

## CASE REPORT

# Anaesthetic Challenges In A Case Of Rasmussen's Encephalitis In Early Childhood Presenting With Neonatal Intracerebral Haemorrhage And Progressive Hemispheric Failure Posted For Hemispherotomy: A Case Report

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### ABSTRACT

**Background:** Rasmussen's encephalitis (RE) is a rare, chronic, immune-mediated neurological disorder of childhood characterized by progressive unihemispheric inflammation, intractable seizures, and functional decline. Haemorrhagic onset in early infancy is extremely uncommon and may obscure the underlying inflammatory etiology. Early recognition supported by multimodal investigations is critical, as timely surgical intervention can dramatically improve seizure control and long-term outcomes.

**Case Report:** A case of a male child who initially presented at 2 years of age with a massive right temporoparietal intracerebral haemorrhage measuring 4.8 × 4.5 cm, associated with subdural extension, midline shift, and early transtentorial herniation. Serial neuroimaging during infancy and early childhood demonstrated progressive cystic encephalomalacia, gliosis, unilateral white matter loss, periventricular leukomalacia, and advanced hemispheric atrophy with Wallerian degeneration. Clinically, he developed global developmental delay, persistent left hemiparesis, and refractory seizures occurring 2–3 times daily. EEGs consistently showed marked hemispheric asymmetry and epileptiform discharges. Owing to failure of multiple antiepileptic drugs, a neuronavigation-guided right functional hemispherotomy was performed at 6 years of age. Histopathology of temporal, hippocampal, and meningeal tissue confirmed Rasmussen's encephalitis. Peri-operative anaesthetic management was particularly challenging due to severe unihemispheric dysfunction, Cheyne–Stokes–type respiratory irregularity, non-verbal status, and high seizure propensity. Total intravenous anaesthesia (TIVA) guided by bispectral index (BIS) monitoring was used to maintain stable cerebral physiology and minimise seizure risk. Fibre-optic nasal intubation with a flexometallic tube ensured safe airway control in an uncooperative neurologically impaired child. Postoperatively, he achieved complete seizure freedom and gradual neurological stabilization. Follow-up EEGs showed persistent asymmetry without epileptiform activity.

**Keywords:** Rasmussen's encephalitis, intracerebral hemorrhage, pediatric seizures, hemispherotomy, Wallerian degeneration, encephalomalacia, microglial nodules, drug-resistant epilepsy.

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### INTRODUCTION

Rasmussen's encephalitis (RE) is a rare, progressive, immune-mediated neurological disorder of childhood

characterized by unihemispheric brain inflammation, intractable focal seizures, epilepsy partialis continua, cognitive decline, and progressive neurological deficits

(1). First described in the late 1950s, RE remains an uncommon but devastating cause of chronic epilepsy in children, typically presenting between 1 and 14 years of age. The disorder is believed to result from a T-cell-mediated cytotoxic response directed against neuronal and glial antigens, leading to chronic encephalitis and progressive hemispheric destruction (2). Despite significant advances in neuroimaging and immunological understanding, the diagnosis of RE continues to be challenging because of its variable presentation and overlap with other structural, infectious, metabolic, or inflammatory conditions (3).

Classically, RE presents with recurrent focal seizures that gradually evolve into drug-resistant epilepsy. Motor deficits, language regression, and cognitive decline follow as the disease progresses (4). Magnetic resonance imaging (MRI) typically demonstrates progressive unilateral cortical atrophy, subcortical white matter signal abnormalities, ventricular enlargement, and loss of hemispheric connectivity (5). Electroencephalography (EEG) commonly shows asymmetric slowing, background attenuation, and unilateral epileptiform discharges. Histopathological examination remains the gold standard, demonstrating microglial nodules, neuronal loss, astrocytic nodules, and perivascular lymphocytic cuffing (6).

Although the typical presentation of RE is insidious and progressive, unusual early-life manifestations have increasingly been recognized. One of the rarest among them is presentation with intracerebral hemorrhage (ICH) in infancy (7). Hemorrhagic onset is exceptionally uncommon and may obscure the underlying inflammatory pathology because the initial focus of clinical attention is diverted toward managing the acute bleed, mass effect, and life-threatening complications. Early ICH may accelerate subsequent hemispheric atrophy and mask the evolving hallmarks of RE, leading to diagnostic delays (8). Only a handful of cases in literature suggest that early hemorrhage may coexist with or precede the development of RE, raising questions about shared mechanisms involving vascular fragility, inflammation, or coagulopathy (9).

