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Case Report

A rare and challenging case of Rasmussen's encephalitis: a pediatric case report

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ABSTRACT

Rasmussen's encephalitis (RE) is a rare, chronic, and progressive neurological disorder characterized by unilateral hemispheric inflammation, intractable seizures, and neurological decline, primarily affecting children under 10 years old. This case report describes a 9-year-old male child born to consanguineous parents presented with a 9-month history of progressive myoclonic jerks, generalized tonic-clonic seizures (GTCS), and right-sided hemiparesis. Despite undergoing left temporo-parieto-occipital craniotomy and aggressive pharmacological management with antiepileptics, immunosuppressants, and corticosteroids, the patient developed super-refractory status epilepticus. Diagnostic imaging revealed left hemispheric atrophy, and cerebrospinal fluid analysis ruled out infections, leading to a diagnosis of Rasmussen'sencephalitis with drug resistant epilepsy. The patient underwent left vertical thalamic functional hemispherotomy, resulting in reduced seizure frequency, improved motor function, and decreased inflammatory markers postoperatively. This case underscores the challenges in managing RE, emphasizing the importance of early diagnosis, a multidisciplinary approach, and timely surgical intervention and challenging medication management.

Keywords: Rasmussen'sencephalitis, Epilepsia partialis continua, Hemispherotomy, Generalized tonic-clonic seizures, Hemiplegia-hemi convulsion-epilepsy syndrome

INTRODUCTION

Rasmussen's Encephalitis is a rare, chronic inflammatory condition of the brain mostly affecting children and involving one cerebral hemisphere that was first described by Rasmussen et al.[1] It is a progressive disease characterised by unilateral hemispheric atrophy, drug-resistant focal epilepsy, progressive hemiplegia, focal intractable seizures, Epilepsia Partialis Continua (EPC) and worsening neurological deficits.[2] The annual global incidence is estimated to be about 2.4 cases per 10 million people under 18 years.[3] It is common in children under the age of 10 with average age at disease onset around 6 years.[4] According to research, pediatric Rasmussen's encephalitis may have had a hereditary predisposition to autoimmune conditions. The most characteristic representation of PRE is progressive cerebral atrophy and epilepsia partialis continua (EPC). If remains untreated, the risk of permanent brain damage is significantly high.[5] The 3 clinical stages that have been proposed that are, the prodromal stage lasts an average of 7.1 months (range: 0 months to 8.1 years) and is characterized by low seizure frequency and mild hemiparesis. The acute stage lasts for eight months on average and is marked by frequent seizures. If the dominant hemisphere is affected, the neurological symptoms manifest as progressive hemiparesis, hemianopia, cognitive decline, and aphasia. The residual stage is the final stage, characterized by irreversible damage and fewer seizures than the acute stage.[6,7]

Various etiological hypotheses had been proposed about Rasmussen's Encephalitis. One prominent theory involves cell-mediated immunity, particularly T-cell-mediated immune responses targeting neurons and astrocytes.[8,9] The cell-mediated hypothesis is further reinforced by a recent study by Granata et al, which proposes that cytotoxic T cells may target a viral protein

present in both neurons and astrocytes.[10] Another theory supports an autoimmune mechanism. In 1994, Rogers et al. identified antibodies against the glutamate receptor (GluR3) in RE patients, implicating autoimmunity in the disease. Additionally, viral triggers such as herpes simplex virus (HSV) and cytomegalovirus (CMV) have been linked to RE, suggesting a potential role of viral infections in initiating or exacerbating the immune response.[11] MRI of the brain has become a key tool for diagnosing and monitoring Rasmussen's encephalitis.[12] Serial MRIs usually demonstrate the progression of signal alterations, hyperintensities and brain atrophy. F-FDG PET reveals widespread unilateral cerebral hypometabolism, which can appear even when MRI shows minimal atrophy.[13]

Patients with Rasmussen's encephalitis exhibit a broad range of EEG abnormalities, which are frequently associated with clinical progression. However, there are no unique EEG patterns that can differentiate Rasmussen's encephalitis from other causes of focal epilepsy.[14] Epileptiform abnormalities are common and often progress to electrographic seizures, although epilepsia partialis continua may not always show visible ictal surface EEG activity. A brain biopsy is not necessary for all patients, as the diagnosis can be confirmed based on other non-invasive criteria. Brain biopsy, in late disease stages, shows a spongy degeneration, due to the coalescence of multifocal neuronal loss cerebrospinal fluid (CSF) analysis is usually normal in most cases, and there are no specific biomarkers available.[15]

However, CSF analysis may reveal elevated protein levels and/or an increased cell count.[16] At least two sequential clinical examinations or MRI studies are needed to meet the respective criteria, Numerous parenchymal macrophages, B-cells, plasma cells, or viral inclusion bodies exclude the diagnosis of RE (Table 1). Treatment strategies for Rasmussen's encephalitis (RE) vary depending on the patient and the stage of the disease, treatment options are selected according to need of patient. There is limited evidence and data to determine the most effective treatment approach for RE.[18] In medical treatment, corticosteroids are frequently used and have demonstrated a strong and effective response in managing the condition.

