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# Patterns of Posterior Circulatory Stroke in Magnetic Resonance Imaging: A Case Series

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## ABSTRACT

The present study aimed to document the patterns of posterior circulatory stroke in magnetic resonance imaging. The four cases in this series highlight the diverse MRI findings in posterior circulatory stroke. A 53-year-old male (Case 1) exhibited an acute infarct in the right medulla, characterized by a focal FLAIR hyperintense area with restricted diffusion, alongside bilateral fronto-temporo-parietal white matter hyperintensities and prominent ventricles and basal cisterns. Hypoplastic left transverse and sigmoid sinuses were noted, with absent flow in the distal right vertebral artery. In Case 2, a 47-year-old male showed a large infarct in the left thalamus, parietal, medial temporal, and occipital lobes with perilesional oedema and mass effect. T2/FLAIR hyperintensities were seen in the bilateral periventricular regions and right frontal deep white matter, and MR angiogram revealed a hypoplastic right vertebral artery and narrowed basilar and posterior cerebellar arteries. Case 3 involved a 75-year-old male with extensive T2/FLAIR hyperintensities and diffusion restriction in the bilateral parieto-occipital, posterior temporal, high parietal, and cerebellar hemispheres, suggesting posterior reversible encephalopathy syndrome, with a small T1 hyperintensity in the left cerebellar hemisphere and absent flow in the left posterior communicating artery. Lastly, Case 4 presented with acute infarcts in the left thalamus, left cerebral peduncle, and left medial temporal lobe, punctate FLAIR hyperintensities in the right midbrain and left occipital lobes, gliosis and hemosiderin deposition in the left occipital region, and bilateral papilledema.

**KEYWORDS:** Posterior circulatory stroke, Magnetic resonance imaging, Case series, India.

## INTRODUCTION

Posterior circulatory strokes (PCS) are a significant subset of ischemic strokes, accounting for approximately 20% of all strokes.(1, 2) These strokes occur due to ischemia in the vertebrobasilar arterial system, which supplies blood to the brainstem, cerebellum, thalamus, and occipital lobes.(3) Despite their prevalence, PCS are often underdiagnosed due to their diverse and sometimes subtle clinical presentations, which can include symptoms such as vertigo, dizziness, ataxia, and visual disturbances.(4) Accurate and timely diagnosis is crucial for effective management and can significantly impact patient outcomes.

Magnetic resonance imaging (MRI) plays a pivotal role in the diagnosis and evaluation of PCS.(5) Advanced MRI techniques, including diffusion-weighted imaging (DWI), fluid-attenuated inversion recovery (FLAIR), and magnetic resonance angiography (MRA), are invaluable in detecting acute ischemic changes, characterizing lesion patterns, and identifying underlying vascular abnormalities.(6-8) DWI is particularly sensitive in detecting acute ischemic lesions,(9) while FLAIR sequences can reveal hyperintensities associated with chronic ischemic changes and vasogenic oedema.(10) MRA provides detailed visualization of the arterial anatomy, aiding in the identification of occlusions, stenosis, and anatomical variations.(11)

The complexity and variability of PCS necessitate a comprehensive imaging approach to fully capture the spectrum of pathological changes. Previous studies have highlighted the diverse radiological presentations of PCS, ranging from discrete focal lesions to extensive areas of infarction with mass effect and oedema.(12)


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Furthermore, the presence of vascular anomalies, such as hypoplastic arteries and venous sinus variations, can complicate the clinical picture and impact treatment decisions.(13) However, there remains a need for detailed case studies that document these variations and their clinical implications in the context of PCS.
This case series aims to contribute to the existing body of knowledge by providing detailed MRI findings from four cases of posterior circulatory stroke. Each case is analysed to document the specific patterns of ischemic changes, associated vascular abnormalities, and any additional findings relevant to the diagnosis and management of PCS. By presenting these detailed cases, we aim to underscore the importance of comprehensive MRI protocols and highlight the variability in PCS presentations. This study emphasizes the critical role of imaging in enhancing our understanding of PCS pathophysiology and improving clinical outcomes through accurate diagnosis and tailored therapeutic strategies.

