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Venous Infarction Mimicking Brain Tumor: Recognition of the Sulcal Tram-Track Sign and Gyriform Diffusion restriction

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Abstract

Purpose

Venous infarctions can demonstrate varied imaging appearances and in certain cases, they may mimic intracranial tumors/space occupying lesions, leading to diagnostic confusion and potentially unnecessary invasive procedures. The purpose of this study was to describe the neuroimaging features of venous infarction presenting as tumor-like cortical lesions and to highlight a distinctive imaging appearance termed the Sulcal Tram-Track Sign.

Methods

A retrospective review was performed of patients with venous infarcts presenting as focal cortical lesions suggestive of space-occupying pathology on MRI. Imaging features were analyzed on conventional MRI sequences, diffusion-weighted imaging (DWI), and susceptibility-weighted imaging (SWI). Particular attention was given to lesion morphology, relationship to cortical sulci, diffusion characteristics, susceptibility findings, and enhancement patterns.

Results

Eleven patients were identified with venous infarction where the lesions were predominantly cortically based and mimicked space occupying lesions. T2-weighted imaging demonstrated paired linear hyperintensities along opposing banks of the affected sulcus in all cases, producing a tram-track appearance. Gyriform diffusion restriction was present in most patients, and susceptibility-weighted imaging demonstrated cord sign corresponding to thrombosed cortical veins in the majority of cases. Contrast enhancement was minimal or absent.

Conclusion

Venous infarction due to cortical vein thrombosis may mimic intracranial tumors on MRI. Recognition of the Sulcal Tram-Track Sign, along with associated diffusion restriction and susceptibility changes, may facilitate early diagnosis and help avoid unnecessary invasive interventions.

Introduction

Cerebral venous thrombosis (CVT) is an uncommon cause of stroke, accounting for approximately 0.5–1% of all cerebrovascular events[1–3]. Unlike arterial stroke, which typically affects older individuals with vascular risk factors, CVT often occurs in younger patients and may present with a broad spectrum of clinical manifestations including headache, seizures, focal neurological deficits, or altered

consciousness[4]. Because clinical symptoms are often nonspecific, neuroimaging plays a central role in establishing the diagnosis.

Among the various forms of venous thrombosis, isolated cortical vein thrombosis (ICVT) represents a rare subtype in which thrombosis occurs within cortical veins without involvement of the major dural venous sinuses[5]. Although uncommon, cortical vein thrombosis can result in significant parenchymal abnormalities including cortical edema, hemorrhage, and venous infarction. The imaging appearance of venous infarction differs from arterial infarction because the pathophysiology involves impaired venous drainage rather than arterial occlusion.

One of the major diagnostic challenges in cortical vein thrombosis is its potential to mimic other intracranial pathologies on imaging. Venous infarcts may appear as tumor-like lesions, particularly when associated with cortical swelling, hemorrhage, or extensive edema. Several case reports have described venous infarctions initially interpreted as gliomas or other intracranial tumors before the correct diagnosis was established. Such diagnostic pitfalls may result in unnecessary biopsy or surgical exploration.

Magnetic resonance imaging (MRI) is the primary imaging modality used to evaluate suspected cerebral venous thrombosis[6]. In addition to demonstrating parenchymal abnormalities, MRI can reveal direct signs of thrombosed cortical veins, particularly on susceptibility-weighted imaging. However, visualization of cortical veins may be difficult due to their small size and anatomical variability. Therefore, recognition of indirect parenchymal imaging patterns can be crucial in suggesting the diagnosis.

In this study, we describe a series of patients with venous infarction due to cortical vein thrombosis with or without sinus thrombosis who presented with tumor-like cortical lesions on MRI. We highlight an imaging pattern characterized by paired T2 hyperintensities along the margins of the affected sulcus, which we term the Sulcal Tram-Track Sign. To our knowledge, this imaging pattern has not been previously systematically described Recognition of this pattern along with diffusion restriction pattern may aid in differentiating venous infarction from neoplastic lesions and improve diagnostic accuracy.

Materials and Methods

This was a retrospective observational study and included patients. Study included patients who presented to our institute between March 2022 and March 2026 who presented with focal cortical lesions initially suspected to represent space-occupying pathology (in outside reports) and who were diagnosed with cortical vein thrombosis on MRI done at our institute. The diagnosis of cortical vein thrombosis with venous infarction was established based on a combination of imaging findings, including direct evidence of cortical vein thrombosis (susceptibility cord sign, loss of cortical vein flow void, or cortical vein filling defect on post-contrast imaging), associated parenchymal changes consistent with venous infarction, and follow-up imaging demonstrating evolution to gliosis or lesion resolution. In one patient with suspected arteriovenous malformation, digital subtraction angiography

(DSA) was performed and excluded a vascular malformation. Follow-up imaging and clinical evolution were used as the reference standard for final diagnosis.

Inclusion Criteria

- Cortically based lesion initially suspected to represent a space-occupying lesion.
- MRI evidence of cortical vein thrombosis.
- Availability of complete MRI examination at our institute.
- Follow-up imaging confirming venous infarction.

Exclusion Criteria

- Inadequate MRI examinations.
- Lesions with confirmed alternative diagnoses.
- Lack of follow-up imaging.