Similarly, intravenous immunoglobulin (IVIG) and plasmapheresis have been utilized and shown to be effective for short-term improvement, though they are generally insufficient for achieving long-term outcomes.[20] In comparison to medical treatments, surgical hemispherotomy is often regarded as a more effective and widely accepted option for Rasmussen's encephalitis (RE). It is highly effective in eliminating seizures and is considered one of the modern treatment approaches for RE.[18] The surgical approach involves a tailored procedure, such as hemispherotomy, to remove the affected hemisphere. It is the only treatment with the potential to effectively control seizures and prevent from further mental deterioration in patients with Rasmussen's encephalitis.[21]

CASE REPORT

A 9-year-old male child with weight of 26kgs, born to consanguineous parents, presented with a 9-month history of progressive seizures characterized by myoclonic jerks, upward eyes rolling, and generalized tonic-clonic seizures (GTCS). According to his mother, the episodes typically began with jerking of right upper limb which spread to lower limb progressing to loss of consciousness.

He experienced two episodes of generalized tonic clonic seizures (GTCS) each characterized by a starring look, clonic movements in his right limbs. At this point, he became unresponsive with rolling of the eyeballs and drooling of saliva with each episode lasting about one minute. There was no associated fever, tongue biting, or spontaneous urination. The patient had no significant antenatal, perinatal, or postnatal history, nor a family history of epilepsy.

Fifteen days prior to presentation, the patient underwent "Neuronavigation-guided left temporo-parieto-occipital craniotomy with left anterior temporal lobectomy, amygdalohippocampectomy, and parietal lobectomy" for refractory GTCS in other medical facility. Postoperatively, he was maintained on a regimen of Tacrolimus 0.25 mg OD, Carbamazepine 300 mg BD, Levetiracetam 150 mg BD, Lacosamide 50 mg BD. However, 10 days after surgery, he experienced recurrent seizures with jerky movements, progressing to status epilepticus.

During the present admission on examination, the patient had a glasgow coma scale (GCS) of 13/15, with reduced power (4/5) in the right upper and lower limbs. Vital signs were stable, and oxygen saturation was 95% on room air. Laboratory investigations, including complete blood count, metabolic profile, and cerebrospinal fluid (CSF) analysis, were unremarkable except for an elevated C-reactive protein (CRP) level of 94.5 mg/dl,

Haemoglobin level of 10.3 g/dl. His pulse rate was 98 beats/minute, blood pressure was 100/70 mmHg, and respiratory rate was 24 breaths per minute. A CT scan revealed left hemispheric atrophy. This case highlights the challenges in managing refractory epilepsy in paediatric patients with consanguineous backgrounds, particularly in the context of post-surgical complications and limitations of aggressive pharmacological intervention.

Patient journey in hospital

A 9-year-old boy presented with recurrent episodes of myoclonic jerky movements and upward rolling of his eyes, each lasting approximately one minute. His mother reported that these episodes had been occurring for the

past nine months. The patient was previously diagnosed with generalized tonic-clonic seizures (GTCS) and underwent surgery 15 days before at another medical facility. However, immediately following the surgery, the frequency of his seizure episodes increased, characterized by jerky movements and a staring look.

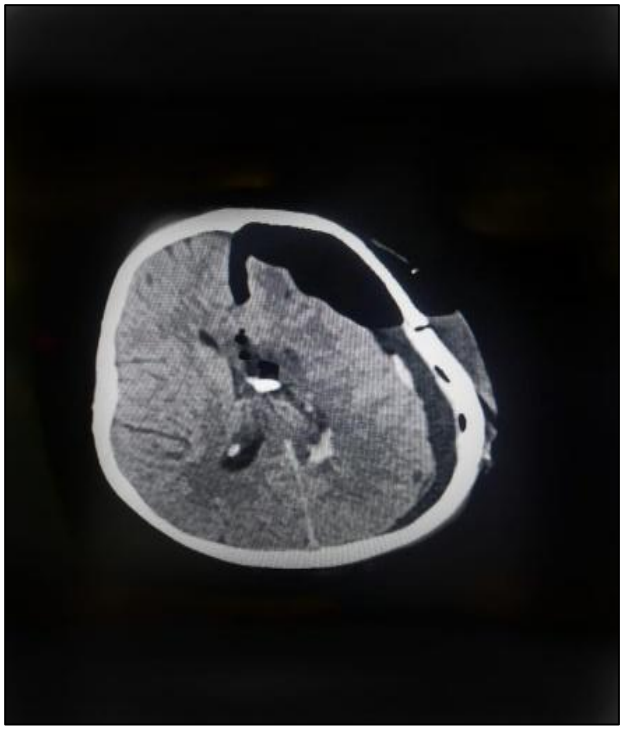

Figure 1: Post operative MRI indicating a left functional hemispherotomy.

