiac anomaly risk factor; and (9) Resource-limited setting with geographic barriers to pediatric cardiac surgery **(2, 19)**.

Clinical Presentation and Diagnostic Approach

Signs and Symptoms

The clinical presentation of cardiogenic brain abscess in children with CCHD follows a recognizable triad: (1) symptoms of the primary cardiac defect (cyanosis, dyspnea, exercise

intolerance, clubbing); (2) systemic inflammatory signs (fever, malaise, anorexia); and (3) focal or generalized neurological manifestations (headache, seizures, focal deficits, altered consciousness). The present patient manifested all three components simultaneously (1, 2, 7).

Headache (cephalgia) — present as a prodromal symptom in this patient for approximately one month — is one of the most consistent early symptoms of brain abscess, occurring in 66% of patients in the Danish nationwide cohort (Bodilsen et al., 2022) (7). Fever was documented in approximately 54% of cases in that series, consistent with this patient's intermittent febrile course (7). The subacute one-month prodromal duration in this case is characteristic of the encapsulated abscess progressively expanding and elevating intracranial pressure before triggering critical neurological deterioration — an insidious presentation well-documented in cardiogenic brain abscess (1, 2, 3).

Laboratory Investigations

The CBC demonstrated the classic haematological profile of cardiogenic brain abscess in uncorrected TOF: severe leukocytosis with neutrophilia (WBC 32,850/uL, neutrophilia 91.3% at admission) reflecting systemic pyogenic response; polycythemia (Hb 17.1 g/dL) reflecting compensatory erythrocytosis. Serial CBC demonstrated progressive decline in leukocytosis (32,850 → 18,540 → 18,260/uL), reflecting initial antibiotic response, though persistent leukocytosis above the reference range indicated ongoing systemic inflammation (8).

The microbiological profile of cardiogenic brain abscess is dominated by streptococcal species (particularly *Streptococcus anginosus* group), anaerobes, and Gram-negative organisms when an odontogenic or gastrointestinal source is implicated. Sterile culture results occur in 50–61% of published cardiogenic brain abscess series (Kagawa et al., 1983), attributable to prior antibiotic administration, fastidious organism growth requirements, and sampling limitations (1, 23). In the present case, both abscess pus and blood cultures yielded no pathogenic growth, consistent with this well-documented pattern.

Neuroimaging

The ESCMID 2023 Brain Abscess Guidelines (Bodilsen et al., 2023) assign a strong recommendation (high quality evidence) to MRI as the preferred modality for brain abscess diagnosis, offering superior sensitivity for early cerebritis, satellite lesions, and perilesional edema (3). In this

case, contrast-enhanced CT (CECT) was pragmatically employed given the elevated risk of prolonged anesthetic exposure in severe uncorrected TOF — a clinically appropriate decision. The ring-enhancing pattern on CECT, with central hypodense purulent core, peripheral enhancing capsule, and perilesional edema, is highly characteristic of encapsulated brain abscess **(3, 8)**. The deep-seated anatomical location in the present patient, without an identified contiguous otorhinological source, is consistent with hematogenous origin via paradoxical embolization **(1, 15)**.

Medical and Neurosurgical Management

Antimicrobial Therapy

The ESCMID 2023 Brain Abscess Guidelines provide the most contemporary evidence-based framework for antimicrobial management. For community-acquired brain abscess in immunocompetent individuals, the recommended empirical regimen is a third-generation cephalosporin (e.g., ceftriaxone) combined with metronidazole (anaerobic coverage), with recommended treatment duration of 6–8 weeks (strong recommendation, moderate quality evidence) **(3)**. This regimen was precisely the empirical protocol administered to the present patient: ceftriaxone 2x600 mg IV plus metronidazole 4x100 mg IV **(3)**.

