IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 25, Issue 2 Ser. 5 (February. 2026), PP 23-28
www.iosrjournals.org

Case Report Of Polymicrogyria Presenting In Adult Patient

Dr. Sachin Bangar
Associate Professor, Department Of Medicine, Dr. V.M.G.M.C Solapur

Dr.Sankalp Santosh Hingmire (Corresponding Author)
Junior resident , Department of Medicine , Dr.V.M.G.M.C Solapur

Dr. Ankita Landge
Junior Resident, Department Of Medicine, Dr. V.M.G.M.C Solapur

Dr. Pooja Patel
Junior Resident, Department Of Medicine, Dr. V.M.G.M.C Solapur

Abstract
Objective: *Polymicrogyria (PMG) is a malformation of cortical development characterised by excessive small gyri and abnormal cortical lamination. Most cases present in childhood with developmental delay and early-onset seizures. Adult-onset PMG with new seizures and minimal neurological deficits is rare and often under-recognised.*
Case Presentation: *A 50-year-old right-handed male presented with two episodes of unprovoked, generalised tonic-clonic seizures. Neurological examination was unremarkable with no focal deficits.*
Electroencephalography (EEG) demonstrated left temporo-occipital epileptiform discharges. High-resolution MRI of the brain with an epilepsy protocol revealed focal, patchy polymicrogyria involving the left medial temporal lobe and adjacent left medial occipital lobe, consistent with a congenital disorder of neuronal migration and cortical organisation.
Management and Outcome: *The patient was diagnosed with focal epilepsy secondary to unilateral temporo-occipital PMG and commenced on appropriate antiseizure medication (ASM).*
Conclusion: *This case illustrates that focal, unilateral PMG can remain clinically silent into late adulthood and present with new-onset seizures. Clinical, electrophysiological, and neuroimaging correlation is essential for accurate diagnosis. Awareness of the broad clinical spectrum of PMG is important for practitioners evaluating adult-onset focal epilepsy.*
Keywords: *polymicrogyria; adult-onset epilepsy; malformation of cortical development; MRI; focal seizures*
Date of Submission: 17-02-2026 Date of Acceptance: 27-02-2026

I. Introduction

Polymicrogyria (PMG) is one of the most frequent malformations of cortical development (MCD) and is pathologically characterised by abnormal cortical lamination with multiple excessively folded, small gyri and fusion of the molecular layer across sulci[1]. It is considered a disorder of late neuronal migration and early cortical organisation, typically arising during mid-to-late gestation (approximately 13-24 weeks)[1][2][3].

The clinical presentation of PMG is heterogeneous and depends on the extent, distribution, and laterality of the abnormal cortex. Manifestations range from normal cognition with isolated seizures to severe encephalopathy, spasticity, developmental delay, microcephaly, and complex medical needs[2][3][4].

In most cases, seizure onset is in early childhood, and many patients develop drug-resistant epilepsy[5][6]. However, the clinical spectrum of PMG has broadened with improved neuroimaging and increasing recognition of milder phenotypes. Focal, unilateral PMG may remain clinically silent or asymptomatic until adolescence or adulthood, presenting with late-onset seizures, often with preserved cognitive function and a normal neurological examination[2][7][8].

This case report describes an unusually late presentation of PMG-associated epilepsy and emphasizes the importance of considering cortical malformations in the differential diagnosis of adult-onset focal seizures.

II. Case Presentation

Patient History

A 50-year-old right-handed male presented to the neurology outpatient clinic following two episodes of unprovoked seizures, characterised by sudden loss of consciousness followed by bilateral tonic-clonic movements lasting 2-3 minutes. He reported post-ictal confusion and fatigue lasting several hours. There was no documented aura or witnessed focal onset.

Detailed history revealed no prior seizures during childhood, adolescence, or adulthood prior to these two episodes. There was no history suggestive of perinatal complications, intrauterine infection, or significant head trauma. The patient denied substance use, neurotoxic exposures, or recent illness , and there was no history of medication non-adherence or acute withdrawal.

No family history of seizures, neurodevelopmental disorder, or genetic syndrome.

Physical Examination

General and systemic examination was unremarkable. On detailed neurological examination:

- Higher mental functions; Cranial nerves; Motor system; Reflexes; Sensory examination; Coordination; Gait; Speech was normal
- No dysmorphism or neurocutaneous markers noted.