Clinical characteristics such as presenting symptoms, presence/absence of seizures, laboratory workup for thrombophillic conditions, prothrombotic state, underlying malignancy/other disorders were noted.

MRI examinations were performed on a 1.5-T system (GE Healthcare, Milwaukee, Wisconsin, USA) using a standardized brain protocol including axial T1-weighted, T2-weighted, FLAIR, DWI (b values 0 and 1000 s/mm^2), SWI/GRE, and post-contrast 3D T1-weighted BRAVO sequences where indicated. Slice thickness ranged from 4–5 mm for routine sequences and 1 mm for volumetric post-contrast imaging.

Digital subtraction angiography was not routinely performed. DSA was obtained only in one patient in whom the lesion was initially suspected to represent an arteriovenous malformation because of associated hemorrhage and apparent vascular flow voids.

Images were reviewed by a single fellowship trained neuroradiologist with 10 years of experience in Neuroradiology. Images were evaluated to assess lesion morphology, signal characteristics, presence of diffusion restriction, susceptibility artifacts corresponding to thrombosed veins, and enhancement patterns.

A consistent imaging pattern was observed across the cohort. T2-weighted images demonstrated paired hyperintensity along the banks of a cortical sulcus in all patients. This pattern corresponded to the parenchymal changes secondary to thrombosis of overlying cortical vein and produced the characteristic tram-track appearance. This was labelled as sulcal tram tack sign. Representative images showing tumour like appearance of venous infarct with tram-track sign on T2W images are shown in Fig. 1.

Descriptive statistics were used to summarize demographic and imaging findings. Continuous variables are presented as mean ± standard deviation, while categorical variables are presented as counts and

percentages.

Results

A total of 11 patients were included in the study. The mean age was 31 ± 10.9 years with age ranging from 16 to 53 years. There was slight male predominance (n = 6 males – 54.5%). Headache and focal seizures were the most common presenting symptoms.

Lesions were most frequently located in the frontal and parietal lobes, reflecting the drainage territories of superficial cortical veins. Location is mentioned in Table 1.

Table 1
Table showing location of lesions

Location	Lobe	Percentage
Frontal	6	54.5%
Parietal	3	27.3%
Temporal	1	9.1%
Peri-rolandic	1	9.1%

Subarachnoid hemorrhage was noted in 2 patients. Sinus venous thrombosis was noted in 6 cases in addition to cortical vein thrombosis, while isolated cortical vein thrombosis was noted in 5 cases. Symptoms and important clinical features are mentioned in Table 2.

Table 2
Symptoms and clinical details

Symptoms and clinical details	Number of patients
Headache	11
Seizures	9
Focal deficit	2
Altered sensorium	0
Isolated cortical vein thrombosis	5
Dural sinus thrombosis	6
Oral contraceptive use	0
Postpartum state	1
Malignancy	0
Associated autoimmune disorder	0
Prothrombotic condition in lab results	0

Additional imaging findings included: Gyriform diffusion restriction in most cases (n = 10, 90.9%), Blooming within the lesion (n = 7, 63.6%). Minimal or absent contrast enhancement (n = 10, 90.9%). Enhancement was noted only in one case in gyriform distribution in subacute phase. Regarding imaging of cortical veins, susceptibility cord sign corresponding to thrombosed cortical veins was noted in 72.7% cases (n = 8), filling defect along cortical vein noted in 8 cases (n = 8, 72.7%) and loss of cortical vein flow void (best seen on Coronal T2 in 54.5% cases (n = 6, 54.5%). These imaging features are shown in representative cases (Fig. 2 and Fig. 3).

We noted that most of these patients had gyriform diffusion restriction (n = 10, 90.9%) (Fig. 2). In this pattern we noted that white matter showed facilitated diffusion and cortex showed restricted diffusion (in part or whole) in these cases of venous infarcts with overlying cortical vein thrombosis. This gyriform pattern of diffusion restriction is shown in Fig. 4. Out of 11 cases, only one case did not show gyriform restriction as the lesion was completely hemorrhagic and mimicked Arteriovenous Malformation (AVM). Due to hemorrhage, diffusion pattern couldn’t be commented upon. The lesion mimicking AVM is shown in Fig. 5. DSA was performed in this patient and demonstrated no evidence of an arteriovenous malformation, thereby supporting the diagnosis of cortical vein thrombosis.

In the majority of cases, the lesions were initially interpreted outside as space-occupying lesions, including primary brain tumors (Table 3). Recognition of venous thrombosis on subsequent imaging led to revision of the diagnosis. Overall misdiagnosis rate at initial reporting was 81.8%.

Table 3
Table showing initial radiologic diagnosis (reported outside our institute) for venous infarcts

Initial radiology diagnosis (outside institute)	Number	Percentage
Space occupying lesion- glioma	7	63.6%
Space occupying lesion- AVM	1	9.1%
Arterial infarct	1	9.1%
Venous infarct	2	18.2%

A comparative case (Fig. 6) that was later confirmed as a high-grade glioma on follow-up imaging initially demonstrated a cortical-based lesion resembling the sulcal tram-track pattern. However, diffusion-weighted imaging showed cortical–subcortical diffusion restriction, rather than the gyriform restriction typical of venous infarction. Interval progression on follow-up imaging confirmed the neoplastic nature of the lesion. Thus, gyriform diffusion restriction in combination with the sulcal tram-track appearance may help distinguish venous infarction from cortical neoplastic lesions.

