Beyond the Classic Pattern: Radiologic Insights Into the Diverse Presentations of PRES

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Abstract

Posterior reversible encephalopathy syndrome is also called Reversible posterior leukoencephalopathy syndrome. Posterior reversible encephalopathy syndrome typically manifests as parieto-occipital vasogenic edema, but radiologic investigations show a much wider range of involvement. Large studies have shown extension of the imaging findings in atypical areas and in atypical pattern. Also posterior reversible encephalopathy syndrome has large number of etiological factors. Given its wide range of triggers, such as hypertension, renal failure, eclampsia, autoimmune illness, and cytotoxic treatments, prompt identification is crucial to enable prompt treatment of underlying causes and avoid irreparable damage or complications. Therefore, being aware of abnormal imaging patterns is essential for timely and efficient treatment and this article aims to review the imaging features of classical posterior reversible encephalopathy syndrome and its variants.

Keyword: Posterior Reversible Encephalopathy Syndrome, Brain Edema , Magnetic Resonance Imaging, Hypertension, Eclampsia

INTRODUCTION

PRES is a clinic-radiologic disorder with acute neurological symptoms and temporary vasogenic edema on neuroimaging as hallmarks. Despite being traditionally linked to posterior parieto-occipital involvement, mounting data indicates that PRES manifests as a variety of radiologic patterns impacting several brain regions. The syndrome arises from diverse etiologies, most commonly severe hypertension, renal dysfunction, eclampsia, exposure to cytotoxic or immunosuppressive drugs, autoimmune diseases, and systemic infections.[1] This leads to failed cerebral autoregulation and breakthrough hyperfusion. Increased vascular permeability may result from damage to the microvascular endothelium caused by an excess of circulating cytokines.Hydrostatic leakage and fluid extravasation and macromolecules enter the nearby brain interstitium through damaged arteriolar walls, causing vasogenic (as opposed to cytotoxic) edema. However cytotoxic components, hemorrhage, and microvascular injury may also appear in atypical or severe presentations.[2]

Since complete recovery is usually attained with prompt treatment of the initiating condition early detection of PRES is essential. However, long-term issues like infarction, blindness, or persistent cognitive decline can arise from a failure to recognize abnormal imaging presentations.

Classical PRES typically demonstrates bilateral parieto-occipital vasogenic edema, often involving the cortical–subcortical junction. However, variant forms extend beyond posterior regions, affecting the frontal and temporal lobes, cerebellum, basal ganglia, thalami, brainstem, corpus callosum and spine (rarely).[3] These atypical patterns may also show restricted diffusion or haemorrhage, potentially mimicking stroke, demyelinating disease or reversible cerebral vasoconstriction syndrome (RCVS). Because such presentations deviate from the expected posterior distribution, recognizing these imaging variants is essential for timely and accurate diagnosis.[1]

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DISCUSSION

1.CLASSIC/TYPICAL PRES

INCIDENCE: Classic PRES is the most commonly occurring type with bilateral parieto-occipital lobe involvement as its hallmark. It can affect the patients of all ages with the peak age of 20-40 years.

IMAGING FEATURES: Symmetrical cortical and subcortical hyperintense signals on T2 and FLAIR-weighted MR imaging in the parieto-occipital lobes of both hemispheres are the most often reported abnormality in PRES. These regions exhibit less attenuation on CT scans and are often hypointense on corresponding T1-weighted MR imaging. Similar areas of altered signal intensity can also be seen in other locations such as the frontal lobes, cerebellum, brainstem and basal ganglia.[4] Diffusion restriction is not seen in majority of the cases, however patchy multifocal areas of decreased ADC values can be seen along with blooming foci in cases of simultaneous cytotoxic injuries. (IMAGE 1A, 1B)

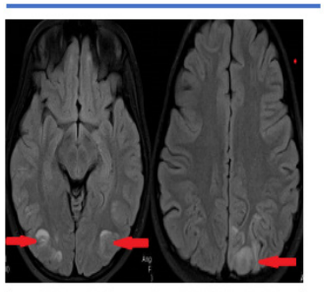

IMAGE 1A – Typical PRES – Axial FLAIR images showing hyperintense signal involving bilateral parieto-occipital lobe (Red arrow).

