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Seizure Freedom After Focal Epilepsy Surgery in a Child With Tuberous Sclerosis Complex and Coexisting Suspected Subependymal Giant Cell Astrocytoma: A Case Report

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Abstract

We report a three-year-old girl with tuberous sclerosis complex and drug-resistant focal epilepsy who achieved clinical seizure freedom after left frontal resection. Seizures began at three months of age and continued despite multiple antiseizure medications, occurring approximately 11-16 times daily. Medical records reported a TSC1 mutation, although the original genetic report was unavailable. Preoperative examination documented developmental delay, absence of spoken language and independent walking, and right-sided hemiparesis graded 4/5. Magnetic resonance imaging demonstrated multiple cortical and subcortical lesions, a prominent left frontal abnormality, and a separate right lateral ventricular lesion considered radiologically suggestive of a subependymal giant cell astrocytoma. Presurgical video-electroencephalography recorded multiple focal motor seizures and subclinical seizure patterns associated with left frontal activity at F3-C3, together with interictal discharges in the same region. Following multidisciplinary assessment, microsurgical resection was performed using neuronavigation, intraoperative neurophysiological monitoring, and bipolar stimulation before resection. Intraoperative electrocorticography was not performed. The subsequent neurosurgical record reported the histological diagnosis of the resected frontal specimen as giant cell astrocytoma, grade I; the original pathology report and slides were unavailable for independent review. The separate ventricular lesion remained unresected. At 18-month follow-up, no clinical seizures had been reported since surgery while antiseizure treatment continued. Independent walking and syllabic speech had emerged, although developmental impairment remained. Right-sided hemiparesis was graded 4/5 both before surgery and at follow-up; the intervening postoperative motor course was incompletely documented. Follow-up electroencephalography demonstrated epileptiform activity despite clinical seizure freedom. This case illustrates a favorable seizure outcome after localized surgery in a child with multiple intracranial lesions, while emphasizing the distinction between clinical seizure control, electroencephalographic findings, and reported pathological diagnoses.

Categories: Neurology, Pediatrics, Neurosurgery
Keywords: drug-resistant epilepsy, frontal lobe epilepsy, neuronavigation, pediatric epilepsy surgery, seizure freedom, subependymal giant cell astrocytoma, tuberous sclerosis complex

Introduction

Tuberous sclerosis complex (TSC) is an autosomal dominant multisystem disorder associated with pathogenic variants in TSC1 or TSC2, which encode hamartin and tuberin, respectively. Impaired regulation of the mechanistic target of rapamycin (mTOR) pathway contributes to abnormal cell growth and the development of lesions in multiple organs, including the brain, skin, kidneys, heart, and lungs. Neurological manifestations include epilepsy, developmental impairment, and TSC-associated neuropsychiatric disorders [1].

The characteristic intracranial lesions include cortical tubers, subependymal nodules, and subependymal giant cell astrocytomas (SEGAs). Cortical tubers are developmental malformations that disrupt cortical architecture and may contribute to epileptogenic networks. Subependymal nodules are hamartomatous lesions along the ventricular walls, whereas SEGAs are typically slow-growing tumors located near the foramen of Monro that may obstruct cerebrospinal fluid circulation and cause hydrocephalus. Distinguishing these lesions is essential because identifying a target for epilepsy surgery and determining whether a ventricular lesion requires treatment involve different clinical considerations [1,2].

Epilepsy affects more than 70% of individuals with TSC, and more than half develop medically refractory

epilepsy [2]. The International League Against Epilepsy defines drug-resistant epilepsy as failure of adequate trials of two tolerated, appropriately selected and administered antiseizure medication regimens, used alone or in combination, to achieve sustained seizure freedom [3]. Previous systematic reviews summarized by Nie et al. reported seizure freedom in approximately 55%-60% of patients undergoing resective surgery for TSC-associated epilepsy [2]. These findings support timely surgical evaluation, including in young children [4]. However, multiple structural lesions and potentially distributed epileptogenic networks complicate surgical planning, which requires assessment of seizure semiology, ictal and interictal video-electroencephalographic findings, and structural imaging [2,4].