Adjunctive dexamethasone for severe perilesional cerebral edema or impending herniation is supported by a strong recommendation in the ESCMID 2023 guidelines, consistent with clinical practice in this case **(3)**. The duration recommendation of 6–8 weeks reflects the poor CNS penetration of many antibiotics and the relative immune sanctuary of the abscess cavity, further supported by Bodilsen et al. (2022) who documented no association between antibiotic timing relative to neurosurgery and 6-month mortality outcome **(7)**.

Surgical Intervention — Rationale and Technique

Surgical evacuation of brain abscess is the cornerstone of management for lesions with diameter >2.5 cm, significant mass effect, or progressive neurological deterioration — all present in the current patient. The ESCMID 2023 guidelines assign a strong recommendation to aspiration or excision of brain abscess whenever feasible **(3)**.

Ratnaike et al. (2011), in a systematic review of 78 years of brain abscess surgical literature, documented a post-CT era mean mortality of 6.6% for aspiration versus 12.7% for craniotomy

excision, supporting aspiration as a preferred first-line approach for most supratentorial parenchymal abscesses **(9)**. However, for deep-seated abscesses — as in the present case — where the abscess capsule may be poorly formed, multiloculated, or inaccessible to simple needle aspiration, open craniotomy provides superior visualization, more complete evacuation of purulent material and fibrinoid debris (a potential recurrence nidus), and the capacity for concomitant decompression **(9, 13)**.

Nathoo et al. (2011), in their landmark series of 973 patients over two decades, documented good outcomes in 81.3% of patients following surgical drainage, with predictors of mortality including intraventricular rupture (OR 12.9), coma (OR 6.8), and multiple abscesses (OR 3.4) **(8)**. Bodilsen et al. (2022), in the Danish nationwide cohort, identified intraventricular rupture (RR 3.48), immunocompromise (RR 2.84), and abscess diameter >3 cm (RR 1.81) as independent 6-month mortality predictors — none of which were present in this patient, conferring a comparatively favorable surgical risk profile **(7)**.

In this patient, the neurosurgeon elected open craniotomy with evacuation of the deep-seated abscess, subdural drainage, and decompressive craniotomy with duraplasty, guided by neuronavigation. Venanzi et al. (2025), in a 30-year single-centre retrospective analysis of 32 pediatric brain abscess patients, documented that the adoption of standardized protocols integrating minimally invasive neurosurgical techniques and prolonged antibiotic regimens led to significant reductions in both clinical and radiological sequelae, effectively eliminating mortality in the recent era **(11)**.

Perioperative Cardiac Management in TOF

The perioperative anesthetic management of a child with uncorrected TOF undergoing non-cardiac surgery carries substantial morbidity and mortality risk. The cardinal perioperative risk is the tetralogy spell (hypercyanotic crisis): acute dynamic RVOT infundibular spasm producing abrupt reduction in pulmonary blood flow, acute increase in right-to-left shunting, critical systemic desaturation, and potentially fatal cardiovascular collapse **(4, 12)**. Key anesthetic goals include: maintenance of systemic vascular resistance; prevention of tachycardia and agitation; avoidance of hypoxemia, hypercapnia, and acidosis. Propranolol — the non-selective beta-blocker administered in

this patient — is the established pharmacological prophylaxis against tetralogy spell via beta-1 receptor blockade of infundibular contraction; its perioperative continuation, including the morning-of-surgery dose, was appropriately mandated in this case **(12, 13)**.

Prognosis

The prognosis of cardiogenic brain abscess has improved dramatically in the CT era. Overall mortality rates that exceeded 40% in the pre-CT era have fallen to 4–13% with appropriate surgical and antibiotic management. Gilard et al. (2019), in a 25-year two-centre pediatric series of 144 patients, documented an overall mortality rate of 4% (3.4% in children), with favorable 3-month outcome in 86% of children **(10)**. Venanzi et al. (2025) documented mortality of 6.5% overall, effectively eliminated in the modern era following adoption of minimally invasive techniques and standardized antibiotic protocols **(11)**.

