Indian Journal of
Applied Radiology

Open Science Publications

Volume 11, Issue 1 - 2025
© Akhil. M. Kulkarni, et al. 2025
www.opensciencepublications.com

Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series

Case Series

Akhil. M. Kulkarni[1,2], Vishal Uttarkar[2] and Suhasini Vittal Rao[1*]

[1]*Consultant fetal medicine specialist, Davangere Scan Centre, Davangere, Karnataka, India.*

[2]*Department of Radiology, SSIMS and RC, Davangere, Karnataka, India.*

***Corresponding author:** Suhasini Vittal Rao, Consultant fetal medicine specialist, Davangere scan centre, Davangere, Karnataka, India. E-mail Id: drsuhasini2010@gmail.com

Copyright: © 2025 Kulkarni AM, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Article Information: **Submission: 08/06/2025; Accepted: 02/08/2025; Published: 06/08/2025**

Abstract

Background: Seizures in paediatric patients are often due to structural brain abnormalities. Neuroimaging is critical for diagnosis and management.

Methods: We retrospectively reviewed the clinical and neuroimaging data of five pediatric patients with drug-resistant seizures. Imaging studies (CT or MRI) were analyzed to identify features that guided diagnosis and treatment.

Results: Each patient had characteristic neuroimaging findings.

Case 1: A 17-year-old female with focal seizures showed right parietal cortical thickening and a T2-FLAIR transmantle sign on MRI (Figure 1A–C), consistent with focal cortical dysplasia.

Case 2: A 15-year-old boy with infantile spasms and left hemiparesis had right hemispheric ventriculomegaly and cortical dysplasia on CT (Figure 2A–C), indicating hemimegalencephaly.

Case 3: A 13-year-old girl with a facial port-wine stain had left parietal gyriform calcifications and hemiatrophy on CT (Figure 3A–C), diagnosing Sturge-Weber syndrome.

Case 4: A 3-year-old girl with developmental delay had multiple calcified cortical tubers and subependymal nodules on CT, including a mass at the foramen of Monro (Figure 4A–C), consistent with tuberous sclerosis.

Case 5: A 1-year-old girl with new-onset seizures had isolated enlargement of the right lateral ventricle on CT (Figure 5), consistent with unilateral hydrocephalus.

In each patient, imaging findings guided targeted management (medical therapy and, when indicated, surgical intervention).

Conclusion: Neuroimaging (MRI and CT) identified the underlying causes of these children's refractory seizures. MRI revealed subtle cortical and white matter abnormalities, while CT clearly showed calcifications and ventricular anomalies. Recognizing these modality-specific features improves diagnostic accuracy and informs treatment planning..

Keywords: Neuroimaging; Paediatric Epilepsy; MRI, CT; Focal Cortical Dysplasia; Hemimegalencephaly; Sturge-Weber Syndrome; Tuberous Sclerosis; Hydrocephalus

ISSN: 2581-3919

Introduction

Seizures are a common neurological presentation in children and can result from a variety of structural brain lesions. Advanced neuroimaging, is vital for detecting these abnormalities in patients with refractory seizures. In this series, we present five children each with drug-resistant seizures due to different etiologies: focal cortical dysplasia, hemimegalencephaly, Sturge-Weber syndrome, tuberous sclerosis, and unilateral hydrocephalus. We highlight how CT and MRI findings led to specific diagnoses and guided management in each case.

Case Presentation

Case 1: Focal Cortical Dysplasia (Right Parietal Lobe)

Clinical presentation: A 17-year-old female presented with a 6-month history of refractory focal seizures and intermittent dizziness, plus one day of new left leg weakness. Seizures began at age 15 with left arm clonic jerking, rightward eye deviation, and postictal confusion. Neurological exam showed mild left leg weakness; EEG was normal. No skin findings or systemic issues.

Imaging findings: A 1.5T brain MRI revealed a lesion in the right parietal cortex. Axial T2-FLAIR images showed subcortical white matter hyperintensity extending from the cortex toward the lateral ventricle (the "Transmantle sign" on (Figure 1A). Coronal T2-FLAIR images demonstrated focal cortical thickening and blurring of the gray–white junction in the right parietal region (Figure 1B), along with patchy hyperintensity in the right parietal gyrus (Figure 1C). These features indicate abnormal cortical development.