## MATERIALS AND METHODS

This case series was conducted in the Department of Radiodiagnosis, Meenakshi Medical College Hospital and Research Institute, Kanchipuram district, Tamil Nadu, India between January and June 2024 (for a duration of six months) with the aim of documenting the patterns of posterior circulatory stroke using magnetic resonance imaging (MRI). The study comprised four cases selected based on specific inclusion criteria to ensure a comprehensive analysis of posterior circulatory stroke manifestations. Patients were selected from those presenting with clinical symptoms suggestive of posterior circulatory stroke, such as vertigo, dizziness, visual disturbances, and ataxia. The inclusion criteria were a confirmed clinical diagnosis of posterior circulatory stroke, availability of complete MRI data (including diffusion-weighted imaging (DWI), fluid-attenuated inversion recovery (FLAIR), and magnetic resonance angiography (MRA)), no contraindications to MRI, and informed consent obtained for participation in the study. Patients with other neurological conditions or incomplete imaging data were excluded from the study.

All MRI scans were performed using a 1.5 Tesla (T) MRI scanner. The imaging protocol included axial DWI with b-values of 0 and 1000 s/mm² to identify acute ischemic changes, axial FLAIR sequences to detect hyperintense lesions in the posterior circulation territory, and time-of-flight MRA to visualize the arterial anatomy and identify any vascular occlusions or stenosis. Additional sequences such as T1-weighted, T2-weighted, and contrast-enhanced imaging were used as needed to further characterize the lesions. Each patient's clinical history, including onset and progression of symptoms, was documented. MRI images were reviewed by two experienced radiologists independently to ensure consistency in the identification and characterization of stroke patterns. Discrepancies were resolved by consensus. The primary outcome measures were the location, size, and extent of ischemic lesions within the posterior circulation territory. Secondary outcomes included the presence of any associated vascular abnormalities such as occlusions, stenosis, or aneurysms, as detected on MRA.

Descriptive statistics were primarily used to summarize the data. The frequency and distribution of lesions were documented, and representative cases were selected to illustrate typical and atypical patterns of posterior circulatory stroke. Detailed qualitative analysis of the MRI findings provided insights into the diversity of stroke presentations within the posterior circulation.

***Ethics approval:*** The study was approved by the institutional review board of the hospital. Informed consent was obtained from all participants or their legal guardians. The study was conducted in accordance with the principles of the Declaration of Helsinki.

### Case series

This case series presents the imaging findings of four patients with posterior circulatory stroke, evaluated using magnetic resonance imaging (MRI) techniques, including axial T1, T2, fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), gradient recalled echo (GRE), coronal T2, and sagittal T1 sequences. The results are summarized as follows:

***Case 1:*** A 53-year-old male presented with an acute infarct characterized by a focal FLAIR hyperintense area with restricted diffusion in the right medulla. Additional findings included discrete T2/FLAIR hyperintensities


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involving the bilateral fronto-temporo-parietal white matter. The ventricles, sulcal spaces, basal cisterns, and fissures were prominent. T2/FLAIR hyperintense signals were noted in the right mastoid air cells, indicating right mastoiditis. MR venogram revealed hypoplastic left transverse and sigmoid sinuses, while MR angiogram showed absent flow in the distal portion of the right vertebral artery and a hypoplastic right posterior communicating artery. The impression was an acute infarct with mild neuroparenchymal atrophy and chronic small vessel ischemic changes.

***Case 2:*** A 47-year-old male exhibited a moderate to large T2/FLAIR hyperintensity in the thalamus, parietal, medial temporal, and occipital lobe on the left side, with restricted diffusion on DWI and corresponding low apparent diffusion coefficient (ADC) values. Perilesional oedema caused a mass effect, effacing the sulcal spaces. No blooming was observed on GRE sequences. Additional findings included T2/FLAIR hyperintensities along the bilateral periventricular regions and deep white matter of the right frontal region, with ventricles, sulcal spaces, basal cisterns, and fissures prominent for age. T2 hyperintensity was noted in the bilateral maxillary sinus, suggesting maxillary sinusitis. The MR venogram was unremarkable, but MR angiogram revealed a hypoplastic right vertebral artery and narrowed basilar and bilateral posterior cerebellar arteries. The impression was an acute infarct of the left posterior cerebral artery (PCA) territory with neuroparenchymal atrophy and small vessel ischemic changes.