In this patient, absence of intraventricular rupture, preserved consciousness (GCS 15), and successful surgical evacuation confer a favorable neurosurgical prognosis. However, the underlying uncorrected TOF remains the primary determinant of long-term outcome. Smith et al. (2019), in the PCCC multicenter study of 3,283 surgically repaired TOF patients, documented 25-year transplant-free survival of 94.5%, demonstrating that timely corrective repair yields near-normal long-term outcomes **(5)**. Nollert et al. (1997), in a 36-year follow-up of 490 TOF repair survivors, documented actuarial survival of 97% at 10 years, 94% at 20 years, 89% at 30 years, and 85% at 36 years, with preoperative polycythemia independently associated with reduced long-term survival ($p = 0.0487$) **(6)**. Without corrective surgery, this patient remains at persistent risk for recurrent brain abscess, cerebrovascular accident, and progressive extracardiac complications of chronic hypoxemia **(4, 12, 13)**.

CONCLUSION AND RECOMMENDATIONS

Conclusion

This case report describes a rare but life-threatening complication of uncorrected TOF in a 6-year-9-month-old Indonesian male child: a deep-seated brain abscess manifesting as recurrent generalized tonic-clonic seizures, persistent fever, and severe systemic hypoxemia (SpO₂ 60% room air). The pathophysiological basis of cardiogenic brain abscess is rooted in paradoxical embolization

of bacteremia-laden venous blood via the right-to-left VSD shunt, circumventing pulmonary capillary bactericidal filtration, facilitated by the hypercoagulable milieu of secondary polycythemia and relative iron deficiency, in the setting of chronically ischemic and immunologically compromised cerebral tissue **(1, 2, 14, 15, 16)**.

The clinical triad of persistent headache, fever, and seizures in a cyanotic child must immediately raise clinical suspicion for intracranial septic complication, mandating urgent neuroimaging. Multidisciplinary management — involving pediatrics, cardiology, neurosurgery, neurology, anesthesiology, and infectious disease — and empirical antibiotic therapy (third-generation cephalosporin plus metronidazole) combined with open craniotomy, subdural drainage, and decompressive craniectomy with duraplasty, guided by neuronavigation, resulted in successful neurological recovery with resolution of seizures and clinical stabilization in this patient **(3, 8, 9)**.

Sterile culture results — present in up to 61% of cardiogenic abscess series — should not preclude empirical broad-spectrum antibiotic therapy **(1)**. Timely complete intracardiac repair of TOF — ideally in infancy — remains both the definitive treatment of the cardiac defect and the most effective prophylaxis against cardiogenic brain abscess and its associated neurological morbidity **(5, 6, 12, 13)**.

Recommendations

- Clinicians at all levels of care must maintain a high index of suspicion for intracranial suppurative complications in any child with known or suspected CCHD who presents with new-onset seizures, persistent fever, or headache, regardless of the apparent absence of an identified primary septic focus. **(1, 2, 3)**
- Urgent contrast-enhanced CT or MRI of the brain should be performed in all cyanotic children with neurological symptoms without clear alternative explanation. **(3)**
- Multidisciplinary team involvement — pediatrics, cardiology, neurosurgery, neurology, anesthesiology, and infectious disease — is mandatory for optimal perioperative management. **(2, 8, 12)**

- Timely corrective cardiac surgery for TOF, ideally within the first year of life, should be the primary preventive strategy against cardiogenic brain abscess and other extracardiac complications. (5, 6, 12, 13)
- In resource-limited settings, public health strategies to improve access to pediatric cardiac surgical care, reduce barriers to follow-up, and increase parental education regarding non-compliance risks are urgently needed. (19)
- Antibiotic prophylaxis against transient bacteremia from dental or mucosal procedures should be discussed and prescribed in all patients with uncorrected CCHD while awaiting corrective surgery. (3, 16)

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