From Super-Refractory Epilepsia Partialis Continua to Full Recovery: Serial EEG Normalization and Two-Year Seizure Freedom After Triple-Anesthetic Therapy

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ABSTRACT

Background

Super-refractory status epilepticus (SRSE) is a devastating neurological emergency with high morbidity and mortality, and fewer than 10% of survivors achieve full recovery (mRS 0). Epilepsia partialis continua (EPC) as a clinical manifestation of SRSE is rare in adults. Long-term seizure remission after SRSE, especially with documented serial EEG normalization, is seldom reported.

Case

A 49-year-old woman with no prior comorbidities presented with focal motor seizures of the left upper limb that evolved into EPC and SRSE. The ictal EEG revealed right central (C4) epileptiform discharges correlating with contralateral motor activity and secondary left temporal spread. Despite treatment with multiple antiseizure medications and escalation to triple-anesthetic therapy (midazolam, propofol, thiopental), seizures persisted for six weeks. Complications included suspected propofol infusion syndrome, hypernatremia, stress-related gastrointestinal bleeding, multidrug-resistant pneumonia and bacteremia (K. pneumoniae, P. aeruginosa, MRSA, A. baumannii), thiopental-induced agranulocytosis, and paroxysmal atrial fibrillation. Seizures gradually abated, and she was successfully extubated. Serial EEGs showed progressive resolution, from hemispheric polyspike discharges to normalized alpha rhythm, paralleling recovery. Follow-up MRI revealed resolution of cortical hyperintensities with residual right hippocampal atrophy. Over two years, antiseizure medications were tapered to valproate monotherapy. She remained seizure-free with normal EEGs and full functional independence (mRS 0).

Conclusion

This case illustrates a rare trajectory of adult-onset EPC evolving into SRSE with complete neurologic and electrophysiologic recovery. It underscores the value of serial EEG monitoring, vigilant critical care, and long-term follow-up in achieving remission even in resource-limited settings.

Keywords: *Epilepsia partialis continua; Super-refractory status epilepticus; EEG normalization; Triple-anesthetic therapy; Hippocampal atrophy; Adult-onset epilepsy*

INTRODUCTION

Status epilepticus (SE) is a neurological emergency associated with substantial morbidity and mortality. A small but severe subset progresses to super-refractory status epilepticus (SRSE), defined as seizure activity that continues or recurs ≥24 hours after the onset of anesthetic therapy or upon withdrawal of anesthetics.[1] SRSE is rare, with an estimated incidence of approximately 0.7 per 100,000 persons annually, and carries an in-hospital mortality rate ranging from 24% to 40%.[2,3] Even among survivors, complete recovery without disability (modified Rankin

Scale 0) occurs in fewer than 10–15% of cases.[2,4]

Epilepsia partialis continua (EPC), a focal motor seizure characterized by continuous or near-continuous clonic activity, is itself uncommon, accounting for less than 3% of all epilepsy cases.[12] It typically occurs in children or young adults and is most often associated with structural, infectious, or autoimmune cortical lesions.[12,13] Adult-onset EPC, particularly when evolving into SRSE, is exceedingly rare.

Given these epidemiologic patterns, the present case of a previously healthy 49-year-old woman who developed EPC progressing to SRSE, with documented EEG evolution, multimodal anesthetic therapy, multiple systemic complications, and eventual full recovery, is remarkable. Documenting such trajectories is clinically relevant not only for their rarity but also for the insights they offer into mechanisms of cortical recovery, management challenges in low- and middle-income countries (LMICs), and the prognostic spectrum of SRSE.[8,11]

CASE PRESENTATION

A 49-year-old woman with no prior comorbidities presented with new-onset focal motor seizures. Two weeks before admission, she noted pricking sensations in her left upper extremity. Six days later, she developed rigidity of the extremities with upward eye deviation, without incontinence or loss of consciousness. Initially treated with phenytoin and valproate at a peripheral hospital, she developed continuous twitching of the left upper limb and recurrent hemifacial jerks, consistent with epilepsia partialis continua (EPC). Despite benzodiazepines, seizures persisted, prompting referral to our tertiary center.