An important observation in our study was the presence of a cortical glioma that partially mimicked the Sulcal Tram-Track Sign. This finding indicates that the sign should not be considered pathognomonic for venous infarction. However, unlike venous infarction, the glioma demonstrated cortical-subcortical diffusion restriction and interval progression on follow-up imaging. Therefore, the Sulcal Tram-Track Sign should be interpreted in conjunction with ancillary findings such as gyriform cortical diffusion restriction, direct evidence of cortical vein thrombosis, and imaging follow-up. The sign is best viewed as a diagnostic clue rather than a standalone diagnostic marker.

Summary of Neuroimaging findings is shown in Table 4.

Table 4
Summary of Neuroimaging findings

Imaging Feature	Number (n = 11)	Percentage
Cortically based sulcus-centered lesion	11	100%
Tram-track T2 hyperintensity along sulcus	11	100%
T1 hypointensity	10	90.9%
Gyriform diffusion restriction	10	90.9%
Susceptibility blooming (SWI/GRE)	7	63.6%
Susceptibility Cord sign / cortical vein filling defect	8	72.7%
Loss of cortical vein flow void	6	54.5%
Minimal or absent enhancement	9	81.8%

All patients received anticoagulation therapy following diagnosis. Follow-up MRI was available in all patients and demonstrated either lesion resolution or evolution to focal gliosis without development of a neoplastic process.

Discussion

Cortical vein thrombosis is an uncommon but clinically important cause of venous infarction. Because cortical veins are small and anatomically variable, thrombosis within these vessels may be difficult to detect on routine imaging. As a result, diagnosis often relies on indirect imaging findings rather than direct visualization of the thrombus.

The pathophysiology of venous infarction differs fundamentally from that of arterial infarction. Occlusion of a cortical vein leads to increased venous pressure, impaired capillary perfusion, and disruption of the blood-brain barrier. This process results in vasogenic edema and, in severe cases, hemorrhagic infarction. The distribution of these abnormalities typically follows the venous drainage territory, which may not correspond to arterial vascular territories.

In the present study, we identified a distinctive imaging pattern in which edema was centered along a cortical sulcus. This produced paired hyperintensities along the opposing banks of the sulcus, creating a tram-track appearance on T2-weighted imaging. We propose the term Sulcal Tram-Track Sign to describe this pattern.

Recognition of this imaging pattern is important because venous infarction may mimic intracranial tumors. Tumor-like appearances may arise from focal cortical swelling, hemorrhage, or associated edema. In our cohort, most lesions were initially suspected to represent neoplastic processes. Previous studies have also described venous infarcts mimicking intracranial tumors[7–11].

Diffusion-weighted imaging findings in cortical vein thrombosis have been previously described. Yıldız et al. reported intravascular clot-related diffusion restriction, termed the “clot signal” sign, in 6/26 patients (23%), and adjacent susceptibility changes on DWI, referred to as the “clot susceptibility sign,” in 16/26 patients (61.5%)[12]. Other studies have also reported heterogeneous DWI patterns in cerebral venous thrombosis[13–15]. In our cohort, intravascular diffusion restriction was observed in 3 patients (27.3%): within the dural sinus in one case, within a cortical vein in one case, and involving both the dural sinus and cortical vein in one case, consistent with the previously described clot signal. However, the most consistent diffusion abnormality in our series was gyriform cortical diffusion restriction, reflecting parenchymal involvement in venous infarction rather than isolated intravascular thrombus signal.

The coexistence of the sulcal tram-track sign and gyriform cortical diffusion restriction may represent a distinctive imaging pattern of cortical vein thrombosis and help distinguish venous infarction from tumor-like cortical lesions. This distinction is particularly important when direct MRI signs of cortical vein thrombosis (such as the cord sign) are absent. In such cases, recognition of the tram-track

appearance along with indirect signs, including loss of cortical vein flow void or filling defects within cortical veins, may provide critical diagnostic clues and help avoid unnecessary biopsy (Fig. 3)

Early diagnosis of cortical vein thrombosis has important clinical implications. Unlike tumors, which may require surgical intervention, venous thrombosis is typically managed with anticoagulation therapy. Prompt recognition of the condition can therefore prevent unnecessary invasive procedures and improve patient outcomes.

Several limitations should be acknowledged. The sample size is relatively small due to the rarity of cortical vein thrombosis presenting as tumor-like lesions. Additionally, the retrospective design may introduce selection bias. Further studies involving larger patient populations would be valuable to validate the reproducibility and diagnostic accuracy of the Sulcal Tram-Track Sign along with gyriform diffusion restriction pattern for diagnosis of these lesions. Interobserver variability was not assessed because all studies were reviewed by a single observer. Because the study did not include a systematic control cohort of cortical tumors or other cortical lesions, sensitivity, specificity, positive predictive value, and negative predictive value of the Sulcal Tram-Track Sign could not be calculated. Future case-control studies including neoplastic and inflammatory mimics will be necessary to determine its diagnostic accuracy.

Conclusion

Venous infarction due to cortical vein thrombosis can present as tumor-like cortical lesions on MRI, representing an important diagnostic pitfall. The Sulcal Tram-Track Sign, characterized by paired T2 hyperintensities along the margins of an affected sulcus, represents a potentially useful imaging marker of cortical vein thrombosis. Recognition of this pattern may facilitate earlier diagnosis and help differentiate venous infarction from intracranial tumors.

