Supplementary Table 1: Demographic, clinical, pathology, and molecular features of pediatric PLNTY cases

Case	Age at presentation of symptoms	Age at first MRI	Sex	Time between initial MRI and surgery (months)	Duration of symptoms prior to surgery (months)	Symptoms	Type of seizures	Recurrence of seizures after surgery	Calcifications on pathology	Cortical dysplasia on pathology	MAP kinase pathway activation
1	11	12	F	5	11	Seizures	Complex focal seizures	No	Yes	No	BRAF V600E mutation
2	14	14	F	2	4	Headache	-	No	Yes (extensive)	No	FGFR2-PASD1 fusion
3	6	6	M	14	16	Seizures	Complex focal seizures	No	Yes	No	FGFR2-SHTN1 fusion
4	13	10#	F	145	131	Seizures	Complex focal seizures	No	Yes (extensive)	No	BRAF V600E mutation
5	9	9	M	63	64	Headache	-	No	No	Yes	BRAF V600E mutation
6	<1	4	F	46	48	Seizures	Simple focal seizures	Yes*	Yes	No	FGFR2 - INA, ATM fusion
7	6	6	M	0	0	Seizures	Complex focal seizures	No	Yes	Yes	FGFR2 – INA fusion
8	14	14	M	2	2	Seizures, Headache	Complex focal seizures	Yes*	No	Yes	FGFR2 - CTNNA3 fusion
9	<1	2	M	5	97	Seizures	Complex focal seizures	Yes*	Yes	No	BRAF V600E mutation
10	9	11	F	65	92	Seizures, Headache	Simple focal seizures	No	No	No	BRAF V600E mutation

FGFR2: Fibroblast growth factor receptor 2; BRAF: B-RAF proto-oncogene; SHTN1: Shootin 1; PASD1: PAS domain-containing protein 1; INA: Internexin Neuronal Intermediate Filament Protein Alpha; CTNNA3: Catenin Alpha 3; ATM: Ataxia-telangiectasia mutated.

*Patients underwent surgical reintervention

#The patient was asymptomatic at the initial MRI.