On arrival, she had ongoing focal motor seizures and was eventually intubated for airway protection. Continuous EEG revealed right central (C4) epileptiform discharges time-locked with contralateral left upper-limb myoclonus and secondary left temporal polyspike spread, consistent with evolving hemispheric involvement.

She progressed to super-refractory status epilepticus (SRSE), unresponsive to multiple antiseizure medications: levetiracetam, valproate, topiramate, phenobarbital, and intermittent benzodiazepines. Anesthetic therapy was escalated sequentially: midazolam (up to 0.4 mg/kg/h, Days 1–7), propofol (up to 5 mg/kg/h, discontinued Day 10 for suspected propofol infusion syndrome with rising CK-MB and AST), and thiopental (initiated Day 12, titrated to burst suppression until Day 28, stopped for agranulocytosis). Seizures persisted for several weeks before gradually abating after approximately six weeks of intensive care.

The ICU course was complicated by propofol infusion syndrome, stress-related upper gastrointestinal bleeding, multidrug-resistant pneumonia and bacteremia (K. pneumoniae, P. aeruginosa, MRSA, A. baumannii), paroxysmal atrial fibrillation managed with amiodarone and metoprolol, and thiopental-induced agranulocytosis. Despite these systemic complications, she demonstrated gradual neurologic recovery and was successfully extubated.

Neuroimaging initially revealed patchy areas of diffusion restriction in the right fronto-parietal cortical and subcortical regions on DWI/FLAIR, consistent with subacute infarcts (Figure 1). The follow-up MRI revealed complete resolution of cortical signal abnormalities and residual right hippocampal atrophy, consistent with post-ictal structural remodeling (Figure 2).

Serial EEGs documented progressive electrophysiologic evolution, from early right-central and temporal polyspike discharges to anesthetic-induced burst suppression, reorganization of background activity, and eventual normalization, mirroring her clinical trajectory (Table 1).

Figure 1. Initial MRI brain (Stroke Protocol).

(A) Axial DWI showing patchy hyperintense signal in the right fronto-parietal cortex. (B) ADC map demonstrating corresponding high signal compatible with subacute infarction. (C) Axial FLAIR depicting hyperintense cortical/subcortical

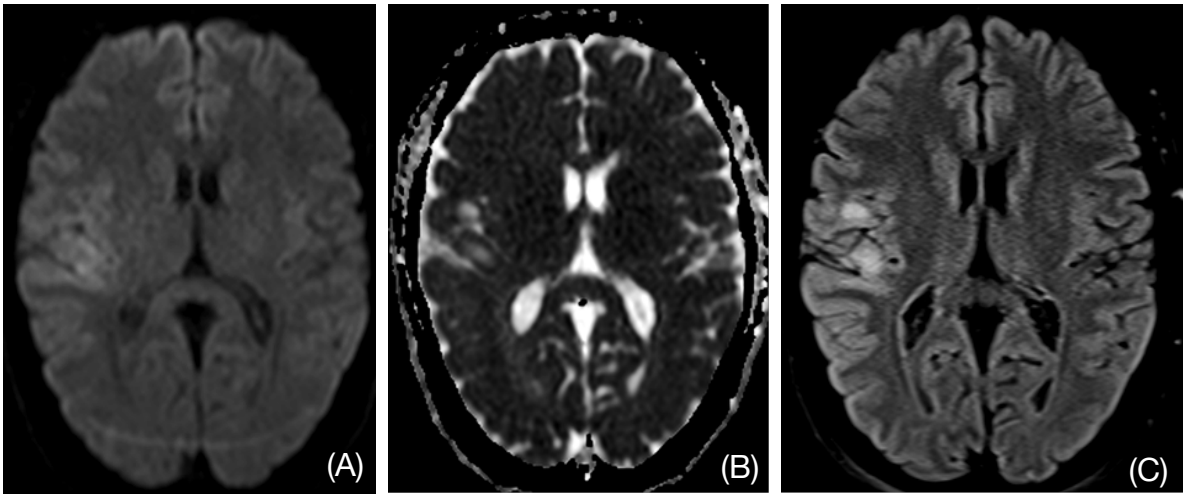


Figure 2. Follow-up MRI brain (Seizure Protocol).
(D) Follow up axial FLAIR sequence showing absence of prior right fronto-parietal cortical hyperintensities and diffuse cerebello-cerebral volume loss. (E) Coronal Flair sequence revealing atrophy of the right hippocampus (arrow)