Abbreviations

ADC
Apparent Diffusion Coefficient
AVM
Arteriovenous Malformation
CVT
Cerebral Venous Thrombosis
DWI
Diffusion–Weighted Imaging
DSA
Digital Subtraction Angiography
FLAIR
Fluid–Attenuated Inversion Recovery

GRE
Gradient Recalled Echo
ICVT
Isolated Cortical Vein Thrombosis
MRI
Magnetic Resonance Imaging
MPRAGE
Magnetization Prepared Rapid Gradient Echo
SWI
Susceptibility–Weighted Imaging
T1W
T1–Weighted Imaging
T2W
T2–Weighted Imaging

Declarations

Ethics Approval and consent to participate: The study has been approved by the appropriate ethics committee.

Informed consent was gathered from the patient and consent to publish images was obtained.

Consent for publication: Written consent to publish information was obtained from study participants.

Availability of data and material: The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Competing interests: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Figures

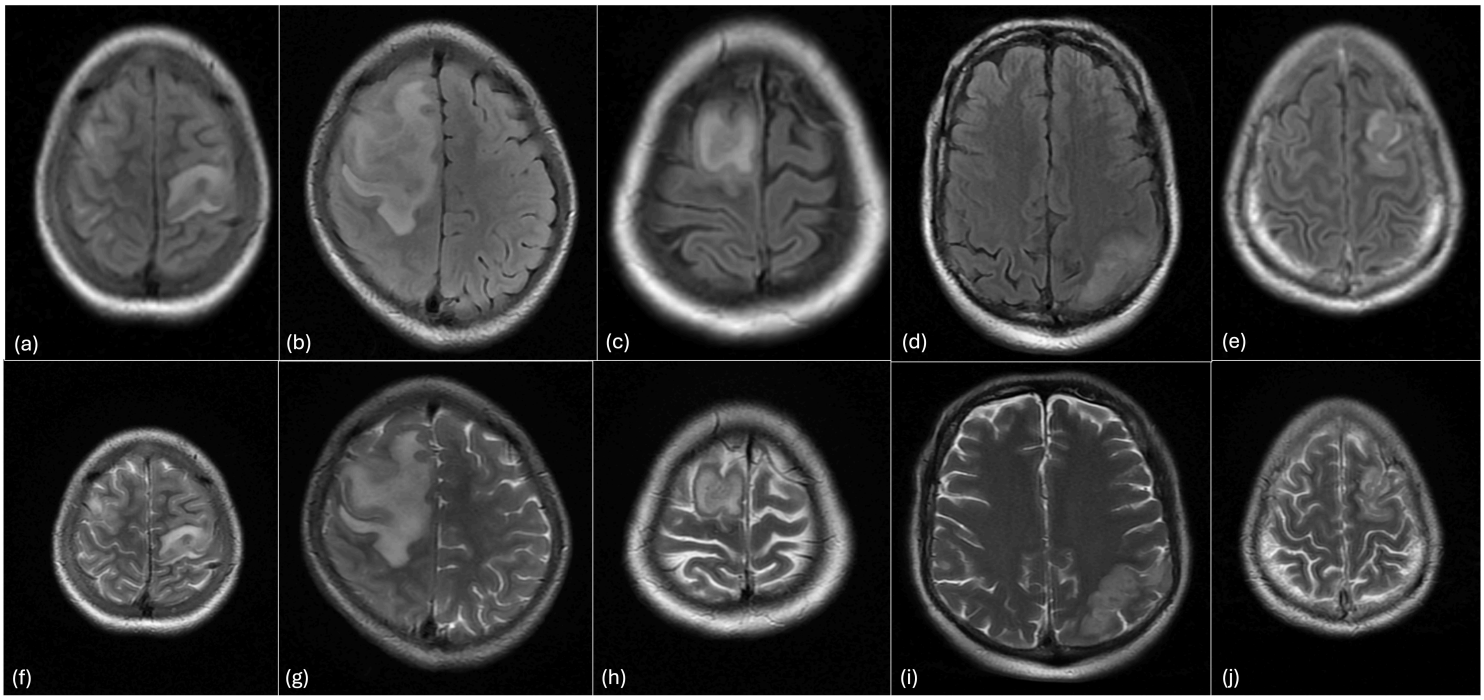


Figure 1

FLAIR axial images (a-e) show multiple representative cortical based lesions mimicking space occupying lesions. Corresponding T2 axial images (f-j) show sulcal tram track sign

