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RESEARCH ARTICLE

Clinical, Etiological and Neuroradiological Profile of Children Presenting with Posterior Reversible Encephalopathy Syndrome (PRES) in a Tertiary Care Center in South India. A Retrospective Study

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Abstract

Posterior Reversible Encephalopathy Syndrome [PRES], is a neurological disorder that is often reversible but has long term neurological consequences. With mortality rates as high as 16%, the pathophysiology of PRES involves two primary mechanisms: vasospasm and cytotoxic edema; and it is commonly accompanied by elevated blood pressure, disturbance is cerebral autoregulation and onset of vasogenic edema. PRES includes PCA territory infarcts, venous thrombosis, demyelinating disorders, vasculitis and encephalitis. A retrospective study was conducted on children admitted between the period of December 2022 to December 2023 at the Institute of Child Health diagnosed with PRES. The data regarding clinical and neuro-radiological presentation, etiology and outcomes were assessed on 30 children who were admitted in this period. Treatment was administered based on clinical observation and varied according to associated medical condition present. PRES should be considered in differential diagnosis in children with complaints of seizures, headache and altered mental states.

Keywords: Investigation of PRES, Management of PRES, Posterior Reversible Encephalopathy Syndrome (PRES), Prevalence, Outcomes

1. Introduction

Posterior Reversible Encephalopathy Syndrome (PRES) is a neurologic condition characterized by vasogenic edema in the brain, presenting with symptoms such as seizures, visual disturbances, and altered mental states. Globally, PRES is increasingly recognized among pediatric patients, though its exact incidence and prevalence in children remain challenging to quantify due to underdiagnosis and varied presentations. Estimates suggest an increasing incidence, particularly in pediatric patients with underlying conditions such as hypertension, autoimmune diseases, renal disorders, and cancer, where PRES may arise as a complication of the disease or its treatments, especially with immunosuppressive or cytotoxic therapies[1].

In pediatric populations, outcomes of PRES are generally favorable with early diagnosis and appropriate management, as many cases resolve with minimal lasting neurological deficits. However, delayed treatment or severe cases can lead to permanent neurological damage or, in rare cases, death. Studies indicate that while mortality rates in PRES are low (ranging from 3% to 6% globally), the rates of residual

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neurological sequelae are higher, particularly in patients with severe presentations or pre-existing systemic diseases. Additionally, recurrent PRES episodes have been documented in pediatric patients, often linked to ongoing exposure to risk factors such as hypertension or chronic renal disease[2].

In India, pediatric PRES cases are increasingly reported, paralleling a rising awareness of the condition and improvements in diagnostic access, such as MRI. The incidence may be influenced by the high prevalence of risk factors such as hypertensive disorders, infections, and malnutrition-associated renal dysfunction, as well as the expanding use of immunosuppressive treatments for autoimmune and oncologic conditions in children. Despite growing awareness, challenges remain in rural areas where access to timely neuroimaging and specialized pediatric care is limited. This may lead to underreporting or delayed diagnoses, affecting outcomes. Addressing these disparities by expanding access to diagnostic facilities and enhancing physician education about PRES in children is crucial for improving recognition and management in the Indian setting.

As a reversible syndrome with a potentially severe impact on neurological health, the prompt identification and management of PRES in children are essential to ensure favorable outcomes[1].

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2. Aim and Objectives

The aim of the study was to determine the demographic data, etiology, clinical manifestation, and neuroimaging findings of posterior reversible encephalopathy syndrome in the age group up to 12 years of age and also to determine the outcome of posterior reversible encephalopathy syndrome. The objective of the study was to perform a retrospective study to assess the prevalence, clinical presentation and clinical outcomes following the treatment.

3. Review of Literature

Pathophysiology: The pathophysiology of PRES in children involves a complex interaction of factors such as endothelial dysfunction, cerebral autoregulatory failure, and systemic inflammatory responses, along with genetic and developmental influences.

3.1 Endothelial Dysfunction and Blood-Brain Barrier Disruption

The Blood-Brain Barrier (BBB) comprises endothelial cells with tight junctions, astrocyte end-feet, and basal lamina, maintaining brain homeostasis by restricting the movement of substances from the blood into the brain. In PRES, endothelial dysfunction compromises this barrier, resulting in vasogenic edema. Pediatric cases are often linked to immune-mediated or chemotherapy-related endothelial activation, which escalates BBB permeability and fluid extravasation into brain parenchyma[4].

3.1.1 Endothelial Cell Activation

The exposure to immunosuppressive agents, chemotherapy, or immune system dysregulation seen in conditions like lupus or nephrotic syndrome can damage endothelial cells and elevate vascular permeability. Elevated levels of pro-inflammatory cytokines, including IL-6 and TNF-α, disrupt the endothelial tight junctions and enhance the movement of proteins and fluid into the interstitial space, causing vasogenic edema[5]. In pediatric patients undergoing chemotherapy, agents such as cisplatin and tacrolimus are particularly implicated in causing endothelial injury and inducing PRES[6].