***Case 3:*** A 75-year-old male showed moderate to large T2/FLAIR hyperintense areas with diffusion restriction and low ADC values in the bilateral parieto-occipital, posterior temporal, high parietal, and bilateral cerebellar hemispheres. A small focus of T1 hyperintensity was noted in the left cerebellar hemisphere with no blooming on GRE. The ventricles, sulcal spaces, basal cisterns, and fissures were prominent for age. MR venogram was normal. MR angiogram indicated absent flow in the left posterior communicating artery. The impression was likely posterior reversible encephalopathy syndrome (PRES) and acute non-haemorrhagic infarcts in the PCA and vertebro-basilar territories.

***Case 4:*** A 65-year-old female showed T2/FLAIR hyperintense areas with restricted diffusion in the left thalamus, left cerebral peduncle of the midbrain, and left medial temporal lobe. There were punctate FLAIR hyperintensities in the right peduncle of the midbrain and left occipital lobes. Areas of CSF intensity with loss of cortico-medullary differentiation and mild perilesional oedema were also observed, along with peripheral blooming on GRE sequences. Bilateral optic nerves appeared thickened and tortuous with a prominent perineural sheath. MR venogram and angiogram were not reported. The impression was an acute infarct with gliosis and hemosiderin deposition in the left occipital region, indicative of sequelae from an old haemorrhagic infarct, and bilateral papilledema.

## DISCUSSION

The four cases presented in this series provide a detailed insight into the varied imaging presentations of posterior circulatory stroke, highlighting the critical role of MRI in accurate diagnosis and management. The diverse findings across these cases emphasize the importance of using a comprehensive imaging protocol, including DWI, FLAIR, GRE, and MRA, to capture the full extent of ischemic changes and associated vascular abnormalities.(14) The first case demonstrated an acute infarct in the right medulla, accompanied by mild neuroparenchymal atrophy and chronic small vessel ischemic changes. The presence of discrete T2/FLAIR hyperintensities in the bilateral fronto-temporo-parietal white matter underscores the multifocal nature of ischemic lesions in posterior circulatory stroke.(15) The hypoplastic left transverse and sigmoid sinuses, as well as the absent flow in the distal right vertebral artery, highlight the importance of vascular imaging in identifying underlying anatomical variations that could predispose patients to ischemic events.(16) These findings are consistent with the literature, which indicates that anatomical variations and chronic small vessel disease significantly contribute to stroke risk in the posterior circulation.(17)

In the second case, the patient exhibited a large infarct in the left PCA territory, causing significant mass effect and perilesional oedema. The absence of blooming on GRE sequences suggested a non-haemorrhagic infarct, while the neuroparenchymal atrophy and small vessel ischemic changes indicated a chronic ischemic process. The hypoplastic right vertebral artery and narrowed basilar and bilateral posterior cerebellar arteries further


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underscore the role of vascular abnormalities in the pathogenesis of posterior circulatory stroke. These findings align with previous studies, which have shown that PCA territory infarcts often present with significant mass effect and are frequently associated with vascular anomalies.(3)

The third case likely represents posterior reversible encephalopathy syndrome (PRES), characterized by extensive T2/FLAIR hyperintensities in the bilateral parieto-occipital, posterior temporal, high parietal, and bilateral cerebellar hemispheres. The small T1 hyperintensity focus on the left cerebellar hemisphere without blooming on GRE indicates a non-haemorrhagic etiology. PRES is often associated with hypertensive encephalopathy, eclampsia, or immunosuppressive therapy and is characterized by vasogenic oedema predominantly in the posterior circulation.(18, 19) The normal MR venogram and absent flow in the left posterior communicating artery observed in this case further illustrate the need for comprehensive imaging to distinguish between different aetiologies of posterior circulatory stroke.