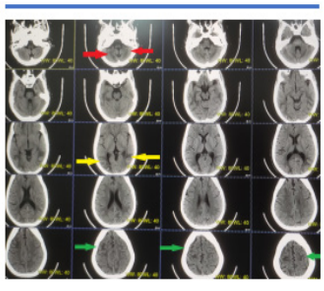

IMAGE 1B – Axial CT images showing hypodensity in cerebellar white matter (red arrow), parieto-occipital white matter (yellow arrow) and superior / inferior frontal lobe (green arrow).

There are few distinct imaging patterns in classical PRES as follows:

A.PARIETAL-OCCIPITAL PATTERN: It is the most common pattern involving over 90% of the PRES cases. CT shows patchy cortical/subcortical hypodensities in parietal occipital lobe. On MRI these corresponds to T1 hypointensities and T2/FLAIR hyperintensities predominantly without diffusion restriction. Angiographic findings may show diffuse vascular narrowing or beaded appearance. If the screening NECT is normal and PRES is suspected on clinical grounds, an MR scan with DWI and T2* (gradient T2W) in addition to the routine sequences (T1 and T2/FLAIR) should be obtained.[5]

B.HOLOHEMISPHERIC WATERSHED PATTERN: It is one of the primary variations that exhibit a linear pattern of vasogenic edema in the frontal, parietal, and occipital lobes, with the temporal lobes being less affected. Linearly arranged focal vasogenic edema or a "string-of-pearl" appearance can be seen representing involvement of the deep white matter watershed area.[6] It seems to define the junction between the anterior cerebral territory and middle cerebral territory, which suggests a distribution between the medial (ACA, PCA) and lateral cerebral branches (MCA).[3]

C.SUPERIOR FRONTAL SULCUS PATTERN: In this pattern, isolated superior frontal sulcal involvement is seen without frontal lobe extension. It can be called a subtype of holohemispheric pattern. Regions of localized or patchy white matter vasogenic edema or non confluent patches of cortical vasogenic edema along the superior frontal sulcus can provide an intermediate impression of this pattern (IMAGE 2).[3] Reduced perfusion between the distal ACA and MCA distributions could be the cause of isolated edema in the superior frontal sulcus.

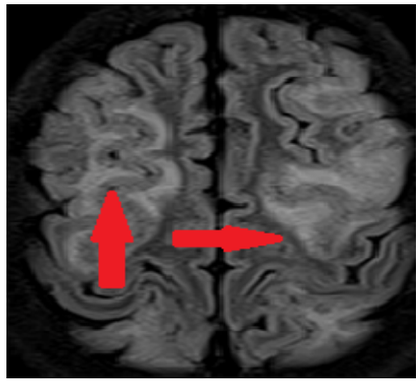

IMAGE 2– Frontal pattern of PRES – Axial FLAIR images showing hyperintense signal at level of inferior frontal sulcus and lobe on either side (red arrow).

D.PARTIAL EXPRESSION OF PRES: The absence of signal intensity abnormality in either the bilateral parietal lobes or the bilateral occipital lobes is referred to as partial manifestation of pres. This variability can be due to the differences in arterial anatomy or preexisting vascular disease.

E.ASYMMETRIC EXPRESSION OF PRES: It includes unilateral involvement of either parietal or occipital lobe or both. Partial and asymmetrical PRES could be related to underappreciation of a subtle PRES pattern in patients with infection, sepsis, shock, or an intrinsic difference in the mechanism that led to more prominent vasogenic edema or severe hypertension.[7]

F.PEDIATRIC PRES:PRES has been rarely reported in paediatric patients due to less prevalence of hypertension. There are reports that suggest PRES in paediatric patients with renovascular diseases, immunosuppressive therapy, hematologic malignancies and systemic diseases such as leukaemia, aplastic anaemia, solid tumors and autoimmune diseases.[8] PRES should be considered in children presenting with encephalopathy, seizures, raised blood pressure or renal disease as a delay in making the diagnosis and initiating the treatment may result in a permanent neurological deficit.[9]