3.1.2 Vascular Endothelial Growth Factor (VEGF)

Elevated levels of VEGF, an endothelial permeability factor, are noted in patients with conditions commonly associated with PRES. VEGF disrupts endothelial junctions, enhancing leakage and fluid accumulation in the brain, thereby contributing to the vasogenic edema observed in PRES. Anti-VEGF therapies have been proposed as a potential intervention to stabilize endothelial barriers, though they are still under study and not yet widely implemented in pediatric care[7].

3.2 Cerebral Autoregulation Failure and Hyperperfusion

Autoregulatory mechanisms help maintain consistent Cerebral Blood Flow (CBF) despite fluctuations in systemic blood pressure. When autoregulation fails,

as seen in acute hypertensive episodes or in response to chemotherapy, cerebral vessels in the posterior regions become more susceptible to hyperperfusion. Hyperperfusion can damage the endothelial barrier, leading to capillary stress and plasma leakage, which causes the characteristic vasogenic edema in the posterior brain regions[1].

3.2.1 Regional Vulnerability of the Posterior Circulation

The posterior regions of the brain, supplied by the vertebrobasilar system, have lower levels of sympathetic innervation compared to anterior regions, making them more vulnerable to hyperperfusion-related injury. This susceptibility is a primary factor in the localization of edema in the parieto-occipital regions, as these areas are less capable of constricting in response to elevated blood pressures, thus failing to prevent plasma leakage into the brain parenchyma[5].

3.2.2 Impact of Hypoperfusion

While hyperperfusion is a major factor, some cases of PRES involve hypoperfusion due to vasospasm or thrombosis, which exacerbates endothelial injury. Hypoxic conditions within the brain initiate inflammatory cascades, further compromising the BBB. This dual mechanism-hyperperfusion and regional hypoperfusion-illustrates the complexities in cerebral blood flow dysregulation in PRES, especially in pediatric patients who may have underlying renal or autoimmune disorders[8].

3.3 Systemic Inflammation and Immunologic Factors

Pediatric PRES often coincides with inflammatory conditions, such as autoimmune diseases or infections, where systemic inflammation plays a significant role.

3.3.1 Cytokine-Mediated Endothelial Injury

Cytokines, especially IL-1β, IL-6 and TNF-α, increase vascular permeability and endothelial adhesion. In pediatric cases, conditions like systemic lupus erythematosus or Henoch-Schönlein purpura produce high levels of these cytokines, which compromise endothelial function and enhance BBB permeability, promoting the hallmark vasogenic edema in PRES[9].

3.3.2 Complement Activation and Oxidative Stress

Activation of the complement cascade, particularly in autoimmune disorders, produces inflammatory components like C5a, which exacerbate endothelial damage. Additionally, Reactive Oxygen Species (ROS) generated by the immune response further impair BBB integrity, triggering the edema observed in PRES[6].

4. Pediatric Neuroimaging in PRES

Imaging plays a pivotal role in diagnosing PRES, with MRI as the gold standard. Characteristic findings include symmetrical, subcortical vasogenic edema, predominantly in the parieto-occipital regions.

5. MRI and Advanced Imaging Techniques

5.1 Fluid-Attenuated Inversion Recovery (FLAIR)

FLAIR sequences are particularly sensitive to the white matter vasogenic edema characteristic of PRES. In pediatric patients, FLAIR often reveals hyperintense areas in the parieto-occipital lobes, although other areas such as the frontal, temporal, and cerebellar regions may also be affected depending on the severity[10].

5.2 Diffusion-Weighted Imaging (DWI)

DWI can help differentiate between vasogenic and cytotoxic edema, an important distinction as cytotoxic edema indicates irreversible damage. Most pediatric PRES cases present with reversible vasogenic edema, characterized by increased diffusivity on DWI sequences, supporting the potential for full recovery if promptly managed[2].

5.3 Perfusion Imaging

Recent advances in perfusion MRI have enabled more precise detection of cerebral perfusion abnormalities. Hyperperfusion in the posterior brain regions is commonly observed in PRES, while hypoperfusion may indicate regions with vasospasm or vascular compromise. Perfusion imaging may also help in monitoring treatment response and detecting early recurrence[11].

5.4 Susceptibility-Weighted Imaging (SWI)

SWI is useful in detecting microhemorrhages or subtle vascular changes that may accompany PRES, especially in severe cases. In pediatric patients with chronic hypertension or immunosuppressive therapy, SWI can identify these changes early, guiding modifications in therapy to prevent complications[12].