The normal neurological examination was particularly noteworthy given the structural brain abnormality that would later be identified.

Investigations

Laboratory investigations:

Complete blood count, serum electrolytes (sodium, potassium, chloride, bicarbonate), blood urea nitrogen, serum creatinine, liver function tests (bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase), fasting glucose, and serum calcium were all within normal limits.

Electroencephalography (EEG):

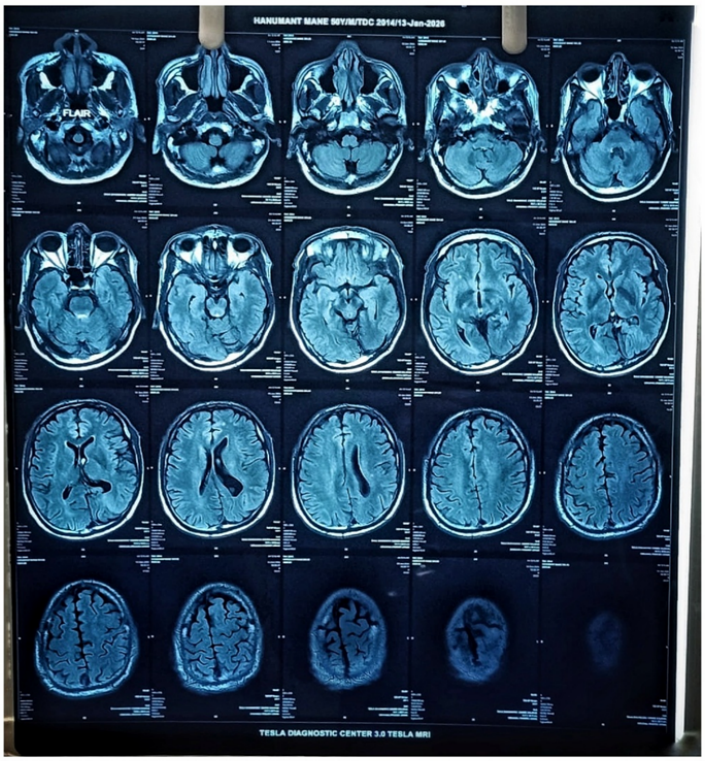
Scalp video-EEG monitoring was performed in the interictal state. The recording showed:

Interictal findings: Focal, repetitive sharp spike and wave discharges localized to the left posterior temporal and occipital regions (T5, O1, Pz electrodes).

The finding of left temporo-occipital epileptiform discharges provided electrophysiological localisation of the seizure focus and was concordant with the structural imaging findings (see below).

Neuroimaging:

MRI brain with an epilepsy protocol (3 Tesla, including high-resolution T1 and T2 FLAIR sequences) was performed. The study revealed:



Imaging Finding	Description
Location	Left medial temporal and adjacent left medial occipital lobe
Morphology	Focal, patchy polymicrogyria with abnormal cortical architecture
Cortical surface	Multiple small, irregular gyri; festooned appearance
Cortical thickness	6-8 mm (thickened compared to normal cortex, 3-4 mm)
Grey-white junction	Irregular and indistinct at the site of PMG
Associated findings	No hippocampal sclerosis; no cortical signal abnormality

Table 1: Summary of MRI Findings in Brain Epilepsy Protocol

Diagnosis: The imaging appearance was consistent with **focal, unilateral, left temporo-occipital polymicrogyria**, classified as a congenital malformation of cortical development due to abnormal neuronal migration and cortical organisation.

Management

The patient in this case was initiated on levetiracetam 500 mg twice daily, Common side effects (behaviour, mood, somnolence) were counselled, and regular follow up was advised .

III. Discussion

Epidemiology and Clinical Spectrum

PMG is estimated to account for approximately 16% of all malformations of cortical development, with a paediatric prevalence around 2.3 per 10,000 children[4][5]. The distribution of PMG patterns was as follows: perisylvian (61%), generalised/bilateral (13%), frontal (5%), parasagittal parieto-occipital (3%), and other localised patterns (18%)[5]. Associated clinical features included global developmental delay (55%), spasticity (44%), microcephaly (22%), and seizures (78%)[5]. Seizure onset was typically in infancy or early childhood; only a minority presented in adolescence or adulthood[5].