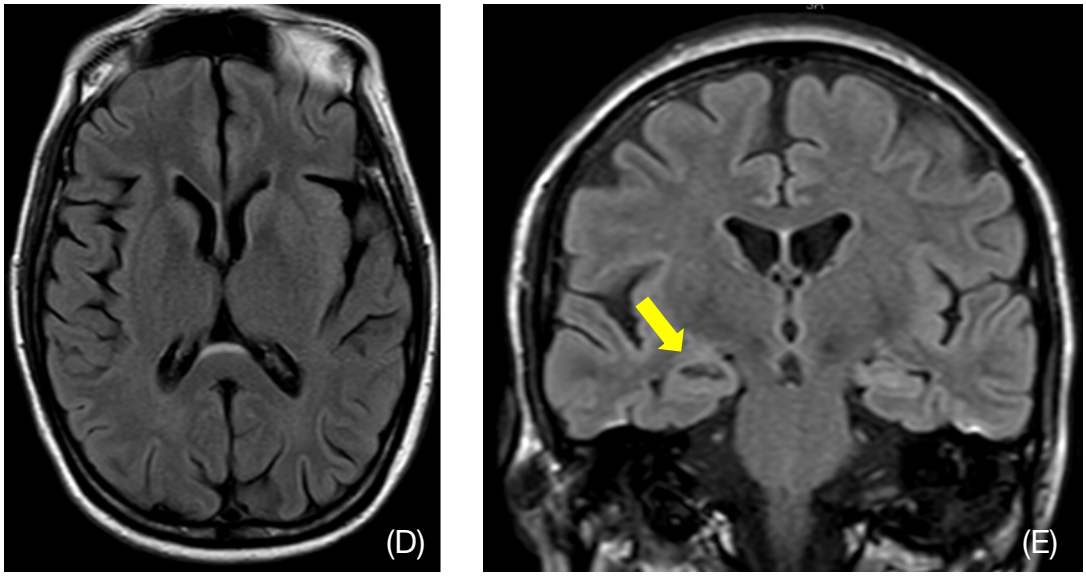
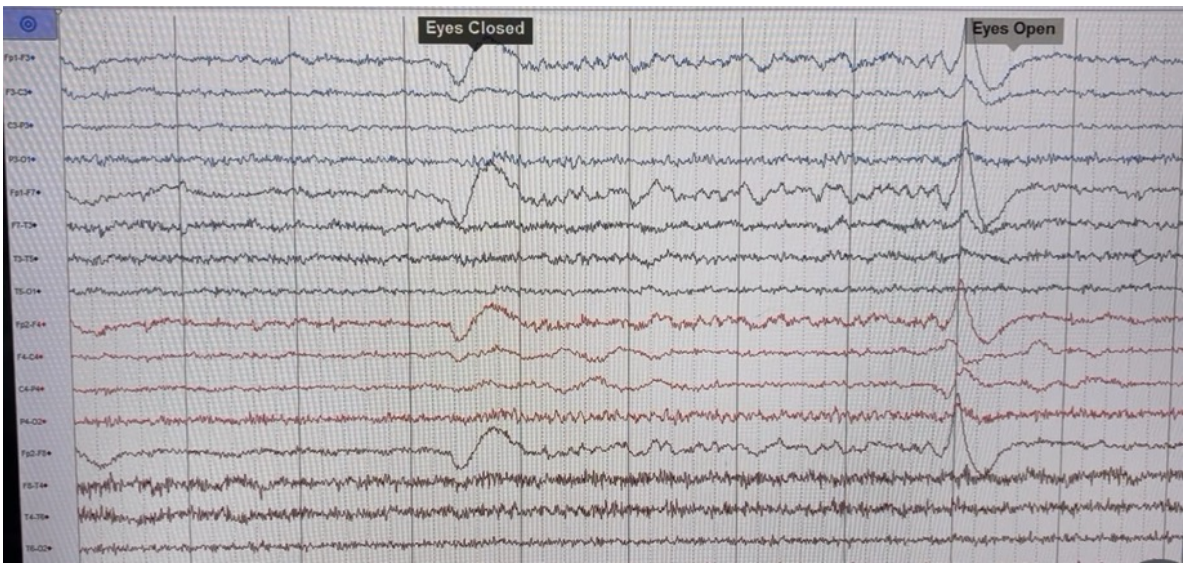