Diagnosis: Imaging findings are diagnostic of right parietal type II focal cortical dysplasia.

Management: The patient was treated with antiepileptic medications. (Cortical resection is often curative for FCD if medication fails.)

Focal Cortical Dysplasia (FCD)

Focal cortical dysplasia (FCD) is a common cause of drug-resistant epilepsy in children, often associated with cortical malformations. MRI is the imaging modality of choice for detection and classification.

Imaging Features (MRI):

- Cortical thickening.
- Blurring of the gray–white matter junction with abnormal subcortical architecture.
- T2/FLAIR hyperintensity in white matter, sometimes extending as a Transmantle sign [1,5].
- T2/FLAIR signal changes in gray matter.
- Abnormal sulcal or gyral patterns, segmental/lobar hypoplasia or atrophy.
- Typically, no edema, calcification, or contrast enhancement [6].

Classification: [3,4]

- Type I: Subtle or non-visible on MRI; may show mild blurring in subcortical U-fibers, often temporal [5,12].
- Type IIa: Cortical thickening, blurring of junction, abnormal gyral/sulcal pattern [5].
- Type IIb: Same as IIa plus a transmantle sign (seen in 94% of cases) [1,5].
- Type III: Associated with adjacent lesions (e.g., hippocampal sclerosis, tumors, vascular malformations, gliosis); imaging dominated by associated abnormality [5].

Clinical Relevance: Surgical resection of the epileptogenic cortex typically leads to good seizure control. Presence of the transmantle sign is associated with better postoperative outcomes [8].

Case 2: Hemimegalencephaly (Right Cerebral Hemisphere)

Clinical presentation: A 15-year-old boy had a history of infantile spasms for 6 months of age, developmental delay, and persistent left hemiparesis. Neurological exam confirmed left-sided spastic weakness. There was no history of skin lesions or perinatal complications.

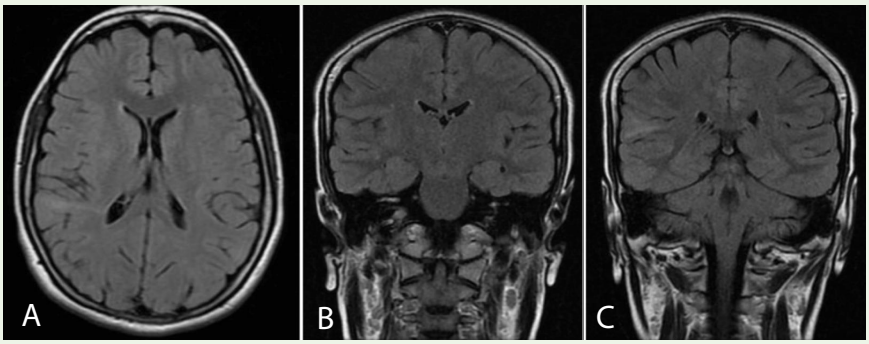

Figure 1: A: Axial T2-FLAIR MRI showing subcortical white matter hyperintensity extending from the right parietal cortex to the lateral ventricle (Transmantle sign). **B**: Coronal T2-FLAIR MRI demonstrating cortical thickening and a blurred gray–white matter junction in the right parietal lobe. **C**: Coronal T2-FLAIR MRI showing focal hyperintensity in the right parietal gyrus

Citation: Kulkarni AM, Uttarkar V, Rao SV. Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series. Indian J Appl Radiol. 2025;11(1): 217.

Imaging findings: Non-contrast CT of the head revealed marked asymmetry of the cerebral hemispheres. The right hemisphere was enlarged with severe dilation of the right lateral ventricle (monoventricle) due to blockage at the foramen of Monro and leftward midline shift (Figure 2A). The right occipital and temporal cortex appeared thinned and effaced. The right hemisphere showed diffuse low-density white matter and scattered areas of calcification. The cortical sulci in the right frontal and parietal lobes were effaced and the gyri were abnormally enlarged (Figure 2B–C), suggestive of diffuse pachygyria.

Diagnosis: These imaging features—unilateral hemispheric enlargement with ventriculomegaly, cortical malformation, and contralateral shift—are diagnostic of right-sided hemimegalencephaly.

Management: The patient was treated medically with antiepileptic drugs and underwent placement of a ventriculoperitoneal shunt to relieve hydrocephalus.