However, emerging literature has recognised a broader clinical spectrum of PMG. Focal, unilateral PMG—particularly when limited in extent—can be associated with normal neurodevelopment, preserved cognition, and no neurological deficit[2][7][8]. In such cases, seizures may present later in life or even remain subclinical. The present case exemplifies this milder end of the phenotypic spectrum: a 50-year-old with normal

development, intact cognition, normal neurological examination, and new-onset seizures secondary to focal unilateral PMG.

Developmental Mechanisms

PMG arises from a disruption of late neuronal migration and early cortical organisation during mid-to-late gestation (13-24 weeks in utero) [1]. Current understanding implicates:

- **Pial surface disruption:** The integrity of the pial basement membrane is crucial for proper neural guidance. Pial rupture allows neurons to migrate past the cortical plate into subcortical regions (over-migration) and disorganises the normal radial glial guidance system [1][3].
- **Vascular insufficiency:** Intrauterine ischaemia or hypoperfusion may result in focal disruption of the pial surface and abnormal cortical lamination [1][3].
- **Excessive proliferation and disorganisation:** Dysregulation of progenitor proliferation may lead to abnormal layering and excessive gyral folding [1].

Aetiological Factors

The aetiology of PMG is heterogeneous and includes:

1. **Genetic causes:** Both isolated and syndromic forms; causative genes identified include *GPR56*, *PAX6*, *RELN*, *TUBA1A*, genes in the PI3K-AKT-mTOR pathway, and many others. Inheritance patterns include autosomal dominant, autosomal recessive, X-linked, and oligogenic inheritance [1][3][6].
2. **Intrauterine infections:** Particularly cytomegalovirus (CMV) and other TORCH infections; ZIKV more recently recognised as a cause of PMG and other MCDs [1][3].
3. **Vascular insults:** Placental insufficiency, twin-twin transfusion syndrome, and umbilical cord accidents leading to in utero ischaemia [1][3].
4. **Metabolic and chromosomal disorders:** Rare associations with metabolic disease, chromosomal anomalies (e.g. trisomy 21, Turner syndrome), and maternal exposure to toxins [1][3].
5. **Idiopathic/sporadic:** In many cases, particularly unilateral focal PMG without syndromic features or strong family history, the specific aetiology remains undetermined [2][8].

In the present patient—an older adult with unilateral focal PMG, no syndromic features, no known intrauterine infection or perinatal insult, and no family history—a sporadic etiology (likely vascular or genetic) is suspected, though definitive determination would require additional genetic testing and detailed obstetric history.

Neuroimaging Characteristics

MRI Appearance

High-resolution MRI is the gold standard for diagnosing PMG [2][9]. Typical features include:

- **Cortical surface:** Multiple small, irregular gyri imparting a "bumpy" or "festooned" appearance, in contrast to the normal smooth gyral pattern.
- **Cortical thickness:** Often increased, typically 6-8 mm (normal 3-4 mm) due to disorganised cortical architecture and fusion of the molecular layer.
- **Grey-white junction:** Irregular and indistinct, particularly at the site of PMG.
- **Intervening sulci:** Shallow or absent, reflecting fusion of the microgyri.
- **Signal characteristics:** Generally normal T1 and T2 signal; no associated abnormal signal intensity or restricted diffusion (unless complicated by seizure, stroke, or other comorbidity).
- **Associated abnormalities:** May include other MCDs, white matter abnormalities, or vascular dysgenesis depending on the underlying aetiology[9].

Lobar Distribution

While perisylvian PMG is the most common pattern (61% in large series), regional or lobar patterns occur [5]. The temporo-occipital involvement seen in this case—a focal lobar pattern affecting medial temporal and medial occipital regions—represents a less common distribution but is well-recognised in the literature [2] [5] [9].

Distinction from Other Cortical Malformations

Differential diagnosis on imaging includes:

- **Focal cortical dysplasia (FCD):** Typically shows cortical-subcortical blurring (transmantle sign), abnormal gyral pattern, and dysmorphic grey matter. PMG typically shows intact cortical surface but abnormal gyral pattern and irregular grey-white junction without prominent transmantle involvement[1][9].
- **Heterotopia:** Nodular or band-like ectopic grey matter within the white matter, distinguished from PMG by location and morphology[9].