Table 1. Evolution of EEG Findings and Clinical Correlation

Date / Phase	EEG Findings	Clinical Status / Therapy	Interpretation
Feb 2023 (Initial ictal)	Right central (C4) epileptiform discharges with left upper-limb myoclonus; intermittent left temporal polyspikes with hemispheric spread; diffuse slowing.	EPC with persistent left UE twitching and hemifacial automatisms; unresponsive to phenytoin/valproate/ benzodiazepines.	Active ictal cortex with central focus correlating to contralateral motor signs; secondary temporal recruitment.
Mar 2023 (Deep sedation)	**Burst-suppression** on thiopental; no sustained focal discharges under anesthesia.	Triple anesthetic (midazolam, propofol - stopped for suspected PRIS, thiopental to burst suppression) + multi-ASM regimen.	Seizure suppression by anesthetic coma; cortical excitability pharmaco-controlled.
May 2023 (Post-anesthetic recovery)	Diffuse theta with re-emerging posterior alpha; no epileptiform discharges.	Awakening, no overt clinical seizures; beginning ventilatory wean and hemodynamic stabilization.	Early cortical recovery with improving background organization.
Mar 2024 (Outpatient)	Symmetric alpha rhythm (10 Hz); no epileptiform discharges.	Seizure-free; ASMs consolidating toward monotherapy.	EEG normalization consistent with clinical remission.
Dec 2024 (Latest follow-up)	Normal background; sustained absence of epileptiform activity.	mRS 0, seizure-free; on valproate monotherapy.	Sustained electro-physiologic remission.

Figure 2. Follow-up EEG (double-banana montage) demonstrating complete electrophysiologic normalization.
Epoch recorded during relaxed wakefulness showing symmetric, well-formed 10 Hz posterior dominant alpha rhythm reactive to eye opening and closing. No spikes, sharp waves, or slowing are seen over the previously active right central (C4) region, consistent with full recovery.



Follow-up EEGs in March and December 2024 confirmed fully normalized background activity, with well-formed 10 Hz posterior alpha rhythm, preserved reactivity, and no epileptiform discharges over the previously active C4 region (Figure 2).

She was discharged on valproate, topiramate, and levetiracetam, which were later tapered to valproate monotherapy. At her most recent follow-up in October 2025, she remained seizure-free for nearly two years, with normal neurologic examination, normal EEGs, and complete functional recovery (mRS 0).

This case represents a rare clinical course, progression from EPC to SRSE requiring triple-anesthetic therapy, complicated by multiple systemic insults, yet culminating in structural and electrophysiologic recovery.

DISCUSSION

This case underscores an uncommon progression of epilepsia partialis continua (EPC) in a middle-aged adult, evolving into super-refractory status epilepticus (SRSE) with complete structural and electrophysiologic recovery.

SRSE is defined as status epilepticus that persists or recurs ≥24 hours after the onset of anesthetic therapy, including recurrence upon anesthetic withdrawal.[1] It carries significant morbidity and mortality: in a large review of 596 cases, only 35% of patients returned to baseline function, while 35% died, and 13% remained severely disabled.[1] More recent meta-analyses report an in-hospital mortality rate of 24% and favorable functional outcomes (mRS 0–2) in fewer than 15% of survivors.[2] Against this backdrop, our patient's two-year seizure freedom, serial EEG normalization, and mRS 0 recovery represent an exceptional outcome.
In this case, the ictal EEG demonstrated right central (C4) epileptiform discharges correlating with contralateral left upper-limb myoclonus and secondary left-temporal spread. This pattern reflects a perirolandic epileptogenic focus with hemispheric propagation via cortico-cortical networks, a phenomenon described in prolonged focal motor status epilepticus.[3]