The degree of vasogenic edema in PRES has been categorized using FLAIR images by Hinchey et al.[10] and Mckinney et al. [11] as:-

i.MILD PRES: Characterized by cortical or subcortical

white matter edema without herniation, mass effect, parenchymal bleeding, or minor involvement of the brainstem, cerebellum, or basal ganglia.

ii.MODERATE PRES: It is characterized by confluent edema that extends from the cortex to the deep white matter without extending to the ventricular edge or by modest involvement of the brainstem, basal ganglia, or cerebellum. Moderate was also used to describe a minor mass effect without any herniation or midline shift, especially if there was parenchymal bleeding.

iii.SEVERE PRES: In this type, either herniation or mass effect is present or involvement of all three of the following structures – the cerebellum, brainstem, and basal ganglia is seen. It can be life threatening or with irreversible and permanent damage.

DIFFERENTIAL DIAGNOSIS: The primary differential of PRES includes Reversible cerebral vasoconstriction syndrome. The limited involvement of a solitary sulcus or few adjacent sulci is the differentiating feature from PRES. The other differentials include Vasculitis (differentiating feature being random or asymmetrical distribution of lesions and lack of parietal occipital predominance), hypoglycemia (differentiated on the basis of clinical profile), acute cerebral ischemia infarction (cytotoxic edema leading to diffusion restriction in an arterial territory), SMART syndrome (typically unilateral with cortical thickening , gyral enhancement and mass effect) , cerebral amyloid angiopathy (differentiated on basis of presence of multiple microhaemorrhages at grey white matter junction) and thrombotic microanagiopathy (differentiated on the basis of clinical profile).

2.ATYPICAL PRESS AND VARIANTS

INCIDENCE: Atypical PRES involves regions outside the classic parieto-occipital distribution, including the frontal and temporal lobes, cerebellum, basal ganglia, thalami, and brainstem.1 Other atypical sites include deep white matter, corpus callosum splenium and cervical spinal cord. These presentations are clinically important because they can mimic stroke, encephalitis, or demyelinating disease, leading to delayed or incorrect diagnosis.2 Recognizing such atypical patterns is crucial, as timely identification directly influences management and helps prevent irreversible complications such as infarction or persistent neurological deficits.

IMAGING FEATURES: The distinctive atypical variants of PRES include:

A.ATYPICAL UNILATERAL

B.CENTRAL PRES: Characterized by involvement of the brainstem (especially the pons) or basal ganglia without involvement of the cortical or subcortical white matter (IMAGE 3).

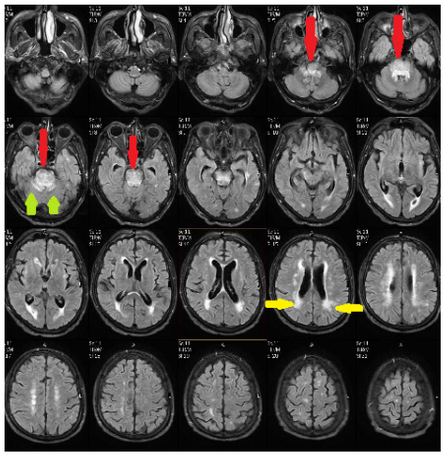

IMAGE3: Central PRES – Axial FLAIR images showing hyperintense signal involving brainstem (red arrow) and cerebellar hemisphere (blue arrow). In a patient receiving chemotherapy. In addition extensive chronic white matter ischemic foci are seen (yellow arrow).

A. HEMORRHAGIC PRES: The incidence of intracranial hemorrhage in PRES is approximately 15%. 12 In PRES, there are three different types of hemorrhage: intraparenchymal hematoma, sulcal subarachnoid hemorrhage, and small patchy hemorrhage (IMAGE 4). In two of its patients, Mazamaesso et al. 12 reported unusual PRES characteristics that resembled tumors. One of them imitated a primary brain tumor, and the other a cerebral hemorrhagic metastasis. The pathophysiology behind hemorrhagic PRES has not yet been

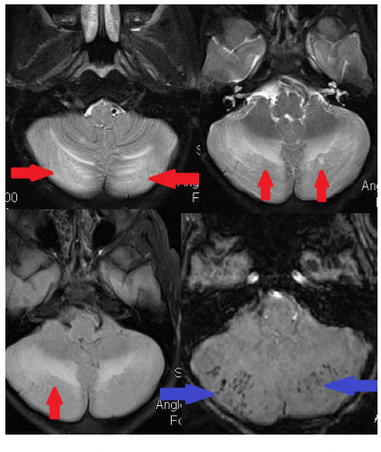

IMAGE 4: Haemorrhagic PRES – Axial FLAIR and GRE images showing FLAIR hyperintense signal involving bilateral cerebellar hemisphere and fissures (red arrow) with foci of susceptibility on GRE images (blue arrow).

a.well understood.