We report a three-year-old girl with TSC and multiple intracranial lesions who achieved clinical seizure freedom for 18 months following left frontal resection, while a separate right ventricular lesion remained unresected. Seizures had continued despite successive medication regimens that included valproate, oxcarbazepine, topiramate, and vigabatrin, supporting referral for presurgical evaluation. The clinical relevance of this case lies in distinguishing the electroclinically localized surgical target from other structural abnormalities requiring independent assessment and surveillance. It also highlights the need to distinguish clinical seizure freedom from electrographic normalization and to interpret lesion identity cautiously when original histopathological documentation is unavailable.

Case Presentation

A three-year-old girl with TSC was evaluated for frequent focal seizures and developmental delay. Seizures began at three months of age as brief episodes of right-hand clonic jerking without reported loss of consciousness. Their frequency increased over time. During the later presurgical assessment, approximately 11-16 seizures were reported daily, generally lasting up to one minute and involving right-hand jerking and blinking. Clinical records identified a TSC1 mutation, but the original genetic laboratory report, specific variant, zygosity, testing method, and parental testing results were unavailable.

The documented treatment history included valproate, oxcarbazepine, topiramate, vigabatrin, lacosamide, and levetiracetam. Early records described persistent seizures during valproate treatment at up to 40 mg/kg/day, followed by combined valproate and oxcarbazepine treatment, with oxcarbazepine increased to 60 mg/kg/day. Approximately 100 or more daily seizures were reported during these treatment stages. After topiramate was added at 5 mg/kg/day while oxcarbazepine was being reduced, approximately 10-20 daily seizures remained. Subsequent treatment with valproate 30 mg/kg/day, topiramate 5.5 mg/kg/day, and vigabatrin 110 mg/kg/day did not achieve seizure freedom, with approximately 10-15 daily seizures reported. Complete durations and maximum tolerated doses of all medication trials could not be reconstructed.

A record approximately three months before surgery described participation in a blinded lacosamide/placebo trial. The treatment allocation and its relationship to subsequent prescribed lacosamide were unavailable, and dose units were inconsistent across documents. Levetiracetam was introduced during that admission with planned titration and gradual valproate withdrawal. A later record, approximately two weeks before surgery, listed vigabatrin 750 mg twice daily, topiramate 50 mg twice daily, levetiracetam 62.5 mg twice daily, and lacosamide syrup 12 mL/day. The syrup concentration and the beginning of this record were unavailable, preventing verification of the lacosamide dose in milligrams and reconstruction of the complete regimen. The exact medication schedule on the operative date was not available.

Preoperative neurosurgical examination documented right-sided hemiparesis with strength graded 4/5 and brisker tendon reflexes on the right. The child had no spoken language and could stand with support but did not walk independently. She recognized family members but had not acquired self-care skills. Pupillary responses were preserved, the face was symmetrical, and no bulbar or bladder dysfunction was documented. A preoperative speech-language assessment described severe speech and language underdevelopment and motor alalia. Standardized developmental scores were unavailable.

Cardiac masses in both ventricles had been detected prenatally and confirmed by neonatal echocardiography without valvular obstruction. Preoperative echocardiography described masses consistent with rhabdomyomas near the ventricular apices, measuring 8.7 × 8.0 mm in the left ventricle and 8.5 × 5.1 mm in the right ventricle, with preserved left ventricular systolic function. An abdominal ultrasound was reported as showing no structural abnormalities. Comprehensive renal, retinal, and dermatological surveillance records were unavailable, preventing a complete description of systemic TSC assessment.

Preoperative brain magnetic resonance imaging (MRI) demonstrated multiple cortical and subcortical lesions, subependymal nodules, a prominent left frontal abnormality described as a transmantle tuber, and a separate lesion in the anterior horn of the right lateral ventricle considered radiologically suggestive of a subependymal giant cell astrocytoma (SEGA) (Figure *1*). The displayed images are post-contrast T1-weighted acquisitions. An earlier clinical summary reported no interval change in the right ventricular subependymal lesion on MRI approximately nine months before surgery compared with an examination about one year earlier. However, lesion-specific measurements and a complete longitudinal growth assessment were unavailable.