Supplementary Table 2: Imaging features of pediatric PLNTY

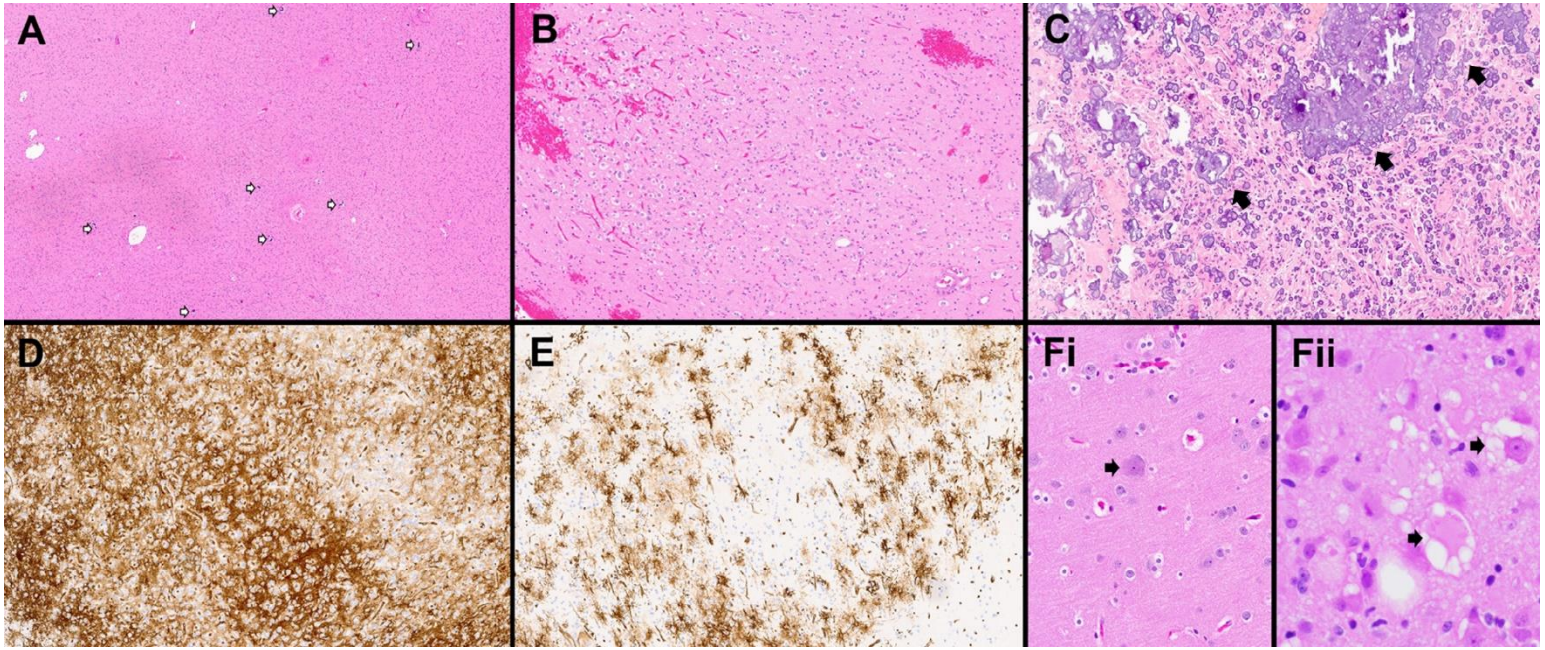
Case	Tumor location	Tumor side	Cortical-subcortical location	Margins	Morphology	Cysts (size mm)	Calcification	Calcification Pattern	Contrast Enhancement	Contrast Enhancement Pattern	ASL Perfusion	T1w signal*	T2w signal*	T2 FLAIR signal*	TLS
1	Medial Temporal	Left	Yes	Ill-defined	Solid and cystic	Yes (1.8)	Yes	Punctate	Yes	Mild and Nodular	_	Mixed	Hyperintense	Hyperintense	No
2	Inferior Parietal	Left	No	Well-delineated	Solid and cystic	Yes (17.2)	Yes	Chunky	No	_	_	Mixed	Mixed	Mixed	No
3	Postero-Lateral Temporal	Right	Yes	Ill-defined	Solid and cystic	Yes (5.5)	Yes	Punctate	Yes	Mild and Scarce	Mildly Elevated	Hyperintense	Hyperintense	Hyperintense	Yes
4	Occipital	Right	Yes	Ill-defined	Solid and cystic (on follow-up)	Yes (3.2)	Yes	Chunky (eventually)	No	_	_	Hyperintense	Hyperintense	Hyperintense	No
5	Antero-Lateral Temporal	Right	No	Ill-defined	Solid	No	Yes	Punctate	Yes	Mild and Patchy	Mildly Elevated	Hypointense	Hyperintense	Hyperintense	No
6	Medial Parietal	Left	Yes	Ill-defined	Solid and cystic	Yes (3.1)	Yes	Chunky	No	_	_	Mixed	Mixed	Mixed	Yes
7	Antero-Lateral Temporal	Right	Yes	Ill-defined	Solid and cystic	Yes (8.4)	Yes	Punctate	Yes	Mild and Scarce	_	Isointense	Hyperintense	Hyperintense	No
8	Postero-Lateral Temporal	Left	Yes	Ill-defined	Cystic	Yes (9.3)	No	_	No	_	_	Hypointense	Hyperintense	Mixed	Yes
9	Medial Temporal	Right	Yes	Ill-defined	Solid and cystic	Yes (4.4)	Yes	Punctate	No	_	_	Isointense	Hyperintense	Hyperintense	Yes
10	Antero-inferior Temporal	Right	Yes	Ill-defined	Solid and Cystic	Yes (8.7)	No	_	No	_	Low	Isointense	Hyperintense	Hyperintense	No

ASL: Arterial spin label, TSL: Transmantle-like sign, *: relative to normal cortical signal