- **Schizencephaly:** Cleft lined by grey matter extending from cortex to ventricle; distinct from PMG[9].
- **Type I lissencephaly:** Smooth cortical surface with thick cortical ribbon and white matter abnormality; distinct from the irregular surface of PMG[9].

Adult Presentation and Diagnostic Challenges

PMG is most commonly diagnosed in childhood. However, adult-onset PMG with new-onset seizures and normal neurological examination has been increasingly recognised in the modern neuroimaging era[2][7][8]. Diagnostic challenges in adults include:

1. Low clinical suspicion
2. Normal neurological examination:
3. Mimicry of other conditions:
4. Variable MRI appearance

In the present case, the patient's age, normal examination, and initially unremarkable history might have led to diagnostic delay without appropriate neuroimaging and clinical correlation.

Management Principles

Antiseizure Medication (ASM): The choice of ASM should be tailored to the type of seizure and individual patient factors. For focal-onset seizures, first-line options include levetiracetam, lamotrigine, oxcarbazepine, and lacosamide [11][12].

The patient in this case was initiated on levetiracetam 500 mg twice daily, Common side effects (behaviour, mood, somnolence) were counselled, and regular follow up was advised .

Counselling

Patients should be counselled regarding:

- The congenital nature of the cortical malformation and its non-progressive character [2][3].
- Risk of seizure recurrence without medication [2].
- Importance of medication adherence and regular follow-up [11][12].
- Lifestyle measures (sleep hygiene, stress reduction, avoidance of known triggers) [11].
- Driving restrictions based on local regulations [11].
- Preconception counselling if considering pregnancy
- Genetic implications and potential benefit of genetic testing in some cases [3][6].

Neuropsychological Evaluation

While this patient had normal cognition on screening, comprehensive neuropsychological testing may reveal subtle deficits in memory, attention, or visual processing depending on the location and extent of PMG and the presence of interictal or ictal activity [2][7]. Such testing is particularly important in younger patients with PMG to guide educational planning and occupational rehabilitation [2].

Genetic Evaluation

Genetic testing is increasingly recommended for patients with PMG, particularly those with:

- Early or severe disease, Family history of neurological disease or PMG, Syndromic features or developmental abnormalities, Bilateral or extensive PMG [3][6].

Management of Comorbidities

Patients with PMG may have associated depression, anxiety, or cognitive decline related to the malformation, repeated seizures, or ASM effects [2][4]. Regular screening and management of psychiatric and cognitive comorbidities is important [2][4][11].

Learning Points and Clinical Implications

1. **Polymicrogyria is not exclusively a paediatric diagnosis.** Focal, unilateral PMG can remain asymptomatic into adulthood and present with new-onset seizures, particularly in the 4th-5th decade or later.
2. **Normal neurological examination does not exclude significant structural brain abnormality.** Careful neuroimaging is essential in all patients with new-onset focal epilepsy, regardless of examination findings.
3. **High-resolution MRI with epilepsy protocols is essential for accurate diagnosis.** Standard brain MRI may miss subtle focal PMG; dedicated sequences and expert radiological review are needed.
4. **Clinical-EEG-imaging correlation is crucial.** Concordance of seizure localisation on EEG with the location of PMG on MRI supports the diagnosis and guides management.
5. **PMG-associated adult-onset seizures may have a relatively favourable prognosis.** Focal, unilateral PMG is often controlled with monotherapy ASM, though long-term data are limited.

6. **Genetic evaluation and counselling should be considered** even in apparently sporadic cases, as emerging evidence illuminates the genetic heterogeneity of PMG.
7. **Multidisciplinary care** involving neurology, neuroimaging expertise, neuropsychology, and genetic services optimises outcomes in PMG-associated epilepsy.

IV. Conclusion

This case describes an unusual presentation of polymicrogyria: late-onset epilepsy in a 50-year-old male with focal, unilateral left temporo-occipital PMG and preserved neurological function. While most PMG cases present in childhood, this report emphasises that milder phenotypes with focal, unilateral involvement may remain clinically silent until adulthood.

The diagnostic approach centred on clinical correlation of seizure semiology, electrophysiological localisation on EEG, and high-resolution neuroimaging findings. The patient's excellent response to monotherapy ASM highlight the generally favourable prognosis of focal PMG-associated epilepsy when appropriately managed.