Hemimegalencephaly

Hemimegalencephaly is a rare malformation of cortical development involving hamartomatous overgrowth of one cerebral hemisphere. It accounts for ~0.2% of childhood epilepsy cases and is often associated with developmental delay and infantile spasms [9,10].

Imaging Features:

MRI is the most sensitive modality, but CT and ultrasound can also detect key features [11]:

- Enlargement of the affected cerebral hemisphere
- Asymmetric ventriculomegaly (increased or sometimes small lateral ventricle)
- Cortical malformations: polymicrogyria, pachygyria, lissencephaly, agyria [9]
- Enlarged gyri, shallow sulci
- Calvarial thickening on the affected side
- Displacement of the posterior falx to the contralateral side
- Grey matter heterotopia
- White matter calcifications
- May be associated with developmental venous anomalies (DVAs)

Nuclear imaging (SPECT/PET) typically shows hypometabolism in the affected hemisphere [10].

Classification Insight:

- Isolated hemimegalencephaly.
- Syndromic forms (e.g., with epidermal nevus, NF1, tuberous sclerosis, CLOVES) [9-11].
- Total form involves brainstem and cerebellum.

Clinical Relevance: Imaging confirms diagnosis and helps assess surgical eligibility. Hemispherectomy offers seizure control in ≥60% of refractory cases, though contralateral abnormalities may worsen prognosis [10]

Case 3: Sturge-Weber Syndrome (Left Cerebral Hemisphere)

Clinical presentation: A 13-year-old girl with a congenital port-wine stain on the left side of her face presented with multiple seizures (first at 9 months of age). Neurological exam was otherwise normal. The facial capillary malformation suggested a neurocutaneous syndrome.

Imaging findings: Non-contrast cranial CT showed classic signs. Gyriform (tram-track) calcifications were present in the cortex of the left parietal lobe (Figure 3A-C). There was marked atrophy of the left cerebral hemisphere, with cortical thinning and ipsilateral displacement of the falx (Figure 3B). These findings reflect chronic leptomeningeal vascular malformations.

Diagnosis: The clinical and imaging findings are characteristic of left-sided Sturge-Weber syndrome.

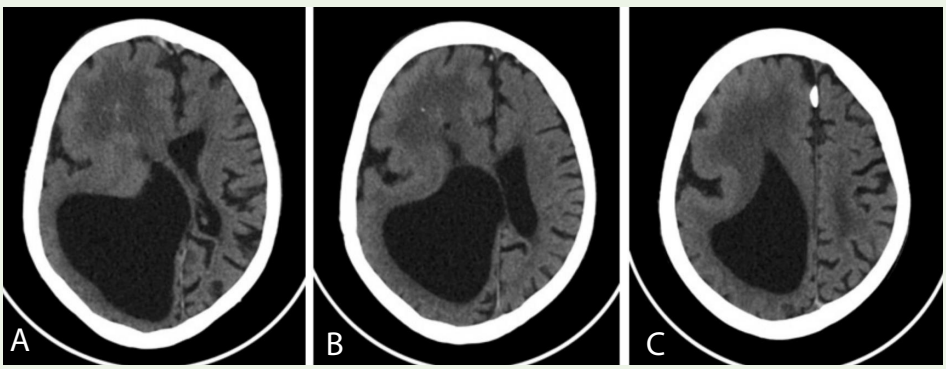

Figure 2: A: Axial CT image showing an enlarged right lateral ventricle (monoventricle) with midline shift to the left, indicating obstruction at the right foramen of Monro. **B**: Axial CT image showing diffuse low density of right hemispheric white matter and scattered calcified foci in the right frontal lobe. **C**: Axial CT image showing effaced cortical sulci and enlarged gyri in the right frontal and parietal lobes (Pachygyria).

Citation: Kulkarni AM, Uttarkar V, Rao SV. Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series. Indian J Appl Radiol. 2025;11(1): 217.

