Online Table 1: Clinical data of patients with AE.

Case no.	Age/Sex	Clinical presentation	EEG	CSF	Symptom duration (Days)	Treatment	Diagnosis[#]
1	62/M	Recurrent convulsions	Normal	Normal	35	IVIG and steroids	Anti-LGI 1 encephalitis
2	44/F	Memory deterioration, rave, arms and legs twitch	Severe abnormalities (δ wave activity, and partially mixed with θ waves over prefrontal, frontal and central leads) without epileptiform discharges	Elevated total cell counts, elevated total leucocytes, and decreased chloride	65	IVIG, steroids and antiepileptic drugs	Anti-LGI 1 encephalitis
3	61/M	Memory deterioration, and paroxysmal convulsion	Normal	Elevated glucose and protein, and decreased chloride	60	IVIG, antiepileptic drugs and symptomatic treatment	Anti-LGI 1 encephalitis
4	67/F	Headache, memory deterioration, and paroxysmal convulsion	Severe abnormalities (δ wave activity, and partially mixed with θ waves over each lead) without	Elevated total cell counts, elevated protein, and decreased chloride	84	Steroids, immunosuppressive agents and	Anti-LGI 1 encephalitis

			epileptiform discharges			antiepileptic drugs	
5	68/F	Memory deterioration	Normal	Normal	30	IVIG and steroids	Anti-LGI 1 encephalitis
6	51/M	Headache, discontinuity convulsion, slow response, psychiatric disorder, walking unstable	Slow wave activity in left frontotemporal	Elevated total cell counts, total leucocytes and proteins	90	Chemotherapy and antiepileptic drugs	Anti-GABAbR encephalitis
7	47/M	Headache, convulsion and unconsciousness, memory deterioration	Severe abnormalities (δ wave activity, and partially mixed with θ waves in the left hemisphere) without epileptiform discharges	Elevated total cell counts and total leucocytes	18	IVIG, steroids and plasma exchange	Anti-GABAbR encephalitis
8	35/M	Headache, dysphasia, bad right hand movement, and amorphous type	Mild abnormalities (α rhythm irregular and increased β activity in the bilateral frontotemporal lead) without epileptiform discharges	Elevated total cell counts, total leucocytes and proteins	20	Steroids and antiepileptic drugs	Anti-NMDAR encephalitis

9	29/M	Paroxysmal binocular skew, unconsciousness, speech disorders	Diffuse slow wave activity	Elevated leucocytes and decreased glucose	42	IVIG, immunosuppressive agents and plasma exchange	Anti-NMDAR encephalitis

Note: -AE, autoimmune encephalitis; Anti-NMDAR, anti-N-methyl-D-aspartic acid receptor; Anti-LGI 1, anti-leucine rich, glioma-inactivated 1; anti-GABAbR, Anti-gamma-aminobutyric acid b receptor; EEG, electroencephalogram; CSF, cerebrospinal fluid; IVIG, intravenous immunoglobulin.

Finally diagnosed by autoantibody assay testing from patients' blood serum and cerebrospinal fluid.

Online Table 2: Conventional MRI characteristics and 3D pCASL features of 9 patients with AE.

Case No.	Lesion location	T1WI	T2WI	T2 FLAIR	Contrast enhancement	Perfusion
1	R. HT	→	→	↑	NA	↑↑
2	Bil. H	↓	↑	↑↑	None	↑↑ (R); ↑ (L)
3	Bil. H	→	→	↑ (R); → (L)	None	↑↑ (R); ↑ (L)
4	Bil. H	↓	↑	↑↑ (R); ↑ (L)	None	↑↑ (R); ↑ (L)
5	Bil. H	→	→	→ (R); ↑ (L)	None	↑ (R); ↑↑ (L)
6	L. H	↓	↑	↑	Mild patchy enhancement	↑↑
7	Bil. H★	→ (First); ↓ (Second); ↓ (Third)	→ (First); ↑ (Second); ↑ (Third)	→ (First); ↑↑ (Second); ↑ (Third)	None	NA (First); ↑↑(Second) ; ↓↓ (Third)
8	L. FPTI	→	↑	↑	None	↑
9	L. FT	↓	↑	↑	None	↑↑

Note: -R =right, L = left, Bil = bilateral, F = frontal lobe, T = temporal lobe, H = hippocampus, P = parietal lobe, I = insula lobe; NA = not applicable; T1WI = T1-weighted imaging; T2WI = T2-weighted imaging; T2 FLAIR = T2 fluid-attenuated inversion recovery; ↑ = mildly increased signal/perfusion; ↑↑ = markedly increased signal/perfusion; ↓ = mildly decreased signal; ↓↓ = markedly decreased perfusion; → = Isointensity.

★ Conventional MRI was normal and 3D pCASL was not performed on the first MRI examination.

Online Table 3: The CBF and rCBF values of the lesions with AE and the healthy controls (mean ± SD).