Awareness of the broad clinical spectrum of PMG is essential for neurologists and other practitioners evaluating adult-onset focal epilepsy. Future large prospective cohorts of adult-onset PMG will further clarify prognosis, optimal management strategies, and outcomes with contemporary ASMs and potential surgical interventions.

Conflict of Interest

The authors declare no conflict of interest.

Funding

No specific funding was received for this work.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report.

References

[1]. Leventer RJ, Jeste V, Vespignani H, Et Al. Clinical And Imaging Heterogeneity Of Polymicrogyria: A Study Of 328 Patients. Brain. 2010;133(5):1415-1427. Doi:10.1093/Brain/Awq062
[2]. Guerrini R, Dobyns WB. Malformations Of Cortical Development: Clinical Features And Genetic Causes. Lancet Neurol. 2014;13(9):883-893. Doi:10.1016/S1474-4422(14)70142-8
[3]. Stutterd CA, Leventer RJ. Polymicrogyria: Pathology, Fetal Origins And Mechanisms. Acta Neuropathol Commun. 2014;2:100. Doi:10.1186/S40478-014-0100-2
[4]. Kolbjer H, Saarinen T, Gissler M, Et Al. Polymicrogyria: Epidemiology, Imaging, And Clinical Aspects In A Population-Based Cohort. Brain Commun. 2023;5(4):Fcad213. Doi:10.1093/Braincomms/Fcad213
[5]. Kerr ML, Jain K, Mayr V, Et Al. Polymicrogyria-Associated Epilepsy: A Multi-Center Phenotypic Study From The Epilepsy Phenome/Genome Project. Epilepsia. 2014;55(9):1376-1388. Doi:10.1111/Epi.12698
[6]. Poduri A, Evrony GD, Cai X, Et Al. Somatic And Germline Mutations In The MTOR Gene In Focal Cortical Dysplasia And Epilepsy. Nat Genet. 2012;44(11):1231-1236. Doi:10.1038/Ng.2406
[7]. Cherif F, Et Al. Polymicrogyria Revealed By Epileptic Seizures In Adults: About Two Cases. Surg J Med Case Rep. 2021;3(5):1080-1082.
[8]. Jissendi-Tchofo P, Et Al. Current Concepts Of Polymicrogyria. Dev Med Child Neurol. 2010;52(7):596-606. Doi:10.1111/J.1469-8749.2010.03659.X
[9]. Hayakawa K, Yamamoto S, Okada T, Et Al. The Changing MR Imaging Appearance Of Polymicrogyria. AJNR Am J Neuroradiol. 2002;23(9):1493-1500.
[10]. Burneo JG, Et Al. Clinical Features Of Unilateral Polymicrogyria And Epilepsy In Adults. Epilepsy Open. 2021. [Abstract]
[11]. Glauser T, Ben-Menachem E, Bourgeois B, Et Al. Updated ILAE Evidence Review Of Antiepileptic Drug Efficacy And Effectiveness As Initial Monotherapy For Epileptic Seizures And Syndromes. Epilepsia. 2013;54(3):551-563. Doi:10.1111/Epi.12074
[12]. Kwan P, Brodie MJ. Early Identification Of Refractory Epilepsy. N Engl J Med. 2000;342(5):314-319. Doi:10.1056/NEJM200002033420503
[13]. Barba C, Et Al. Seizure Outcome In Drug-Resistant Epilepsy In The Setting Of Polymicrogyria. Seizure. 2024;121:1-7. Doi:10.1016/J.Seizure.2024.06.012
[14]. Dobyns WB, Et Al. Malformations Of Cortical Development: Clinical Features And Genetic Causes. Lancet Neurol. 2016;15(3):313-326. Doi:10.1016/S1474-4422(15)00151-6
[15]. Guerrini R. Polymicrogyria And Other Disturbances Of Cortical Development. In: Epilepsy And The Interictal State. Wiley-Blackwell; 2007. P. 45-67.

Figure Legends

Figure 1. Axial T2-weighted MRI image through the medial temporal lobes demonstrating focal, patchy polymicrogyria (arrows) involving the left medial temporal lobe with characteristic irregular gyral pattern, cortical thickening (6-8 mm), and indistinct grey-white matter junction. Normal right medial temporal lobe shown for comparison