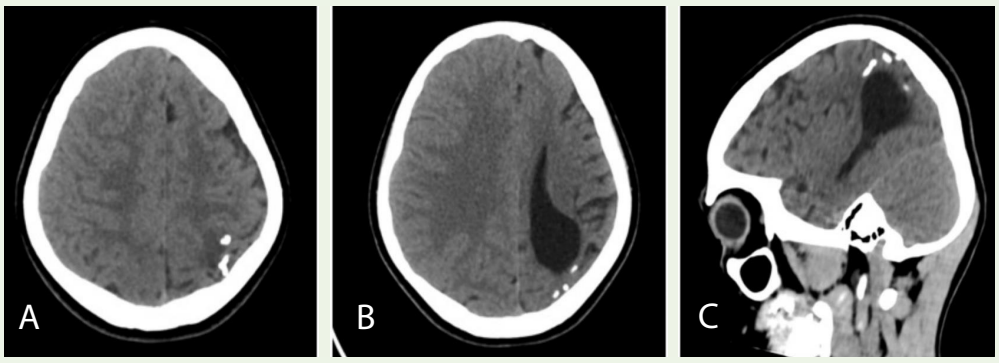

Figure 3: A: Axial CT image showing gyriform (Tram-track) calcifications in the left parietal cortex. **B**: Axial CT image showing atrophy of the left cerebral hemisphere with ipsilateral falcine displacement. **C**: Sagittal CT image demonstrating the left parietal cortical calcifications (Tram-track sulcal calcifications).

Management: The patient was managed with antiepileptic medications to control seizures; neurosurgical intervention was not performed.

Sturge Weber Syndrome

Sturge-Weber syndrome (SWS) is a neurocutaneous disorder characterized by facial port-wine stain and leptomeningeal angiomatosis, most often affecting the ophthalmic branch of the trigeminal nerve [13,14]. Seizures, typically refractory, occur in up to 89% of cases, often within the first few years of life [15,16].

Imaging Feature

- **CT** is the initial modality of choice for identifying characteristic:
  - Gyriform ("tram-track") cortical and subcortical calcifications [22,23].
  - Cerebral hemiatrophy with ipsilateral calvarial thickening.
  - Enlarged choroid plexus.
  - Asymmetric cavernous sinus or sinus enlargement [24].
  - Dyke-Davidoff-Masson appearance in severe cases.
- **MRI** better delineates:
  - Leptomeningeal enhancement on post-contrast T1 due to pial angiomatosis [15]
  - T2 low signal in subcortical white matter (from calcification or accelerated myelination).
  - Dilated transmedullary and deep venous systems.
  - SWI/GE sequences enhance calcification detection as signal dropouts.
  - MR spectroscopy may show decreased NAA [25].
- **DSA** findings include absent superficial cortical veins and abnormal deep venous drainage [16].

Classification: [19]

- Type I: Facial and leptomeningeal angiomas ± glaucoma
- Type II: Facial angioma only
- Type III: Isolated leptomeningeal angioma

Clinical Relevance

Neuroimaging is critical in confirming diagnosis and evaluating seizure etiology. CT is useful for early calcification detection; MRI provides superior detail of pial angiomas and venous abnormalities. Surgical resection is rarely indicated; management focuses on seizure control and ophthalmologic assessment [13].

Case 4: Tuberous Sclerosis

Clinical presentation: A 3-year-old girl with global developmental delay began having generalized seizures at 6 months of age. There were no focal neurological deficits or noted skin lesions on examination.

Imaging findings: Cranial CT revealed multiple typical TSC abnormalities. A calcified mass was noted at the left foramen of Monro, extending into the left lateral ventricle and causing mild dilation of the left ventricle (Figure 4A). Many calcified subependymal nodules lined the walls of both lateral ventricles (Figure 4B). The cerebral cortex contained numerous tubers seen as mixed-density lesions in the bilateral frontal and parietal lobes, with scattered calcifications (Figure 4C). Adjacent white matter showed areas of low attenuation. These findings are classic for tuberous sclerosis complex (TSC).

Diagnosis: The imaging findings of multiple cortical tubers and calcified subependymal nodules (including a large lesion at the foramen of Monro) confirm the diagnosis of TSC, likely with a subependymal giant cell astrocytoma.

Management: The patient was treated with antiepileptic drugs. Neurosurgery was consulted regarding the foramen of Monro mass; if it enlarges, surgical resection (or medical therapy such as mTOR inhibitors) may be required to prevent obstruction.

Tuberous Sclerosis

Tuberous sclerosis complex (TSC) is a multisystem genetic

Citation: Kulkarni AM, Uttarkar V, Rao SV. Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series. Indian J Appl Radiol. 2025;11(1): 217.