The fourth case presented a complex scenario with acute infarcts in the left thalamus, left cerebral peduncle, and left medial temporal lobe, along with punctate FLAIR hyperintensities in the right midbrain and left occipital lobes. The presence of gliosis and hemosiderin deposition in the left occipital region suggests a sequelae of a previous haemorrhagic infarct, indicating a recurrent stroke event.(20) Bilateral papilledema and thickened, tortuous optic nerves with a prominent perineural sheath are notable findings, suggesting increased intracranial pressure, possibly secondary to the acute ischemic events.(21) This case underscores the complexity of stroke diagnosis and the necessity of detailed imaging to identify both acute and chronic changes.

## CONCLUSION

This case series highlights the variability in MRI findings in posterior circulatory stroke, emphasizing the necessity for a comprehensive imaging approach. Each case underscores the critical role of advanced imaging techniques in diagnosing and understanding the pathophysiology of posterior circulation strokes. The findings also stress the importance of identifying anatomical and vascular variations that may predispose individuals to ischemic events, thereby aiding in the development of targeted therapeutic strategies. Future research should focus on larger cohorts to validate these findings and explore the prognostic implications of the various imaging patterns observed in posterior circulatory stroke.

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**Table 1: Imaging characteristics of the patients included in the case series**

| | Case 1 | Case 2 | Case 3 | Case 4 |
|---|---|---|---|---|
| Age | 53 years | 47 years | 75 years | 65 years |
| Gender | Male | Male | Male | Female |
| Technique | Axial: T1, T2, FLAIR, DWI, GRE; Coronal: T2; Sagittal: T1 | | | |
| Findings | Focal FLAIR hyperintense area showing restricted diffusion – Right medulla<br>Discrete T2/FLAIR hyperintensities involving bilateral fronto-temporo-parietal white matter<br>Ventricles, sulcal spaces, basal cisterns and fissures were prominent<br>T2/FLAIR hyperintense signals in right mastoid air cells (right mastoiditis) | Moderate to large sized T2/FLAIR hyperintensity in thalamus, parietal, medial temporal and occipital lobe on left side showing restricted diffusion on DWI and corresponding low value on ADC with perilesional edema causing mass effect in the form of effacement of sulcal spaces. No blooming on GRE<br>T2/FLAIR hyperintensities noted along bilateral periventricular regions and deep white matter of right frontal region<br>Ventricles, sulcal spaces, basal cisterns and fissures were prominent for age<br>T2 hyperintensity noted in bilateral maxillary sinus (maxillary sinusitis) | Moderate to large sized T2/FLAIR hyperintense areas showing diffusion restriction and corresponding low ADC values – bilateral parieto-occipital, posterior temporal, high parietal and bilateral cerebellar hemispheres.<br>Small focus of T1 hyperintensity noted in left cerebellar hemisphere with no blooming on GRE<br>Ventricles, sulcal spaces, basal cisterns and fissures were prominent for age | T2/FLAIR hyperintense areas with restricted diffusion – left thalamus, left cerebral peduncle of midbrain and left medial temporal lobe<br>Punctuate areas FLAIR hyperintensities – right peduncle of midbrain and left occipital loves<br>CSF intensity area with loss of cortico-medullary differentiation and mild perilesional oedema.<br>Areas of peripheral blooming noted on GRE<br>Bilateral optic nerve appears thickened and tortuous with prominent perineural sheath |
| MR Venogram | Hypoplastic left transverse and sigmoid sinuses | | | Normal |