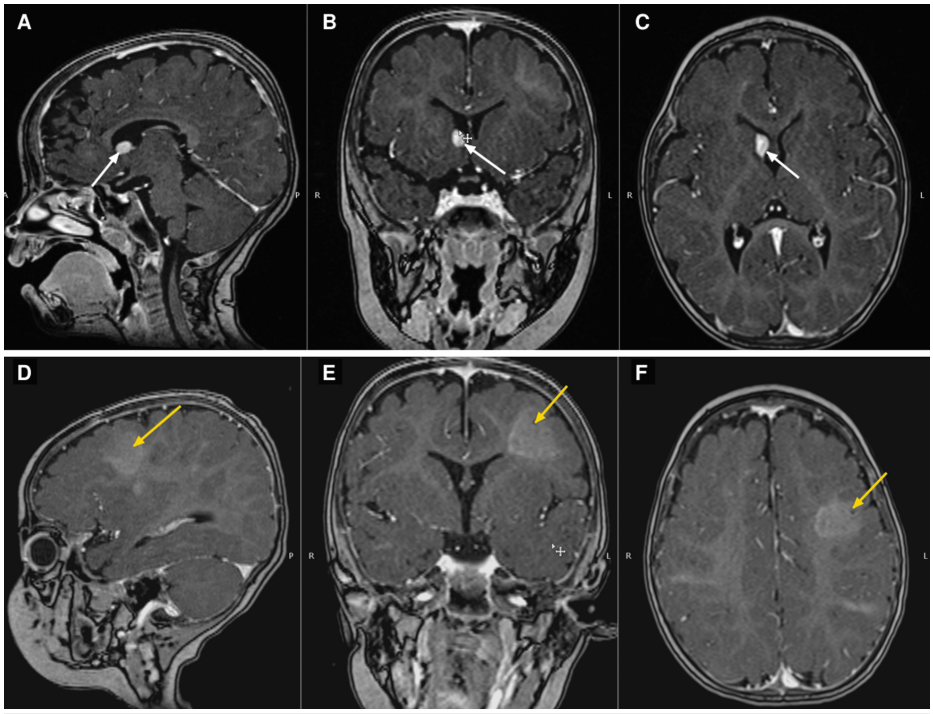


FIGURE 1: Preoperative brain MRI showing left frontal and right ventricular lesions.

Preoperative contrast-enhanced T1-weighted brain magnetic resonance imaging (MRI). (A-C) Sagittal, coronal, and axial images demonstrate an enhancing lesion in the anterior horn of the right lateral ventricle, considered radiologically suggestive of a subependymal giant cell astrocytoma (white arrows). (D-F) Sagittal, coronal, and axial images demonstrate the left frontal abnormality corresponding to the region selected for epilepsy surgery (yellow arrows). The right ventricular lesion was anatomically separate from the surgical target and remained unresected.

An earlier video-electroencephalography (video-EEG) report, approximately nine months before surgery, documented 67 hours of recording during wakefulness and physiological sleep. A total of 146 seizures were recorded, comprising 68 electroclinical and 78 electrical-only events. Epileptiform activity predominantly involved the left frontal and centroparietal regions, with an emphasis at F3 and propagation to other left hemispheric regions and occasional right posterior involvement. Clinical events included right-hand clonic movements, movements involving the eyes and head, and less frequent leg movements. EEG discharges usually preceded the visible clinical manifestations.

A subsequent presurgical video-EEG assessment, approximately two months before surgery, included wakefulness, sleep, and awakening. Interictal regional epileptiform discharges were recorded over the left frontal region, maximal at F3-C3, during wakefulness and sleep. Multiple subclinical focal seizure patterns were also recorded during sleep in the same region. Clinical events consisted of changes in postural tone with slow forward leaning, frequent blinking, and occasional right-arm clonic jerking, most often during wakefulness or after awakening. These events were associated with sustained epileptiform activity tending to become rhythmic at F3-C3. Clinical onset and termination were indistinct, and the electrographic activity outlasted the visible manifestations. This later report described multiple events without specifying their exact number or formally assessing stereotypy. Its precise recording duration could not be established because the heading and narrative provided different durations.