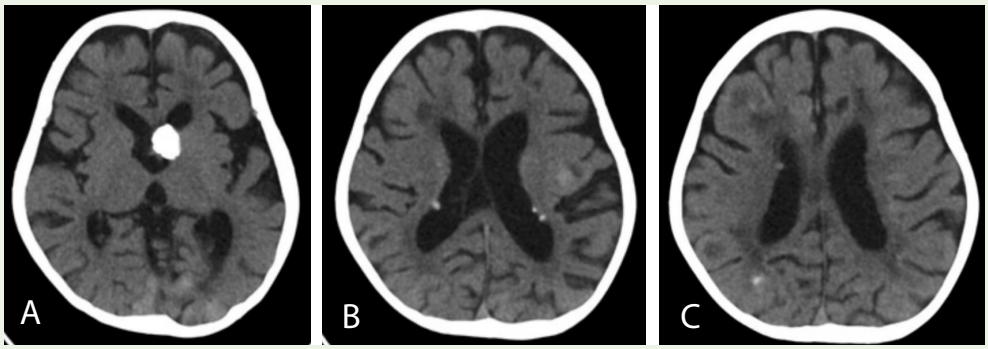

Figure 4: A: Axial CT image showing a calcified mass at the left foramen of Monro extending into the left lateral ventricle causing mild ventricular dilation. **B**: Axial CT image showing multiple calcified subependymal nodules along the walls of both lateral ventricles. **C**: Axial CT image showing multiple cortical tubers in the bilateral frontal and parietal lobes, with scattered calcifications and adjacent white matter changes.

disorder characterized by hamartomatous growths affecting the brain, skin, kidneys, lungs, and heart. About 75% of affected children present with seizures [26].

Imaging Features:

➢ **Neurological (MRI/CT)**

- **Cortical/Subcortical Tubers**: Seen in ~50% in the frontal lobe; high T2, low T1 signal; may enhance in ~10% and frequently calcify after age 2 [26].
- **Subependymal Nodules**: Often calcified (88%) [31]; iso/high T2 and high T1 signal; may resemble SEGA but distinguished by growth [32,33].
- **Subependymal Giant Cell Astrocytomas (SEGA)**: Typically appear near foramen of Monro, show growth and strong enhancement [32,33].
- **White Matter Abnormalities**: Radial bands are relatively specific to TSC [34].
- **Other CNS findings**: Corpus callosum dysgenesis, cerebellar atrophy, infarcts, aneurysms, arachnoid cysts, and rarely, chordomas.

➢ **Abdominal (CT/MRI/US)**

- **Renal Angiomyolipomas (AMLs)**: Seen in 55–75% of cases; often bilateral and large, may lack visible fat [26,29].
- **Renal Cysts**, **oncocytomas**, and **renal cell carcinomas** (26,29).
- **Retroperitoneal Lymphangiomyomatosis** and **hepatic AMLs** also occur [29].

➢ **Thoracic (HRCT/CT)**

- **Lymphangioleiomyomatosis (LAM)** in 25–40% of females [29]; can lead to pneumothorax and chylous effusion.
- **Multifocal Micronodular Pneumocyte Hyperplasia (MMPH)**: Benign nodular lung lesions (rare).
- **Cardiac Rhabdomyomas** and **myocardial fatty foci** [36,37].

➢ **Musculoskeletal & Skin (Radiographs/Clinical):**

- Sclerotic bone lesions in 40–66% of patients [26].
- Classic skin lesions include hypomelanotic macules, facial angiofibromas, shagreen patches, periungual fibromas, and forehead plaques [26,38,39].

Clinical Relevance

Imaging plays a pivotal role in diagnosing and monitoring CNS and visceral manifestations of TSC. Early identification of tubers and SEGAs can guide seizure management and surgical decisions. Multimodal imaging (MRI, CT, US) is often required due to the multisystem nature of the disease.

Case 5: Unilateral Hydrocephalus (Right Lateral Ventricle)

Clinical presentation: A 1-year-old girl presented with three episodes of unprovoked focal seizures. There were no focal deficits. Birth history was notable only for twin gestation delivered by

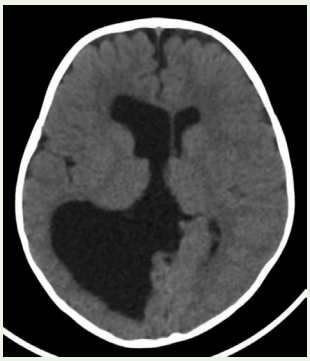

Figure 5: Axial CT image showing marked dilation of the right lateral ventricle with thinning of the adjacent right cerebral hemisphere and midline shift to the left, consistent with unilateral hydrocephalus due to foramen of Monro stenosis.