Management required escalation to triple-anesthetic therapy; midazolam, propofol, and thiopental. Although evidence is limited, this approach aligns with reported SRSE management strategies that rely heavily on expert consensus, retrospective cohorts, and small case series rather than randomized trials.[4] Adjunctive measures such as ketamine, immunotherapy, or ketogenic diet have been used in rescue settings, though evidence of benefit remains limited.[5,6]

Follow-up MRI demonstrated resolution of cortical hyperintensities and right hippocampal atrophy, consistent with excitotoxic neuronal injury after prolonged seizure activity. Hippocampal volume loss is a recognized sequela of status epilepticus and is thought to result from glutamate-mediated neurotoxicity and subsequent network reorganization.[7,8] Serial imaging studies confirm that hippocampal atrophy may continue to progress even after clinical resolution, underscoring the vulnerability of mesial temporal structures to prolonged seizures.[9]

Equally notable was the electrophysiologic recovery documented through serial EEGs, progressing from right-hemispheric polyspikes and burst suppression to a fully symmetric 10 Hz alpha rhythm without epileptiform discharges. Such complete normalization is rarely observed after SRSE; most survivors exhibit residual slowing or interictal abnormalities despite clinical improvement.[1,2] This suggests that, under optimal metabolic and neurocritical conditions, cortical networks can undergo functional reorganization and restore physiologic oscillatory balance.

The patient's ICU course was complicated by propofol infusion syndrome, thiopental-induced agranulocytosis, and multidrug-resistant infections, all of which are recognized adverse events contributing to poor outcomes in SRSE cohorts.[10] Nonetheless, her gradual neurologic improvement and eventual extubation emphasize that sustained and closely monitored supportive care can outweigh these risks when delivered meticulously.

Treatment in a low- to middle-income tertiary center imposed additional constraints, limited continuous EEG availability, delayed MRI, and absence of advanced adjuncts such as ketogenic therapy or neuromodulation. Despite this, the patient achieved complete remission. Reports from resource-limited environments confirm that rigorous, prolonged supportive management alone can yield meaningful recoveries in SRSE when guided by physiologic monitoring and multidisciplinary coordination.[11]

In summary, this case represents a rare adult-onset EPC originating from the right central cortex and progressing to SRSE, followed by full clinical, radiologic, and electrophysiologic recovery. The correlation between structural resolution, serial EEG normalization, and functional outcome underscores the potential for cortical reorganization after prolonged epileptic insult and highlights that favorable prognoses remain possible even in resource-constrained settings.

LIMITATIONS

Our evaluation was limited by the absence of early imaging and continuous electrophysiologic data, which might have clarified the temporal evolution of cortical injury and epileptogenic focus. The initial MRI and EEG during ictus were unavailable for review due to technical constraints, and no cerebrospinal fluid or autoimmune studies were performed because the clinical picture was inconsistent with autoimmune encephalitis. Furthermore, treatment occurred in a resource-limited tertiary center lacking continuous EEG monitoring, urgent MRI access, and advanced adjunctive therapies such as ketogenic diet or neuromodulation.

Despite these limitations, the case remains notable for its serial EEG documentation, demonstrating progressive normalization, and for the seizure protocol MRI confirming cortical resolution with hippocampal atrophy. These findings support a well-documented recovery trajectory and highlight the potential for favorable outcomes even in settings where diagnostic and therapeutic modalities are constrained.[12]

CONCLUSION

This case illustrates an exceptionally rare trajectory of adult-onset epilepsia partialis continua (EPC) evolving into super-refractory status epilepticus (SRSE) with full clinical, radiologic, and electrophysiologic recovery. Against the backdrop of global SRSE outcomes, with fewer than 15% of patients achieving functional independence,[1,2] the patient's two-year seizure freedom and complete EEG normalization represent an extraordinary recovery pattern. This report underscores that even in resource-limited settings, aggressive and sustained neurocritical care can yield favorable outcomes when guided by vigilant physiologic monitoring and multidisciplinary management. The sequential normalization of EEG and resolution of cortical hyperintensities, with only residual hippocampal atrophy, provide objective evidence of cortical reorganization after prolonged seizure activity.[8–10] Beyond its rarity, this case exemplifies the potential for meaningful neurologic recovery in SRSE and reinforces the importance of persistence, individualized treatment, and comprehensive follow-up in achieving durable remission and restoring functional independence.[12]

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