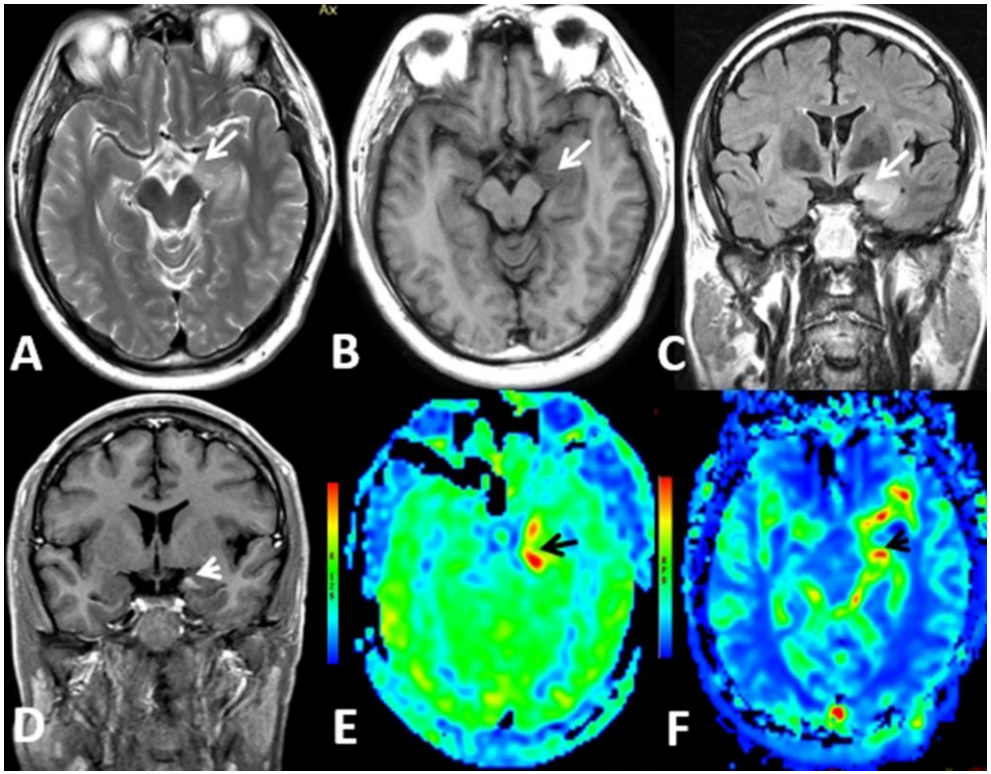
Location		AE Group	Control group	P value
Hippocampus	CBF	89.6±22.28	43.1±5.47	< 0.001
	rCBF	2.28±0.61	1.10±0.15	< 0.001
Temporal lobe	CBF	73.7±8.32	50.1±5.45	< 0.05
	rCBF	2.44±0.47	1.28±0.14	< 0.05
Insula lobe	CBF	56.9	50.18±6.99	NA
	rCBF	2.17	1.27±0.17	NA
Frontal lobe	CBF	61.25±13.08	52.81±4.03	NA
	rCBF	2.12±0.79	1.34±0.14	NA
Cerebellar hemisphere	CBF	37.14±6.98	39.16±2.51	> 0.05

Note: -NA, not applicable; CBF, cerebral blood flow; rCBF, relative CBF; Unit of CBF was ml/100 g/min.

Online Table 4: Perfusion features of patients with AE in the literature.

References	Methods	Type of AE	Type of manuscript (No. of patients)	Findings
(Llorens et al 2010)[9]	Tc-99m HMPAO SPECT	Anti-NMDAR encephalitis	Letter to the Editor (one patient)	Multiple focal regions of increased radiotracer uptake in frontal basal-limbic region
(Sachs et al 2017)[6]	ASL	Anti-NMDAR encephalitis	Case report (one patient)	Focally increased CBF in the right temporoparietal region before laboratory diagnosis and MRI abnormalities
(Vallabhaneni et al 2018)[8]	CTP	Anti-GAD65 encephalitis	Case report (one patient)	Focally increased CBF and CBV in the left parietooccipital region
(Lapucci et al 2019)[36]	ASL and ^{18}F-FDG PET/CT	Anti-NMDAR encephalitis	Letter to the Editor (one patient)	ASL and ^{18}F-FDG showed bilateral occipito-parietal hypoperfusion/hypometabolism
(Li et al 2019)[37]	ASL and ^{18}F-FDG PET/CT	Anti-LGI 1 encephalitis	Case report (one patient)	ASL and ^{18} F-FDG PET showed no abnormal perfusion/metabolism in the bilateral hippocampus
(Dinoto et al 2021) [12]	ASL and ^{18}F-FDG PET/CT	Anti-LGI 1 encephalitis and seronegative LE	Brief communication (two patients)	ASL and ^{18}F-FDG PET/CT are strongly concordant in limbic encephalitis (LE)
(Espinosa-Jovel C et al 2016)[30]	ASL and ^{18}F-FDG PET/CT	Anti-LGI 1 encephalitis	Case report (one patient)	Hyperperfusion/hypermetabolism could be related to an autonomic focal status epilepticus rather than the LGI-1 encephalitis itself

Note: -Tc-99m HMPAO SPECT, technetium-99m hexamethyl propylene amine oxime SPECT; ^{18}F-FDG PET/CT, ^{18}F-fluoro-2-deoxy-glucose PET/CT; ASL, arterial spin labeling; CTP, computed tomography perfusion; ASL, arterial spin labeling; Anti-NMDAR, anti-N-methyl-D-aspartic acid receptor; Anti-LGI 1, anti-leucine rich, glioma-inactivated 1; Anti-GAD65, anti-glutamate decarboxylase 65; CBF, cerebral blood flow; CBV, cerebral blood volume.



Online FIG 1: MR imaging of Case 6. (**A**) Axial T2WI and (**C**) Coronal T2 FLAIR showed swelling and hyperintensities in the left hippocampus (white arrows), with hypointensities on axial T1WI (B) (white arrow). (D) Coronal postcontrast TIWI revealed mild patchy enhancement (white arrowhead). (E) 3D pCASL and (F) dynamic susceptibility-weighted contrast-enhanced imaging (DSC) showed marked hyperperfusion in the corresponding region (black arrow and arrowhead, respectively).