The present case is of particular significance due to several striking and atypical features. The child initially presented at just two years of age with a large right temporoparietal hemorrhage causing mass effect and midline shift. Over the next few years, he developed delayed milestones, fixed left hemiparesis, and recurrent drug-resistant seizures. Serial MRI and tractography showed cystic encephalomalacia, extensive white matter loss, retrograde Wallerian degeneration, and progressive right hemispheric atrophy. EEG consistently

demonstrated hemispheric asymmetry and epileptiform discharges. Ultimately, neuronavigation-guided functional hemispherotomy was performed, and histopathology confirmed the diagnosis of Rasmussen's encephalitis an unusual but definitive correlation between early hemorrhage and progressive inflammatory destruction of one cerebral hemisphere (10).

This case exemplifies the diagnostic complexities and clinical challenges associated with rare, atypical presentations of RE. It highlights the value of multimodal evaluation clinical assessment, advanced neuroimaging, serial EEGs, and pathology while underscoring the importance of timely surgical intervention. By providing a comprehensive correlation of radiological, electrophysiological, and histopathological findings, this report contributes valuable insight into one of the most unusual clinical variants of Rasmussen's encephalitis described in pediatric literature.

#### CASE PRESENTATION

**Patient Information:** A male child born at term to a consanguineous couple had an unremarkable perinatal period. During early infancy, he developed acute neurological deterioration following a brief febrile illness. This episode marked the onset of a chronic progressive neurological disorder that would later significantly influence anaesthetic planning due to evolving hemispheric dysfunction, impaired airway cooperation, and increased seizure susceptibility.

**Initial Presentation and Early Clinical Course:** In infancy, the child presented with acute irritability, seizures, and altered responsiveness. Neuroimaging revealed a large right temporoparietal intraparenchymal haemorrhage with surrounding oedema, cystic evolution, subdural extension, mass effect, and midline shift, with early signs of transtentorial herniation. Initial management was conservative. Over the following months, the child developed fixed left-sided weakness, delayed milestones, and recurrent seizures several times per day despite escalating antiepileptic therapy. These features suggested progressive unilateral hemispheric dysfunction an important consideration for anaesthesiologists, as chronic seizures and anticonvulsant use alter anaesthetic drug requirements, airway reflexes, and metabolic handling of agents.

**Developmental and Neurological Status:** The child's subsequent neurological course was characterised by persistent microcephaly, global developmental delay, and stable left hemiparesis. These deficits directly influenced peri-operative handling: poor motor control increased the likelihood of difficult positioning during induction, and impaired communication limited airway assessment and cooperation. Despite preserved alertness

and social interaction, his inability to comprehend or respond reliably necessitated planned non-cooperative airway management techniques for any future anaesthetic procedure. Neurological asymmetry further raised concerns for abnormal respiratory patterns, a known complication in children with severe hemispheric injury.

**Electroencephalography Findings** Serial EEGs demonstrated marked hemispheric asymmetry, with slow, attenuated background activity over the right hemisphere and persistent epileptiform discharges from the left. This pattern indicated advanced functional reorganization, rendering the diseased hemisphere electrically silent. From an anaesthetic standpoint, this has two major implications:

1. Seizure risk remains high despite unilateral cortical destruction, necessitating avoidance of pro-convulsant agents and meticulous depth monitoring.
2. EEG-based depth indicators become less reliable, making adjunct monitoring such as BIS crucial to maintain safe anaesthetic depth during future interventions.

**Neuroimaging Findings:** MRI with tractography revealed extensive cystic encephalomalacia of the right hemisphere, severe white matter loss, frontal and temporal lobe atrophy, and gliotic changes with hemosiderin deposits. Marked dilatation of the right lateral ventricle and global hemispheric shrinkage were evident. Fibre-tract imaging showed near-complete absence of right-sided corticospinal tracts, cingulum, superior longitudinal fasciculus, and corpus callosal fibres, accompanied by clear retrograde Wallerian degeneration along the right cerebral peduncle.