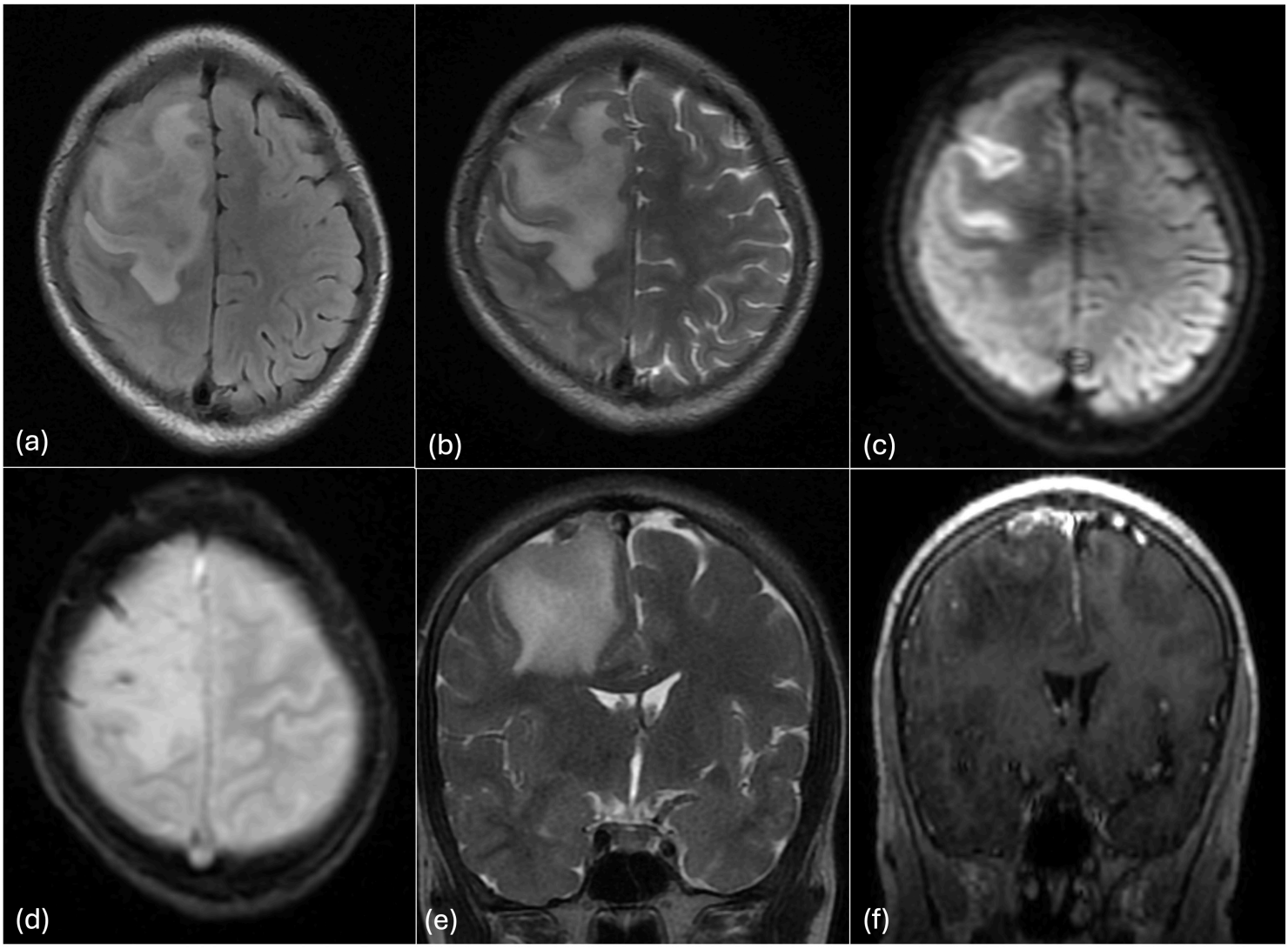


Figure 2

Representative case images FLAIR axial (a) shows FLAIR hyperintense lesion in right frontal lobe. T2W axial image (b) shows sulcal tram track appearance of lesion. DWI axial image (c) shows gyriform diffusion restriction. GRE axial image (d) demonstrates susceptibility cord sign with blooming in cortical vein as well as small focus of parenchymal blooming. T2W coronal image (e) shows loss of flow void in overlying cortical vein. Post contrast T1W MPRAGE image coronal section (f) shows filling defect in cortical vein, no sinus thrombosis was noted.