Following multidisciplinary review, the team unanimously recommended left frontal resection on the basis of the seizure manifestations, predominantly left-sided EEG findings, and corresponding structural abnormality. Additional functional imaging and invasive EEG monitoring were not performed because the team considered the available findings sufficient for surgical planning. The remaining intracranial lesions were not selected as surgical targets, although their possible participation in the epileptogenic network could not be definitively excluded.

Microsurgical resection was performed using Brainlab neuronavigation, intraoperative neurophysiological monitoring, and bipolar stimulation before resection. The operative report described an enlarged, abnormally convoluted posterior portion of the superior frontal gyrus. Anatomical landmarks, neuronavigation, and neurophysiological mapping guided the planned cortical incision anterior to the precentral gyrus. The abnormal tissue was resected en bloc under neurophysiological monitoring and was

described as dense, extending into the middle frontal gyrus and medially toward the lateral ventricular wall. Intraoperative electrocorticography was not performed. Detailed stimulation parameters and responses were unavailable. Operative duration was three hours and ten minutes, and estimated blood loss was 60 mL. The separate right ventricular lesion remained unresected.

The subsequent neurosurgical follow-up record described the histological diagnosis of the resected frontal specimen as "giant cell astrocytoma, grade I." The original histopathological report and slides were unavailable for independent review. This reported diagnosis is retained without independently reclassifying the frontal specimen as either a cortical tuber or a histologically confirmed SEGA, and without conflating it with the separate ventricular lesion.

At approximately 18 months after surgery, caregivers reported no clinical seizures since the operation. The outcome was classified as Engel class I on the basis of reported clinical seizure freedom while antiseizure medication continued. A more specific Engel subclass was not assigned because the available follow-up documentation did not provide sufficient detail. The follow-up narrative reported discontinuation of vigabatrin during the postoperative period, but another medication listing in the same record included it. The complete regimen at this visit, and the reason and precise timing of vigabatrin discontinuation, could therefore not be established reliably. Ophthalmological monitoring records were unavailable for review.

Caregivers reported developmental gains, including independent walking and the emergence of syllabic speech. These abilities were also documented at the follow-up examination, although developmental limitations remained and standardized longitudinal developmental testing was unavailable. Right-sided hemiparesis was documented at 4/5, with brisker tendon reflexes on the right, matching the recorded preoperative strength grade. The available records did not establish the presence or duration of any transient postoperative worsening.

An EEG approximately five months after surgery was reported as showing no epileptiform activity. A later EEG, approximately 17 months after surgery, was summarized in the neurosurgical follow-up record as showing epileptiform discharges in the centroparietotemporal regions, predominantly on the left, despite continued clinical seizure freedom. The original follow-up EEG report and tracing were unavailable, preventing assessment of electrode-level distribution, discharge morphology, or associated clinical events.

Follow-up contrast-enhanced MRI approximately 17 months after surgery demonstrated postoperative left frontal changes without substantial interval change compared with the preceding study (Figure *2*). At the latest documented neurosurgical visit, continued neurological follow-up and antiseizure treatment were recommended, together with repeat contrast-enhanced brain MRI after one year and neurosurgical reassessment. Subsequent surveillance outcomes were unavailable. An earlier neurological discharge recommendation had mentioned possible everolimus treatment if epilepsy surgery was not feasible; it did not establish a treatment plan specifically directed at the right ventricular lesion.