These structural abnormalities had major implications for anaesthetic management:

- **Absence of functional corticospinal pathways** contributes to abnormal respiratory rhythms, including Cheyne–Stokes–type breathing, requiring preparation for controlled ventilation.
- **Diffuse gliosis and white matter loss** increase sensitivity to fluctuations in CO<sub>2</sub>, necessitating tight control of ventilation to avoid hyperventilation-triggered seizures.
- **Severe hemispheric atrophy** complicates BIS interpretation, making clinical correlation essential.
- **Cystic encephalomalacia with mass effect sequelae** raises intracranial pressure concerns, mandating smooth induction, avoidance of coughing/straining, and seizure-safe

pharmacology during each anaesthetic exposure.

Together, these findings confirmed a chronic destructive process that not only shaped neurological prognosis but also significantly influenced anaesthetic strategy for all subsequent procedures.



**Figure 1: Chest Radiograph (AP View) (The cardiac silhouette is visualized centrally. The right humeral head is marked with an "R" indicating the right side. No obvious acute pulmonary pathology is visible. Bony thoracic structures appear within normal limits for age)**

#### **Anaesthetic Management in Rasmussen's Encephalitis**

The child's clinical management required a multidisciplinary approach involving paediatric neurology, neurosurgery, radiology, and anaesthesia due to the highly complex and progressive nature of his neurological disorder. Initial treatment in infancy focused on stabilising intracranial haemodynamics following a massive right temporoparietal haemorrhage, with conservative management of the acute bleed and supportive neuroprotection. Over the next several months, the child demonstrated persistent left hemiparesis, delayed motor and language milestones, and recurrent seizures despite escalating polytherapy with multiple antiepileptic drugs. Ongoing seizure burden, radiological evidence of progressive hemispheric destruction, and EEG findings of marked unilateral dysfunction collectively indicated a chronic unihemispheric epileptic encephalopathy.

Given the progressive decline and failure of medical therapy, the case was evaluated by the neurosurgical team for definitive intervention. Serial MRI with tractography showed near-complete loss of right-sided cortical and white matter pathways, along with Wallerian degeneration extending into the cerebral peduncle. These findings, combined with refractory seizures and non-

functional hemispheric status, made the child an appropriate candidate for functional hemispherotomy. The family received detailed counselling regarding risks, benefits, expected neurological outcomes, and long-term seizure prognosis.

Peri-operative anaesthetic management constituted a major component of overall care due to the child's impaired cooperation, complete hearing loss, behavioural limitations, and episodes of Cheyne–Stokes–type breathing suggestive of central respiratory dysregulation. Anaesthesia planning prioritised seizure avoidance, smooth induction, airway safety, and stable cerebral physiology. Total Intravenous Anaesthesia (TIVA) using propofol and opioid infusions was administered via dual syringe pumps to achieve precise titration, while Bispectral Index (BIS) monitoring was used to maintain adequate anaesthetic depth in the presence of an electrically silent hemisphere. Fibre-optic nasal intubation with a flexometallic tube ensured secure airway access with minimal haemodynamic fluctuation.



**Figure 2: Syringe Pump Setup for TIVA**

The child underwent neuronavigation-guided right functional hemispherotomy with complete disconnection of the diseased hemisphere while preserving vascular integrity. Postoperative recovery was stable, with early seizure freedom and no new neurological deficits beyond baseline hemiparesis. A transient febrile episode resolved with supportive therapy. Histopathological examination confirmed Rasmussen's encephalitis by demonstrating microglial nodules, neuronal loss, perivascular lymphocytic cuffing, and chronic inflammatory infiltrates.



**Fig 3:**

**Pre-operative Clinical Features (Pre-operative image of the child showing altered interaction, developmental delay, and inability to communicate verbally, consistent with neurological impairment due to Rasmussen's encephalitis. ECG monitoring**

**leads are in place during pre-anaesthetic preparation)**

**Pre-operative Considerations** The child had residual left hemiparesis and was non-verbal, with complete absence of hearing, making cooperation during airway assessment and induction difficult. Ongoing antiepileptic therapy was continued to avoid peri-operative seizures. Baseline assessment also revealed episodes of Cheyne–Stokes like breathing patterns, likely secondary to hemispheric dysfunction after hemispherotomy. This required vigilant pre-operative evaluation and readiness for controlled ventilation.