Citation: Kulkarni AM, Uttarkar V, Rao SV. Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series. Indian J Appl Radiol. 2025;11(1): 217.

Table 1: Summary of the Cases.

Case	Diagnosis	Age / Sex	Clinical Features	Imaging Modality	Imaging Findings	Final Diagnosis	Management
1	Focal Cortical Dysplasia (FCD)	17/F	Refractory focal seizures; left leg weakness	MRI	T2-FLAIR hyperintensity in right parietal gyrus; cortical thickening; blurred gray-white junction; Transmantle sign	FCD Type IIb (Right Parietal Lobe)	Antiepileptic drugs; surgical option if needed
2	Hemimegalencephaly	15/M	Infantile spasms, left hemiparesis, developmental delay	CT	Right hemispheric enlargement, monoventricular hydrocephalus, midline shift, diffuse pachygyria, white matter calcifications	Hemimegalencephaly (Right Cerebral Hemisphere)	Antiepileptic drugs; VP shunting
3	Sturge-Weber Syndrome (SWS)	13/F	Facial port-wine stain, seizures, developmental delay	CT	Tram-track cortical calcifications, left hemispheric atrophy, falcine shift	SWS (Left Cerebral Hemisphere)	Antiepileptic drugs; ophthalmology follow-up
4	Tuberous Sclerosis Complex (TSC)	3/F	Global developmental delay, generalized seizures	CT	Cortical tubers, subependymal nodules, calcified mass at foramen of Monro (likely SEGA), white matter hypodensities	TSC with Subependymal Giant Cell Astrocytoma	Antiepileptic drugs; neurosurgical evaluation
5	Unilateral Hydrocephalus (UHC)	1/F	Focal seizures; born from twin gestation	CT	Gross right lateral ventricle dilation, midline shift, thinning of right temporal lobe; no obstructing mass	UHC due to Right Foramen of Monro Stenosis (40)	VP shunting; antiepileptic drugs

C-section; no other complications were reported. No skin lesions were identified.

Imaging findings: Cranial CT showed marked enlargement of the right lateral ventricle with marked thinning of the adjacent right temporal lobe and effacement of cortical sulci (Figure 5).

The enlarged ventricle caused a midline shift (~8 mm) to the left. No mass lesion or hemorrhage was seen. These findings indicate significant unilateral right-sided hydrocephalus.

Diagnosis: Imaging confirmed an isolated dilation of the right lateral ventricle, most likely due to congenital stenosis of the right foramen of Monro (given the absence of a tumor or hemorrhage).

Management: The patient was treated with antiepileptic medication and underwent right ventriculoperitoneal shunt placement to relieve the hydrocephalus.

Unilateral Hydrocephalus Due To Stenosis of Foramen of Munro

Unilateral hydrocephalus is a rare condition typically resulting from **agenesis or stenosis of the foramen of Monro**, transient obstruction, or associated brain malformations such as forms of **holoprosencephaly** [40].

Imaging Features:

- **CT and MRI** show:
  - Marked asymmetric dilatation of a single lateral ventricle (typically right or left).
  - Thinning of adjacent brain parenchyma due to mass effect.
  - Effacement of cortical sulci and midline shift.
  - Absence of mass, hemorrhage, or infection in idiopathic cases.
- MRI may better visualize:
  - Membranous occlusion at the foramen of Monro.
  - Associated cortical malformations or interhemispheric anomalies.

Clinical Relevance

Accurate imaging diagnosis helps distinguish congenital obstruction from secondary causes like tumors or cysts. Management often involves ventriculoperitoneal shunting to relieve pressure and prevent further parenchymal damage [40].