The patient was subsequently admitted to the Neurology department of our tertiary care hospital, where he exhibited persistent symptoms, including myoclonic jerky movements in his right limbs and upward rolling of his eyes, with each episode lasting about one minute. Due to the severity of his condition, he was admitted to the pediatric intensive care unit (PICU). He was prescribed a combination of antiepileptic drugs, immunosuppressants, corticosteroids, and antibiotics to control seizures and prevent potential infections. He was started on Tacrolimus 0.25 mg PO, along with other antiepileptics such as Clobazam, Carbamazepine orally and IV Methylprednisolone.

Despite the aggressive pharmacological intervention, the patient continued to experience frequent seizure episodes, with myoclonic jerky movements originating in his right upper limb and spreading to his right lower limb. Cerebrospinal fluid (CSF) analysis revealed no culture growth and were negative for infections, leading to the suspicion of autoimmune encephalitis.

Based on imaging findings and the patient's resistance to antiepileptic drugs, he was diagnosed with "RASMUSSEN'S ENCEPHALITIS WITH DRUG-RESISTANT EPILEPSY". This rare and severe neurological disorder is characterized by progressive inflammation of one hemisphere of the brain, leading to intractable seizures and neurological decline. Further management strategies, including potential immunotherapy (e.g., steroids, IVIG) and surgical interventions, were being considered before surgery.

Despite being treated with multiple antiepileptic medications and immunotherapy, the seizures remained uncontrolled and new onset of hypertension suspected to be contributed by Methylprednisolone. As a result, the patient eventually underwent a left functional hemispherotomy due to the refractory nature of seizures.

The patient was treated with antibiotics, antiepileptics, analgesics, dopamine agonists, and other supportive medications. Post operatively, the patient was awake and conscious (GCS 15/15) but experienced headache, fever and aphasia with right hemiplegia. Seizure frequency and overall function gradually improved, leading to the discontinuation of carbamazepine and a reduction in other antiepileptics dosage, with some skin issues treated by topical corticosteroid cream.

Table 1: Diagnostic criteria for Rasmussen's encephalitis.

	PART A (all of three)
Clinical features	Focal seizures, with or without EPC, and unilateral cortical deficit
EEG	Unihemispheric slowing with or without epileptiform activity and unilateral seizure onset
MRI	Unihemispheric focal cortical atrophy and at least one of the following: - Grey or white matter T2/FLAIR hyperintense signal, Hyperintense signal or atrophy of the ipsilateral cauda
	PART B (at least two of three)
Clinical features	EPC or progressive unilateral cortical deficits
MRI	Progressive unihemispheric focal cortical atrophy
Histopathology	T-cell dominated encephalitis with activated microglial cells (cerebral biopsy) and reactive astrogliosis

EEG-electroencephalogram, EPC-Epilepsia Partialis Continua, FLAIR-fluid-attenuated inversion recovery, MRI-magnetic resonance imaging.

Table 2: Preoperative management.

Drug	Dose	ROA	Freq	Duration in days	Indication
Carbamazepine	200 mg	PO	BD	D1-D10	Seizures
Lacosamide	50 mg	PO	BD	D1-D2	Seizures
Levetiracetam	750 mg	PO	BD	D1-D2	Seizures
Tacrolimus	0.25 mg	PO	OD	D2-D5	Immunosuppressant
Tacrolimus	0.5 mg	PO	OD	D6-D8	Immunosuppressant
5%IVIG	50 gm	IV (slow)	onflow	D2-D4	Immunomodulant
Methylprednisolone	500 mg	IV	OD	D3-D4	Steroid
Clobazam	10 mg	PO	OD	D3-D10	Seizures
Brivaracetam	75 mg	IV	BD	D3-D10	Seizures
Phenergan	25 mg	PO	BD	D4-D9	Insomnia
Syp.Triclofos	10 ml	PO	BD	D4-D9	Insomnia
Amlodipine	5 mg	PO	BD	D4-D8	Hypertension
Melatonin	3 mg	PO	OD	D3	Insomnia
CBD oil	1 ml	PO	BD	D4-D10	Adjunct for seizures
One time/STAT doses					
Nifedipine	5 mg	PO	STAT	D4	Anti-hypertensive
Clonazepam	0.25 mg	PO	OD	D1	Seizures
Lorazepam	2.5 mg	IV	STAT	D3	Seizures
Midazolam nasal spray	3 puffs	INH	In each nostril	D3	Seizures

PO: per oral; BD: twice daily; OD: once daily; IV: intravenous; STAT: immediately; D: Day

Table 3: Postoperative management.