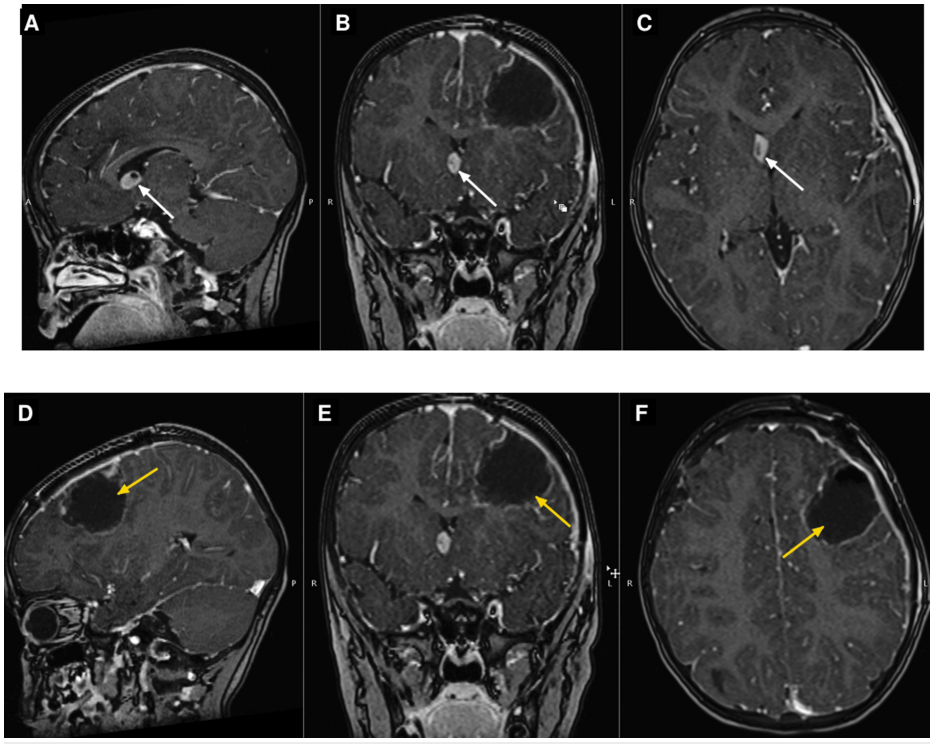


FIGURE 2: Postoperative brain MRI following left frontal resection.

Postoperative contrast-enhanced T1-weighted brain magnetic resonance imaging (MRI). (A-C) Sagittal, coronal, and axial images demonstrate the unresected enhancing lesion in the anterior horn of the right lateral ventricle, considered radiologically suggestive of a subependymal giant cell astrocytoma (white arrows). (D-F) Sagittal, coronal, and axial images demonstrate the postoperative resection cavity in the left frontal lobe (yellow arrows). Panels are arranged in the same anatomical plane order as Figure 1 to facilitate comparison.

Discussion

This case documents 18 months of reported clinical seizure freedom, categorized as Engel class I, after left frontal resection in a young child with TSC and multiple intracranial lesions, while antiseizure treatment continued [5]. Surgical target selection was based on the combined seizure manifestations, predominantly left-sided ictal and interictal EEG findings, and the corresponding frontal MRI abnormality. The separate right ventricular lesion remained unresected. This outcome illustrates the potential value of individualized localization, but a single retrospective case cannot establish general surgical effectiveness or isolate the contribution of surgery from ongoing medication.

The presurgical findings supported a predominantly left frontal hypothesis rather than proving that one lesion accounted for the entire epileptogenic network. The earlier video-EEG demonstrated propagation beyond the left frontal region, and other cortical lesions could not be definitively excluded from contributing to seizures. The target was therefore selected through multidisciplinary assessment rather than lesion size alone. Available treatment records provide evidence of persistent seizures across several medication regimens, including documented doses of valproate, oxcarbazepine, topiramate, and vigabatrin. Nevertheless, incomplete trial durations, uncertainty surrounding blinded lacosamide/placebo treatment, and the unavailable operative-date medication schedule limit independent reconstruction of the full treatment history.

Young age does not preclude consideration of epilepsy surgery. In the series reported by Ravindra et al., children undergoing open resection had a mean age of 2.4 years [6]. Melikyan et al. reported Engel class I outcomes in 54% of 33 children with TSC, with a median follow-up of 5.4 years [7]. These findings provide context for the present outcome, although differences in selection, procedures, and follow-up prevent direct comparison. The present outcome reflects caregiver-reported absence of clinical seizures at 18 months while treatment continued; it does not establish medication-free remission or long-term seizure freedom.