Discussion

This series highlights the complementary roles of CT and MRI in pediatric seizure disorders. In all cases, imaging directly led to the specific etiologic diagnosis. MRI was essential for detecting subtle cortical malformations (e.g. FCD in (Case 1), whereas CT was particularly valuable for identifying calcified lesions (Cases 3 ,4) and gross hydrocephalus (Cases 2, 5). In general, MRI is the modality of choice in epilepsy due to superior soft-tissue contrast, but CT is invaluable for detecting calcifications or acute changes quickly. The characteristic patterns—cortical thickening with transmantle sign for FCD, hemispheric enlargement for hemimegalencephaly, gyriform calcifications with hemiatrophy for Sturge-Weber, multiple calcified tubers for TSC, and one-sided ventricular dilation for unilateral hydrocephalus—guided our differential diagnosis and management. Early imaging led to timely interventions (e.g. shunting or surgical planning) and tailored treatment for each patient.

Disclosure: The authors have no conflicts of interest to disclose.

Citation: Kulkarni AM, Uttarkar V, Rao SV. Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series. Indian J Appl Radiol. 2025;11(1): 217.

None of the authors received outside funding for the production of this original manuscript and no part of this article has been previously published elsewhere.

References

1. Kabat J , Król P (2012) Focal Cortical Dysplasia - Review. Pol J Radiol 77: 35-43.
2. Taylor D, Falconer M, Bruton C, Corsellis J (1971) Focal Dysplasia of the Cerebral Cortex in Epilepsy. J Neurol Neurosurg Psychiatry 34: 369-387.
3. Blümcke I, Thom M, Aronica E, Armstrong DD, Vinters HV, et al. (2011) The clinicopathologic spectrum of focal cortical dysplasias: a consensus classification proposed by an ad hoc Task Force of the ILAE Diagnostic Methods Commission. Epilepsia. 52: 158-174.
4. Najm I, Lal D, Alonso Vanegas M, Cendes F, Lopes-Cendes L, et al. (2022) The ILAE Consensus Classification of Focal Cortical Dysplasia: An Update Proposed by an Ad Hoc Task Force of the ILAE Diagnostic Methods Commission. Epilepsia 63: 1899-1919.
5. Urbach H, Kellner E, Kremers N, Blümcke I, Demerath T (2022) MRI of Focal Cortical Dysplasia. Neuroradiology 64: 443-52.
6. Kirchhof K, Harting I, Bast T, Seitz A (2003) [Focal Cortical Dysplasias: Neuroradiological Findings and Differential Diagnosis]. Rofo 175: 1056-1063.
7. Tassi L, Garbelli R, Colombo N,Bramerio M, Russo GL, et al. (2010) Type I Focal Cortical Dysplasia: Surgical Outcome is Related to Histopathology. Epileptic Disord 12:181-191.
8. Wang D, Deans A, Barkovich A, Tihan T, Barbaro NM, et al.(2013) Transmantle Sign in Focal Cortical Dysplasia: A Unique Radiological Entity with Excellent Prognosis for Seizure Control. J Neurosurg 118: 337-344.
9. Abdel Razek AA, Kandell AY, Elsorogy LG, Elmongy A, Basett AA (2009) Disorders of cortical formation: MR imaging features. AJNR Am J Neuroradiol 30: 4-11.
10. Shorvon SD, Andermann F, Guerrini R (2011) The Causes of Epilepsy. : common and uncommon causes in adults and children
11. Broumandi D, Hayward U, Benzian J, Gonzalez I, Nelson M (2004) Best Cases from the AFIP: Hemimegalencephaly. Radiographics 24: 843-848.
12. Comi AM (2007) Update on Sturge-Weber syndrome: diagnosis, treatment, quantitative measures, and controversies. Lymphat Res Biol 5: 257-264.
13. Hay WW, Hayward AR, Levin MJ, Sondheimer JM (2002) Current Pediatric Diagnosis and Treatment.
14. Griffiths PD (1996) Sturge-Weber syndrome revisited: the role of neuroradiology. Neuropediatrics 27: 284-94.
15. Reiser MF, Semmler W, Hricak H (2007) Magnetic Resonance Tomography.