Drug	Dose	ROA	Freq	Duration in days	Indication
Meropenem	1 gm	IV	TID	D11-D16	Antibiotic
Vancomycin	400 mg	IV	TID	D11-D16	Antibiotic
Ceftriaxone	1.5 gm	IV	BD	D13	Antibiotic
Brivaracetam	50 mg	PO	OD	D11-D24	Anti-epileptic
Carbamazepine	200 mg	PO	BD	D11	Anti-epileptic
Bromocriptine	1.5 mg	PO	BD	D14-D22	Post Encephalitis
Clobazam	10 mg	PO	OD	D11-D14	Encephalitis
Clonazepam	0.25 mg	PO	HS	D13-D15	Sedation
Melatonin	3 mg	PO	OD	D17-D23	Insomnia
Mometasone cream		L/A	HS	D13-D18	Rashes
Amlodipine	5 mg	PO	BD	D13-D18	Hypertension
Dexamethasone	2 mg	IV	TID	D13-D18	Cerebral edema
Tramadol	15 mg	IV	OD	D11-D13	Pain management
CBD oil	1 ml	PO	BD	D11-D18	Adjunct for seizures
Amitriptyline	10 mg	PO	OD (1/2tab)	D19-D24	Insomnia

PO: per oral; BD: twice daily; TID: thrice daily; OD: once daily; IV: intravenous; SOS: as needed; L/A: local application; S/C: subcutaneous; HS: at bedtime; D: Day

DISCUSSION

This case highlights the diagnostic and therapeutic challenges in managing Rasmussen's encephalitis (RE), a rare and severe form of drug-resistant epilepsy. The patient, a 9-year-old boy with consanguineous parents, presented with progressive myoclonic jerky movements and unilateral neurological deficits, consistent with the typical clinical course of RE. The most prevalent symptom of RE is progressive unidirectional loss of function, which was seen in our patient as well and this drug-resistant epilepsy progressed to super-refectory status epilepticus establishment despite aggressive pharmacological intervention. Rasmussen's encephalitis appears quite similar to hemiplegia-hemi convulsion-epilepsy syndrome (HHES). However, in our case, the distinguishing factors between RE and HHES were the radiological findings, the timing of symptom onset, and the clinical progression of the patient. Most individuals with HHES typically experience a seizure-free interval lasting months to years after their initial diagnosis before developing refractory seizures.[22] The medical treatment of Rasmussen's encephalitis is complex and not fully

understood. There are currently no FDA-approved medications specifically for treating Rasmussen's encephalitis (RE). Although immunotherapy might help in reducing further inflammatory damage, it is rarely efficacious alone in controlling seizures. The primary aim of antiepileptic therapy (AEDs) is to reduce the frequency and severity of seizures while also preventing neurological complications and preserving function, which is assessed through motor and cognitive performance. Although antiepileptics are primary choice of treatment, their effectiveness is frequently limited as seen in this case.

Based on the clinical stages in this case report, the patient likely exhibited the "acute stage" of Rasmussen's encephalitis, characterized by drug-resistant epilepsy, jerky movements on the right side of the body, frequent seizures, and neurological symptoms such as upward eye movement and hemiparesis. These clinical features align with the typical progression of the disease during the acute stage, which, if untreated, can advance to the residual stage marked by irreversible neurological damage.

On admission, our patient was prescribed on Carbamazepine 300mg, Lacosamide 50 mg, Levetiracetam 150 mg, Tacrolimus 0.25 mg, Brivaracetam 75 mg, Clobazam orally and IV Methylprednisolone 500 mg BD for 2 days and 5%IVIG 50gm over 3 days. (Table 2) The patient continued to experience frequent seizure episodes with myoclonic jerks (4 episodes in 30 minutes) and developed super-refractory status epilepticus.