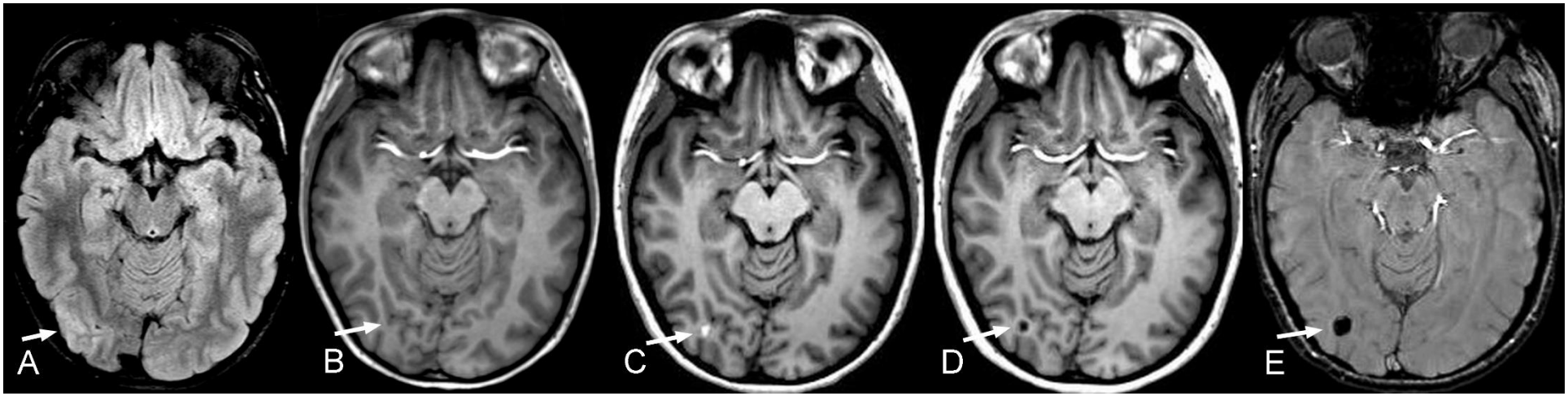
Supplementary Table 3: WHO5 essential diagnostic criteria for PLNTY.

Diffuse growth pattern (at least regionally)
Few (if any) mitotic figures
Oligodendroglioma-like morphology
CD34 expression by tumor cells and by ramified neural cells in the associated cerebral cortex
IDH-wildtype status
MAPK pathway driving genetic abnormalities (typically BRAF p.V600E mutations or FGFR2/FGFR3 fusions).

WHO 5, 2021 5th edition of the World Health Organization Classification of Tumors of the Central Nervous System.



Supplementary Figure 1. Histopathology of PLNTY tumors. A: Lesion with diffuse growth and scattered microcalcifications (white arrows) at low magnification (H&E stain, 40x magnification). B: Focal oligodendroglial-like components with clear perinuclear halos (H&E stain, 100x magnification). C: An example of a tumor with widespread calcifications (black arrows) (H&E stain, 100x magnification). D: All cases exhibited diffuse CD34 staining (CD34 immunostaining, 100x magnification). E: Ramified CD34 staining is observed in the cortex adjacent to the tumor (CD34 immunostaining, 100x magnification). F: In a subset of cases, dysmorphic neurons (black arrows) were detected in the cortex near the tumor, characterized by their large size, abnormally clustered Nissl substance, and occasional cytoplasmic vacuoles (Fi: H&E stain, 200x magnification; Fii: H&E stain, 400x magnification), indicating the presence of adjacent cortical dysplasia.



Supplementary Figure 2. A 10-year-old female (case 4) presented with head trauma. (A) Axial FLAIR sequence shows a faint, ill-defined high signal intensity area in the right occipital lobe (arrow), warranting follow-up. (B) Non-contrast axial T1WI showed no corresponding signal abnormalities. The patient was lost to follow-up and returned years later with seizures, managed medically. (C) Non-contrast axial T1WI reveals a new high T1 signal intensity focus within the same site as the initial FLAIR signal abnormality (arrow). (D) Non-contrast axial T1WI performed 2 years later, prior to surgery, displays a focal hypointense signal abnormality in the same region (arrow). (E) Axial SWI sequence shows a corresponding susceptibility (arrow), indicating the formation of a coarse central calcification over time.