16. Wyllie E, Gupta A, Lachhwani DK (2006) The Treatment of Epilepsy.
17. Pascual-castroviejo I, Viaño J, Moreno F, Palencia R, Fernandez VM, et al. (1996) Hemangiomas of the head, neck, and chest with associated vascular and brain anomalies: a complex neurocutaneous syndrome. AJNR Am J Neuroradiol 17: 461-471.
18. Wong-you-cheong JJ, Woodward PJ, Manning MA, sesterhenn IA (2000) From the Archives of the AFIP: neoplasms of the urinary bladder: radiologic-pathologic correlation. Radiographics. 26: 553-580.
19. Roach E(1992) Neurocutaneous Syndromes. Pediatr Clin North Am 39: 591-620.
20. Menkes JH, Sarnat HB, Maria BL (2006) Child Neurology.
21. Shirley MD, Tang H, Gallione CJ, Baugher JD, Frelin LP, et al.(2013) Sturge-Weber syndrome and port-wine stains caused by somatic mutation in GNAQ. N. Engl. J. Med 368: 1971-1979.
22. Yousem DM, Grossman RI (2010) Neuroradiology.
23. Akpinar E (2004) The tram-track sign: cortical calcifications. Radiology 231: 515-516.
24. Pasquini L, Tortora D, Manunza F, Rossi Espagnet MC, Figà-Talamanca L, et al. (2019) Asymmetric cavernous sinus enlargement: a novel finding in Sturge-Weber syndrome Neuroradiology 61: 595-602.
25. Moore GJ, Slovis TL, Chugani HT (1988) Proton magnetic resonance spectroscopy in children with Sturge-Weber syndrome. J. Child Neurol 13: 332-335.
26. Umeoka S, Koyama T, Miki Y, Akai M, Tustusi K, et al. (2000) Pictorial review of tuberous sclerosis in various organs. Radiographics. 28: e32.
27. Valensi Q (1973) Pulmonary Lymphangiomyoma, a Probable FormeFrust of Tuberous Sclerosis. A Case Report and Survey of the Literature. Am Rev Respir Dis 108:1411-1415.
28. Roach ES, Gomez MR, Northrup H (1988) Tuberous sclerosis complex consensus conference: revised clinical diagnostic criteria. J. Child Neurol 13: 624-628.
29. Logue LG, Acker RE, Sienko AE (2003) Best cases from the AFIP: angiomyolipomas in tuberous sclerosis. Radiographics 23: 241-246.
30. AlRayahi J, Zapotocky M, Ramaswamy V, Hanagandi P, Branson H, et al. (2018) Pediatric Brain Tumor Genetics: What Radiologists Need to Know. Radiographics : a review publication of the Radiological Society of North America, Inc. 38: 2102-2122.
31. Atlas SW(2008) Magnetic resonance imaging of the brain and spine. Lippincott Williams and Wilkins.
32. Kwiatkowski DJ, Whittemore VH, Thiele EA (2010) Tuberous Sclerosis Complex, Genes, Clinical Features and Therapeutics. Wiley-VCH.
33. Barkovich AJ (2005) Pediatric neuroimaging. Lippincott Williams and Wilkins.
34. Bernauer TA(1999) The radial bands sign. Radiology 212: 761-762.
35. AM Larson, SS Hedgire, V Deshpande, Stemmer-Rachamimov, AO, Harisinghani MG,et al. (2012) Pancreatic neuroendocrine tumors in patients with tuberous sclerosis complex Clinical Genetics 82: 558.
36. Shaaya E, Hirshberg J, Rabe OT, Thibert RL, Inglessis I et al. (2013) Cardiac Fat-Containing Lesions Are Common in Tuberous Sclerosis Complex. Am J Med Genet A 161:1662- 1665.
37. Tresoldi S, Munari A, Leo GD, Pompili G, Magistrelli P, et al. (2015) Myocardial Fatty Foci in Adult Patients with Tuberous Sclerosis Complex: Association with Gene Mutation and Multiorgan Involvement.Radiology 277: 398-405.
38. Bell DG, King BF, Hattery RR, Charboneau JW, Hoffman AD, et al. (1991) Imaging characteristics of tuberous sclerosis. AJR Am J Roentgenol 156: 1081-1086.
39. Goodrick S (2015) The road to Vogt's triad The Lancet. Neurology 14: 690.
40. Sivakumar V, Indiran V (2018) Teaching NeuroImages: Congenital membrane causing unilateral hydrocephalus. Neurology, 90: e440-e441.

Citation: Kulkarni AM, Uttarkar V, Rao SV. Role of Neuroimaging in Paediatric Seizure Disorders: A Case Series. Indian J Appl Radiol. 2025;11(1): 217.