B.SPINAL CORD INVOLVEMENT (PRES-SCI):
It is comparatively uncommon but probably underappreciated variation of PRES. Patients with non-enhancing longitudinally widespread T2-hyperintense spinal cord lesions and severe acute hypertension should be evaluated for this diagnosis (IMAGE 5). Patients may show myelopathy-related signs and symptoms or, on the other hand, they may show only mild disproportionate myelopathic features in spite of noticeable MRI abnormalities.[13]

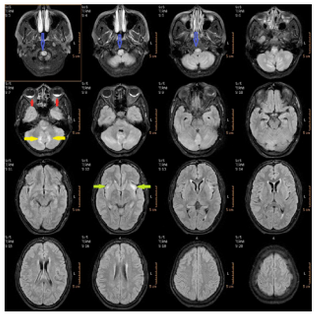

IMAGE 5: PRES SCI – Axial FLAIR images in pediatric patient with renovascular accelerated hypertension showing hyperintense signal in brainstem and cervical spinal cord (blue arrow), cerebellar hemisphere (yellow arrow) and bilateral insular cortex. Sub-hyloid hemorrhage is also seen in both eye globes (red arrow).

DIFFERENTIAL DIAGNOSIS: Central PRES like imaging findings can be seen in ischemia, encephalitis, metabolic disorders, and demyelination. It requires careful attention to diffusion characteristics, symmetry, enhancement patterns, clinical context, and reversibility. Key mimics of hemorrhagic PRES include hypertensive intracerebral hemorrhage, venous sinus thrombosis, cerebral amyloid angiopathy, hemorrhagic encephalitis, reversible cerebral vasoconstriction syndrome(IMAGE 6), and coagulopathy-related hemorrhage. Acute transverse myelitis, neuromyelitis optica spectrum disease (NMOSD), spinal cord infarction, multiple sclerosis, and compressive myelopathy are important differential diagnoses of

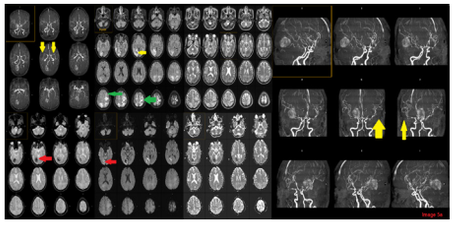

IMAGE 6:REVERSIBLE VASOCONSTRICTION SYNDROME IN PERIPARTUM ANGIOPATHY-- Axial DWI, ADC, FLAIR, T2, GRE images with reformatted MRA images showing intraparenchymal bleed in right temporo-occipital lobe (Red arrow). Multiple FLAIR hyperintense foci are noted in bilateral frontal-parietal and right temporo-occipital lobe (Green arrow). Multiple areas of stenosis with post stenotic nodular dilatation noted in bilateral middle cerebral artery on MRA.

PRES-SCI. Clinical context, the presence of vasogenic edema as opposed to inflammatory or ischemic edema, normal CSF, reversibility, and the lack of necrosis or long-segment enhancement typical of inflammatory or ischemic diseases are all factors helpful in differentiation.

CONCLUSION

Beyond the typical posterior appearance, PRES encompasses a wide range of clinico-radiologic conditions. It is crucial to comprehend its various imaging patterns, such as atypical, central, hemorrhagic, and spinal variations, in order to prevent misdiagnosis and severe neurological damage. Early recognition supported by MRI features helps distinguish PRES from ischemic, inflammatory, infectious, and metabolic mimics.[14] Increased awareness of atypical symptoms is essential for rapid intervention and better outcomes because this condition is treatable when treated promptly. Maintaining focus on thorough imaging evaluation will improve diagnostic precision and direct suitable clinical decision-making.

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