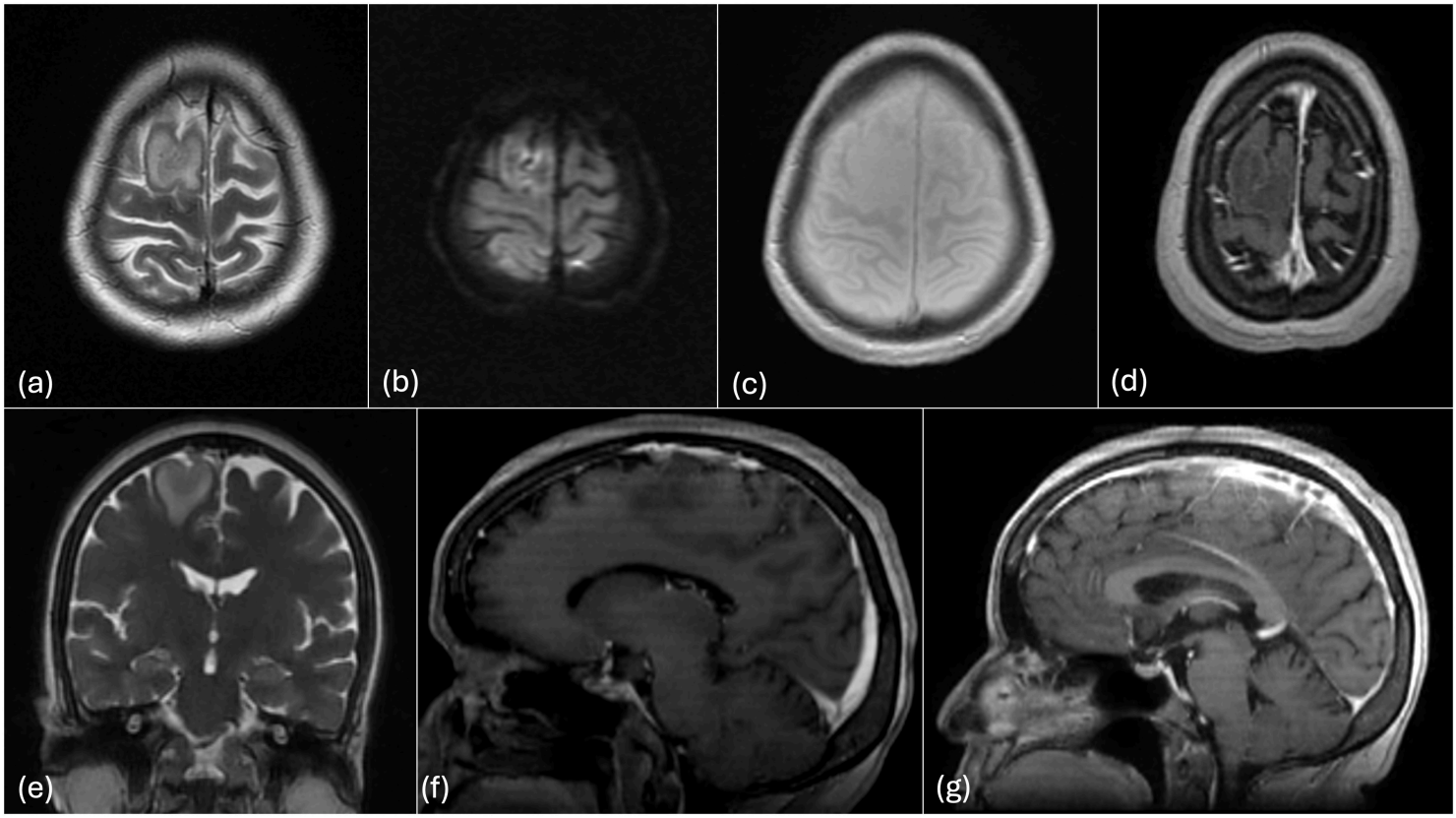


Figure 3

Representative case images T2W axial image (a) shows sulcal tram track appearance of lesion in right frontal lobe. DWI axial image (b) shows gyriform diffusion restriction. GRE axial image (c) shows no evidence of cord sign or blooming. Post contrast T1 MPRAGE axial image (d) shows no enhancement within the lesion. T2W coronal image (e) shows loss of flow void in overlying cortical vein. Post contrast T1W MPRAGE image sagittal section (f) shows filling defect in cortical vein. Post contrast T1W MPRAGE image sagittal section (g) shows filling defect in mid superior sagittal sinus.