Functional outcomes require similarly cautious interpretation. The child had no spoken language before surgery, and a speech-language assessment documented severe language underdevelopment and motor alalia. This limited assessment of expressive language function, and dedicated language mapping was not documented. The operative report places the planned cortical incision anterior to the precentral gyrus but does not establish resection of the precentral cortex or define the target's relationship to the individual

language network. Right-sided hemiparesis was graded 4/5 both before surgery and at follow-up, although the intervening motor course was incompletely documented. Independent walking and syllabic speech emerged after surgery, but the absence of standardized longitudinal developmental assessments prevents quantification of these gains or attribution solely to the intervention.

The right ventricular lesion requires assessment independently of the epilepsy surgery target. TSC management recommendations distinguish acutely symptomatic SEGAs from growing but otherwise asymptomatic lesions and consider surgery, mTOR inhibition, and imaging surveillance according to clinical and radiological findings [1,8]. An earlier clinical summary reported interval stability of the ventricular lesion before surgery. However, lesion-specific measurements and a complete growth history were unavailable, preventing retrospective confirmation of its precise radiological classification or the rationale for observation. At the latest documented visit, repeat contrast-enhanced MRI after one year and neurosurgical reassessment were recommended. Interval growth, ventricular enlargement or hydrocephalus, and new attributable symptoms would warrant reassessment, although patient-specific intervention thresholds were not recorded [1,8]. An earlier recommendation mentioned possible everolimus treatment if epilepsy surgery was not feasible; this did not establish a lesion-specific treatment plan. Subsequent adherence to surveillance and later outcomes were unavailable.

Pathological interpretation is another important limitation. The resected frontal abnormality was described radiologically as a cortical/transmantle lesion, whereas SEGAs typically arise in a subependymal location near the foramen of Monro [1,8]. Rare lesions with SEGA histology within cortical tubers have nevertheless been reported, and cortical tubers and SEGAs may share giant-cell and glioneuronal features [9]. The abbreviated follow-up diagnosis “giant cell astrocytoma, grade I” therefore cannot resolve the classification without the original histological architecture, immunohistochemical findings, and neuropathological review. We retain the reported diagnosis without independently reclassifying the specimen or equating it with the separate unresected ventricular lesion.

Clinical seizure freedom coexisted with epileptiform activity on follow-up EEG. The original follow-up report and tracing were unavailable, and the secondary description of predominantly left-sided centroparietotemporal discharges does not establish their source. These findings cannot demonstrate residual epileptogenic tissue at the resection margin, a new independent focus, or a contribution from the ventricular lesion. Any future clinical seizure recurrence would require renewed electroclinical and imaging assessment before considering additional intervention, with attention to motor and language function.

The retrospective design, 18-month follow-up, unavailable original genetic and pathological reports, incomplete systemic surveillance documentation, and unresolved medication details limit interpretation. The reason for reported vigabatrin discontinuation and the associated ophthalmological monitoring could not be verified. These gaps restrict conclusions about treatment mechanisms, developmental effects, and long-term management. The principal observation remains reported clinical seizure freedom following localized resection despite multiple structural lesions, with continued neurological and radiological follow-up required.

Conclusions

Left frontal resection in a three-year-old girl with TSC was followed by 18 months of reported clinical seizure freedom while antiseizure treatment continued. This case illustrates the potential value of localized surgery when seizure manifestations, EEG findings, and structural imaging support a surgical target despite multiple intracranial lesions. Epileptiform activity on follow-up EEG emphasizes that clinical seizure freedom does not establish electrographic normalization. The reported histological diagnosis of the frontal specimen could not be independently verified, limiting interpretation of its relationship to the separate unresected ventricular lesion. At the latest documented visit, repeat contrast-enhanced MRI after one year and neurosurgical reassessment were recommended. Follow-up was limited to 18 months, preventing conclusions about long-term seizure control, developmental trajectory, or the subsequent course of the ventricular lesion.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mars Abdaev, Yuliy Kubetsky, Sergey Kim

Acquisition, analysis, or interpretation of data: Mars Abdaev, Sergey Kim

Drafting of the manuscript: Mars Abdaev

Critical review of the manuscript for important intellectual content: Mars Abdaev, Yuliy Kubetsky,

Sergey Kim

Supervision: Sergey Kim

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Local Ethics Committee, Federal Neurosurgical Center, Novosibirsk, Russian Federation issued approval 5. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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