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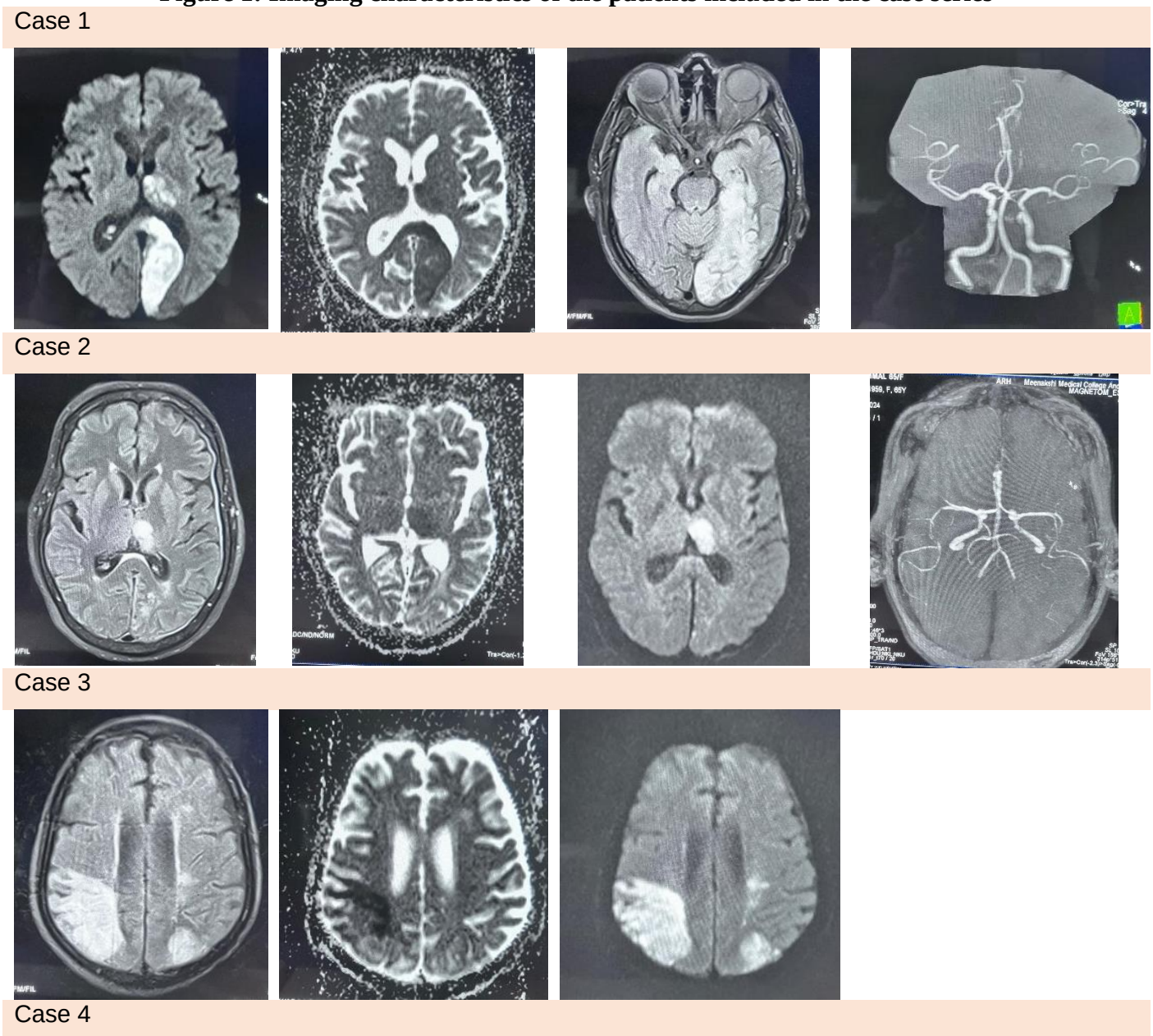
|---|---|---|---|---|
| MR Angiogram | Absent flow noted involving distal portion of right vertebral artery; hypoplastic right posterior communicating artery | | Right vertebral artery appears hypoplastic; Basilar and bilateral posterior cerebellar arteries appear narrowed. | Absent flow in left posterior communicating artery |
| Impression | Acute infarct; mild neuroparenchyma atrophy with chronic small vessel ischemic changes | Acute infarct of left PCA territory<br>Neuroparenchymal atrophy with small vessel ischemic changes | Probably, posterior reversible encephalopathy syndrome<br>Acute non-haemorrhagic infarcts – PCA and vertebra-basilar territories | Acute infarct<br>Gliosis with hemosiderin deposition in left occipital region – sequalae of old haemorrhagic infarct<br>Bilateral papilledema |
| FLAIR, Fluid-attenuated inversion recovery; DWI, Diffusion-weighted imaging; GRE, Gradient recalled echo; ADC, Apparent diffusion coefficient' PCA, Posterior cerebral artery | | | | |

**Figure 1: Imaging characteristics of the patients included in the case series**



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