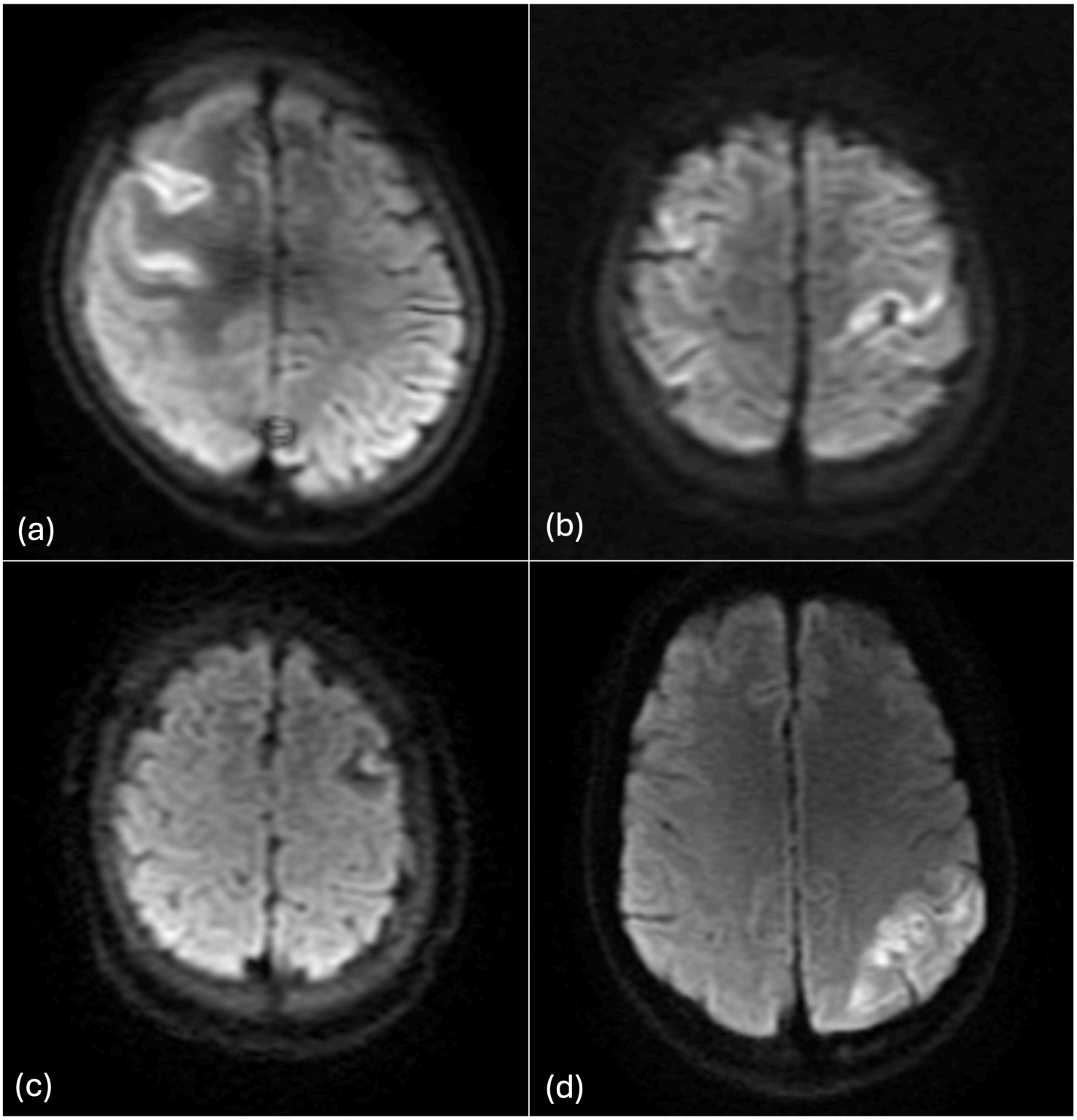


Figure 4

Axial DWI images showing pattern of gyriform diffusion restriction in different cases(a-d)

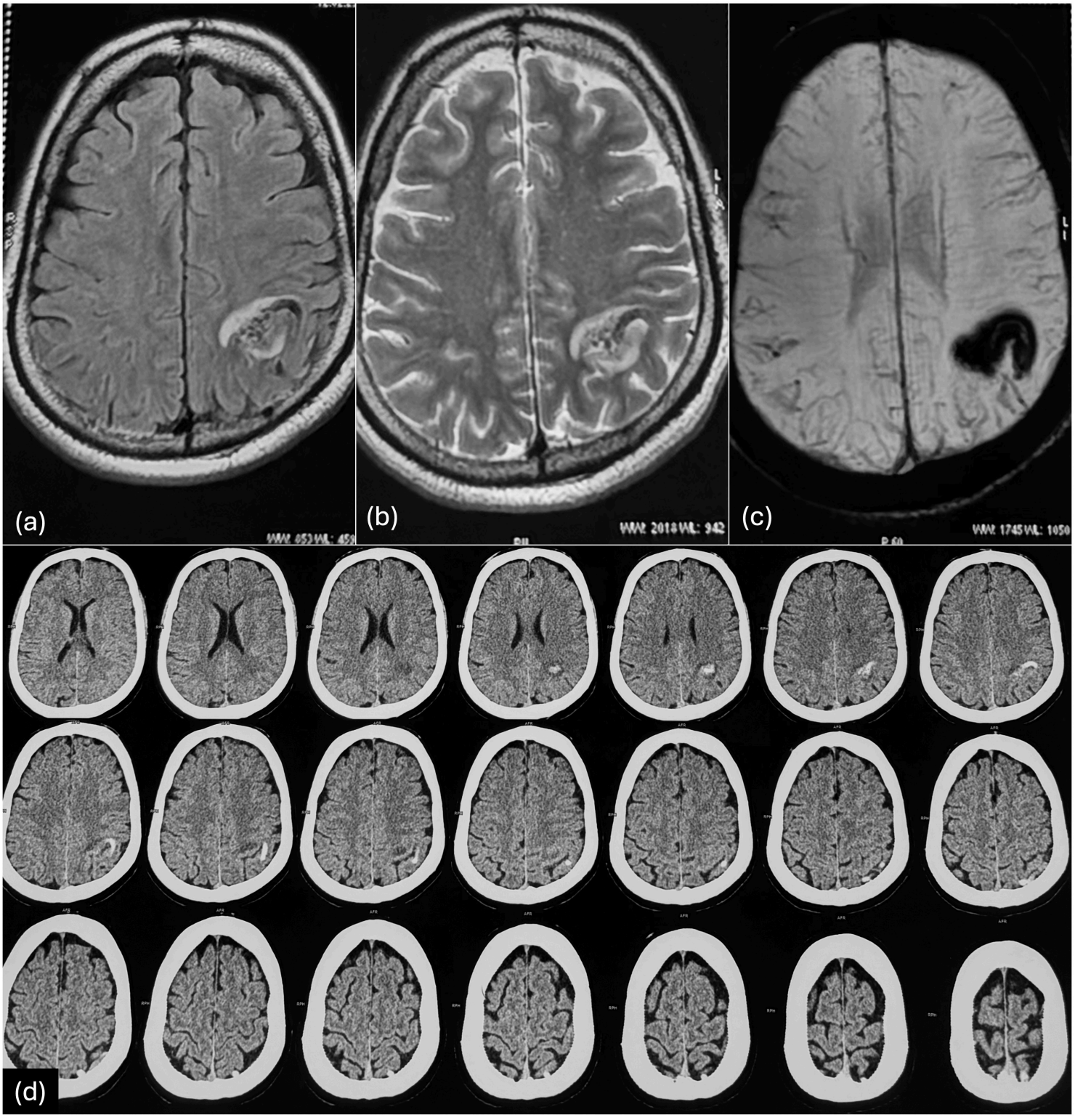


Figure 5

Representative case images FLAIR axial (a) shows FLAIR hyperintense lesion in left parietal lobe. T2W axial image (b) shows sulcal tram track appearance of lesion with appearance of flow voids associated with it, suggestive of ?underlying Arteriovenous malformation (AVM). GRE axial image (c) shows parenchymal blooming suggestive of hemorrhage. Axial CT sections (d) show hemorrhagic lesion in left