Despite antiepileptic medications, patient continued to experience seizures and regained consciousness after one minute but had persistent headaches on the right side. Bilateral pupils were reacting to light. The Humpty Dumpty Scale-fall risk assessment tool for pediatrics showed total score >13 indicating high risk. Sudden spikes in blood pressure observed and recorded at 139/81 mmHg, with a pulse rate of 168 beats per minute, suggesting that the new onset hypertension may be induced by methylprednisolone. For which Amlodipine 5mg was prescribed and Nifedipine 5mg was later added for breakthrough spikes in blood pressure as needed. The IVIG didn't show any significant difference in seizure presentation.

Later, tacrolimus dose was increased to 0.5 mg and administered half an hour before meals. After which Tacrolimus (FK506) levels increased to 4.6ng/ml from 0.58ng/ml. Tacrolimus was held a day prior to surgery to minimize the risk of immunosuppression-related complications during the procedure. Patient continued to experience intermittent myoclonic jerks lasting for one-minute, occasional mild headache radiating to eye. This underscores the need for early consideration of surgical intervention in refractory cases. Parents were explained the need and consequences of hemispherotomy which includes aphasia, right hemiplegia while it offers the best chance of seizure control in patients with unilateral disease. Patient underwent with surgery left vertical thalamic functional hemispherotomy (Figure 1).

Postoperatively, patient treated with antibiotics, antiepileptics, opioid analgesics, dopamine agonist, benzodiazepines, antihypertensives, IV fluids, corticosteroid, sedatives (Table 3) On examination, patient was awake, conscious, GCS: 15/15, experienced intermittent headache, fever spikes 101°F, sleep disturbance, constipation and CRP levels decreased from 94.5 mg/dl to 16.1 mg/dl. Upon physical examination revealed aphasia, right hemiplegia while movement was noticed in the left limbs but showed gradual improvement in seizure frequency and overall function.

Carbamazepine was discontinued post operatively, while brivaracetam was gradually reduced by 25 mg BD every week. Clobazam was decreased by 2.5 mg every week before being discontinued due to decrease in seizure episodes. Development of dark discolouration on buttocks and blisters on right ear suspected to be drug induced reactions which are subsided with topical corticosteroid cream. The combination of the medications used may have contributed to these reactions. Medications like carbamazepine, vancomycin, and corticosteroids can cause such adverse effects, especially when given in combination.[23]

The strength in the right upper limb is rated 2/3, while the strength in the right lower limb is rated 3/3. On speech therapy evaluation, right upper motor neuron (UMN) facial palsy was observed and weakness was seen in oromotor activities. This affected articulation leading to dysarthria. The Patient was advised physiotherapy, after which his condition gradually improved and was discharged after a month in a stable condition with specific advice and discharge medications.

The patient's gradual recovery, marked by improved CRP levels, reduced seizure frequency, and partial restoration of motor function, highlights the importance of a multidisciplinary approach in managing RE. This case emphasizes the critical role of early diagnosis, aggressive medical management, and timely surgical intervention in treating RE. Furthermore, it highlights the need for further research into the pathophysiology and treatment of this devastating condition, particularly in pediatric patients with consanguineous backgrounds.

On discharge, patient was advised to use Tab. Aceclofenac+paracetamol (100 mg+325 mg) BD for pain, Tab.Brivaracetam (25 mg) BD for seizures prophylaxis, Tab.Melatonin (3 mg) OD for insomnia, Tab. Amitriptylline (10 mg) OD for insomnia, Tab. Lansoprazole (30 mg) OD for acid control, Mometasone topical cream (0.1%w/w) OD for skin care.

CONCLUSION

Rasmussen's encephalitis (RE) is a rare, chronic, and progressive neurological disorder characterized by unilateral hemispheric inflammation, intractable seizures, and neurological decline, primarily in children under 10 years old. This case highlights the diagnostic and therapeutic challenges of RE in a 9-year-old boy with consanguineous parents, who presented with drug-resistant epilepsy and progressive unilateral deficits.

Despite aggressive pharmacological intervention, which include antiepileptic drugs, immunosuppressants, and corticosteroids. The patient progressed to super-refractory status epilepticus, necessitating surgical intervention with left vertical thalamic functional hemispherotomy. Postoperatively, the patient showed gradual improvement, with reduced seizure frequency, decreased CRP levels, and partial restoration of motor function, highlighting the importance of a multidisciplinary approach in managing RE. RE should be considered as a possible diagnosis in patients who present with intractable seizures along with investigations such as MRI and electroencephalograms. As the aetiology is largely unknown, the prognosis remains variable, a combination of medical and surgical strategies offers the best chance for seizure control and functional preservation.

As multiple drugs were used to manage various conditions, the risk of overdosing and drug-drug interaction is significant. Active involvement of clinical pharmacy services is essential to identify and minimize these risks, ensuring safer and effective patient care.

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