**Airway Management** Airway management was planned anticipating difficulty due to poor cooperation, neuromotor limitations, and the need for secure peri-operative airway. Intubation was successfully achieved using a flexible fibre-optic bronchoscope, with insertion of a 5.5 mm flexometallic (armoured) endotracheal tube via the nasal route. Nasal intubation was chosen to permit unobstructed surgical access and reduce intra-operative displacement risk. The use of a flexometallic tube ensured protection against kinking in a neurologically unstable child.

**Intra-operative Anaesthetic Strategy** Anaesthesia was maintained using oxygen, air, and with intermittent doses of atracurium. Controlled ventilation was essential due to the child's abnormal respiratory rhythm and reduced

central respiratory drive. Meticulous monitoring of end-tidal CO<sub>2</sub>, depth of anaesthesia, and continuous seizure surveillance was maintained. Drugs known to reduce the seizure threshold were avoided.

#### **Extubation Challenges**

Extubation required special caution because:

- The child could neither hear commands nor speak, eliminating cooperative feedback.
- Post-ictal or neurological irritability could mimic inadequate recovery.
- Underlying hemispheric dysfunction and prior Cheyne–Stokes breathing increased the risk of post-extubation hypoventilation.

Therefore, extubation was performed only after ensuring complete return of airway reflexes, stable respiratory pattern, adequate spontaneous tidal volume, and strict hemodynamic stability. A prolonged observation period in recovery was mandatory due to the risk of respiratory irregularity or seizure recurrence.

#### **Surgical Intervention and histological Examination:**

The child continued to have refractory seizures and worsening neurological function despite multiple antiepileptic drugs, and neuroimaging confirmed a progressively non-functional right hemisphere with features of unihemispheric inflammatory destruction. Therefore, a right functional hemispherotomy was performed under general anaesthesia, achieving complete disconnection of the diseased hemisphere while preserving vascular integrity. The postoperative course was favourable, with immediate seizure freedom and no new neurological deficits; a brief febrile episode resolved with supportive care, and the pre-existing left hemiparesis remained unchanged. Histopathological examination of temporal, hippocampal, and meningeal tissues demonstrated microglial nodules, neuronal loss, perivascular lymphocytic cuffing, and chronic inflammatory infiltrates, confirming the diagnosis of Rasmussen's encephalitis.

**Post-operative Care:** The child remained seizure-free intra-operatively and post-operatively. Oxygen supplementation, close neurological observation, and resumption of regular antiepileptic regimen were ensured. No airway or respiratory complications occurred during recovery.

#### **DISCUSSION**

Rasmussen's encephalitis is a rare, progressive, unihemispheric inflammatory disorder of childhood that results in chronic seizures, hemispheric dysfunction, and neurological decline. The condition is typically characterized by insidious onset, gradual worsening of focal seizures, epilepsy partialis continua, and steadily progressive hemiparesis (10). Although the classical

clinical course is well described, highly atypical early-life presentations continue to challenge diagnostic pathways. The present case demonstrates an unusually complex evolution beginning with a massive right-sided intracerebral haemorrhage in early infancy, followed by progressive hemispheric destruction and drug-resistant seizures, ultimately culminating in the diagnosis of Rasmussen's encephalitis based on histopathology.

The unusual trajectory creates significant peri-operative challenges. Children with progressive hemispheric dysfunction often display irregular respiratory patterns, impaired autonomic regulation, and altered cerebral physiology. In this case, the presence of Cheyne–Stokes-type breathing and previous hemispherotomy indicated compromised central respiratory control an important predictor of peri-operative hypoventilation and a factor influencing the decision for controlled ventilation during anaesthesia. Additionally, the child's inability to hear or speak made assessment of cooperation, airway evaluation, and response to commands exceedingly difficult, reinforcing the need for a carefully planned induction and emergence strategy.

The presence of a large intraparenchymal bleed early in life obscures the early inflammatory etiology. Hemorrhagic presentations are exceptionally uncommon in Rasmussen's encephalitis, with only isolated descriptions in literature. The initial bleed in this child resulted in cystic encephalomalacia, mass effect, and subdural extension. As the child grew, neuroimaging demonstrated characteristic signs of chronic hemispheric injury, including cortical thinning, gliosis, white matter loss, cortical laminar necrosis, and progressive ventricular dilatation. Tractography revealed profound disruption of right-sided corticospinal and association fibers, as well as Wallerian degeneration extending into the ipsilateral cerebral peduncle. These findings indicated ongoing hemispheric degeneration that far exceeded the expected sequelae of a static hemorrhagic insult, thereby suggesting an additional pathological process.