parietal lobe with hyperdense cord sign in cortical vein. DSA cerebral angiography was also done which confirmed absence of AVM and cortical vein thrombosis was diagnosed

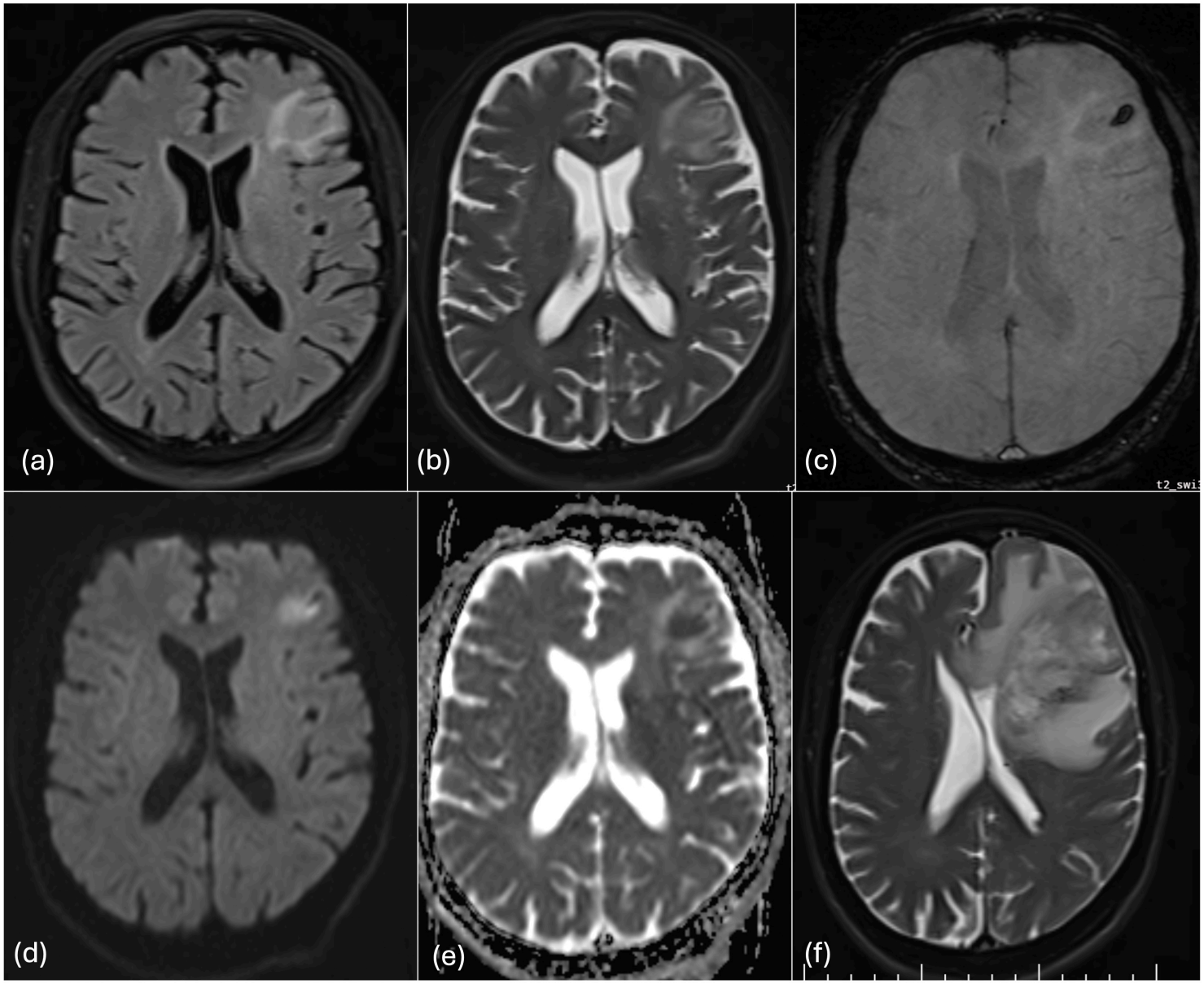


Figure 6

Representative case images FLAIR axial (a) shows FLAIR hyperintense lesion in left frontal lobe. T2W axial image (b) shows cortical based lesion closely mimicking sulcal tram track. SWI axial image (c) parenchymal blooming. DWI axial image (d) and ADC map (e) show cortical-subcortical diffusion restriction in corresponding lesion location (as opposed to gyriform restriction in venous infarcts). Follow up T2W axial image (f) shows progression of lesion, confirming neoplastic nature