These structural and functional deficits directly influence anaesthetic drug responses and airway management. Chronic use of multiple antiepileptic drugs alters hepatic enzyme activity, often increasing the metabolism of anaesthetic agents and neuromuscular blockers. Moreover, maintaining seizure protection intra-operatively becomes a priority, requiring the avoidance of agents that lower seizure threshold. The evolution of seizures further supported a progressive inflammatory etiology rather than a purely structural post-hemorrhagic epilepsy. The recurrent focal seizures continued despite

multiple antiepileptic medications, fitting the profile of medically refractory epilepsy (11).

The electroencephalography pattern was also notable. Persistent hemispheric asymmetry, attenuation of right hemispheric background activity, and epileptiform discharges predominantly over the opposite hemisphere suggested functional reorganization a phenomenon frequently reported in advanced Rasmussen's encephalitis when the affected hemisphere becomes electrically silent. The mismatch between imaging pathology and electrophysiological localization is an important diagnostic clue in unihemispheric destructive processes (12). For anaesthesiologists, this silent hemisphere implies that inadequate anaesthetic depth may not manifest through typical EEG changes, necessitating careful clinical monitoring and avoidance of hyperventilation-induced alkalosis, which can precipitate seizures.

Advanced MRI sequences provided further insight. The absence of right-sided corticospinal tracts, superior longitudinal fasciculus, cingulum, and callosal projections reflected widespread axonal loss. Right frontal and temporal atrophy with gliosis and hemosiderin deposition indicated a chronic inflammatory destructive process superimposed on the initial hemorrhagic event. These patterns are highly consistent with Rasmussen's encephalitis, especially in cases with prolonged disease duration (13). Such extensive structural abnormalities further explain the difficulty in airway assessment and unpredictable respiratory mechanics during induction and extubation. Histopathology remains the most definitive diagnostic modality. Findings in this child microglial nodules, neuronal loss, astrocytic proliferation, chronic perivascular lymphocytic cuffing, and diffuse inflammatory infiltrates represent the classic triad described in Rasmussen's encephalitis. The absence of granulomas, infectious organisms, or vasculitis further supported a primary autoimmune T-cell-mediated encephalitis rather than secondary etiologies (14).

Functional hemispherotomy is considered the most effective treatment for drug-resistant seizures arising from a severely damaged hemisphere. The child's complete seizure freedom after surgery supports the success of disconnection strategies in Rasmussen's encephalitis when the affected hemisphere is no longer salvageable. The stability of pre-existing hemiparesis after surgery is expected, given that the disconnected hemisphere had already lost functional capacity (15). For future procedures under general anaesthesia, such as dental surgery in this case, prior hemispherotomy necessitates careful monitoring of cerebral perfusion,

controlled ventilation, and vigilance for irregular breathing patterns during emergence.

This case underscores the need for clinicians to consider Rasmussen's encephalitis even when early imaging suggests structural injury. Unexplained progressive hemispheric atrophy, drug-resistant seizures, and characteristic histopathology are key markers. Early recognition and prompt surgical management can significantly improve quality of life and long-term neurological outcomes (16).

#### CONCLUSION

Rasmussen's encephalitis is an uncommon but severe unihemispheric inflammatory disorder of childhood, and its diagnosis becomes particularly challenging when early presentations resemble structural lesions such as intracerebral hemorrhage. Anaesthetic management of a pediatric patient with Rasmussen's encephalitis undergoing hemispherotomy is particularly challenging due to refractory seizures, altered neurodevelopment, chronic antiepileptic drug therapy, and the physiological impact of major neurosurgical intervention. Optimal perioperative care requires meticulous preoperative evaluation, continuation of antiepileptic medications, judicious selection of anaesthetic agents with minimal effect on seizure threshold, and strict maintenance of cerebral perfusion and intracranial pressure. Anticipation of massive blood loss, fluid and electrolyte disturbances, and the need for postoperative ventilatory and neurological support is essential. A coordinated multidisciplinary approach is crucial in ensuring safe anaesthetic conduct and favorable neurological outcomes

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