6. Radiological Presentation

PRES typically manifests with vasogenic edema in the posterior brain regions, particularly the parieto-occipital lobes. This distribution is thought to arise due to the unique susceptibility of the posterior circulation, which has less sympathetic innervation than anterior regions and is therefore more vulnerable to changes in blood pressure and autoregulatory failure[13]. MRI remains the gold standard imaging modality for diagnosing PRES, with findings usually highlighting bilateral, symmetrical hyperintensities in the parieto-occipital white matter on T2-weighted and FLAIR sequences[9].

Though the parieto-occipital regions are most commonly affected, PRES can also involve other areas, including the frontal and temporal lobes, cerebellum, basal ganglia, and brainstem. In severe or atypical cases, more extensive or asymmetric involvement may occur, including in the cortex and deep gray matter, which could indicate a higher risk for complications or prolonged recovery[14]. The extent of involvement on neuroimaging correlates with the clinical severity of PRES, underscoring the importance of timely imaging for prognosis and management.

7. Clinical Presentation

Posterior Reversible Encephalopathy Syndrome (PRES) presents with a range of neurological symptoms that reflect the extent and areas of cerebral involvement. Typical symptoms include headache, visual disturbances, seizures, altered mental status, and focal neurological deficits. These symptoms are generally acute or subacute, often developing over hours to days, and vary according to the severity of the underlying causes, such as hypertension, autoimmune disorders, or cytotoxic treatments[1].

7.1 Headache

Headaches in PRES are usually diffuse and severe, associated with elevated intracranial pressure or hypertensive crises. This is one of the earliest and most common symptoms and often coexists with nausea and vomiting.

7.2 Visual Disturbances

Vision changes are a hallmark of PRES and may include blurred vision, hemianopia, cortical blindness, and even complete loss of vision in severe cases. These visual deficits are attributed to the involvement of the occipital and parietal lobes and usually improve with timely intervention[15].

7.3 Seizures

Seizures are frequent in pediatric and adult cases of PRES, occurring in approximately 60-75 % of cases. They are often generalized tonic-clonic but can also present as partial seizures. Status epilepticus can occur in severe cases, necessitating prompt treatment to prevent prolonged seizures and further complications[16].

7.4 Altered Mental Status

Altered consciousness is another frequent feature, ranging from confusion and agitation to drowsiness, stupor, and even coma in extreme cases. Cognitive impairment and disorientation are common, particularly in pediatric patients with severe or extensive brain involvement[14].

7.5 Focal Neurological Deficits

Focal deficits such as hemiparesis, aphasia, or ataxia may arise depending on the areas of the brain affected. These deficits, although less common, can indicate more widespread or atypical PRES involvement.

8. Complications

Posterior Reversible Encephalopathy Syndrome (PRES) is often characterized by a favorable prognosis when identified early and treated promptly. However, complications can arise, particularly in severe or delayed cases, potentially leading to irreversible neurological deficits and other serious outcomes. Complications of PRES can range from prolonged

neurological symptoms to more severe sequelae, including hemorrhage, cytotoxic edema, and chronic epilepsy. Understanding these complications is crucial for effective patient management and prognosis assessment.

8.1 Intracerebral Hemorrhage

Hemorrhage is one of the more severe complications of PRES, observed in approximately 15-20 % of cases[17]. The hemorrhagic component in PRES can vary in severity, from small petechial hemorrhages to large parenchymal hemorrhages, potentially increasing morbidity and affecting recovery. Pathophysiologically, it is thought that the loss of autoregulation and increased vascular permeability in PRES may lead to microvascular injury, predisposing patients to hemorrhage. Hemorrhage in PRES is often associated with higher blood pressure levels and may indicate a poorer prognosis, necessitating aggressive blood pressure control and close monitoring in affected patients[9].

8.2 Cytotoxic Edema and Infarction

Although PRES is primarily associated with vasogenic (extracellular) edema, cytotoxic (intracellular) edema can occur in more severe cases, potentially leading to ischemia and infarction. Cytotoxic edema reflects a breakdown of cellular integrity and is usually irreversible, unlike vasogenic edema, which can typically be resolved with proper management[18]. The presence of cytotoxic edema is associated with poorer outcomes and increased risk of long-term neurological deficits, such as motor and sensory impairments, if it progresses to ischemic infarction[13].

8.3 Seizure Recurrence and Status Epilepticus

Seizures are common in PRES, and although they are typically manageable, a subset of patients may develop status epilepticus or recurrent seizures even after the initial episode has resolved. Recurrent seizures may be more common in children and individuals with extensive or atypical PRES involvement, including those with hemorrhagic complications. Persistent seizures and status epilepticus are associated with increased morbidity and may lead to chronic epilepsy if not managed appropriately[15].

8.4 Persistent Neurological Deficits

Although PRES is termed "reversible," some patients experience persistent neurological deficits, particularly when complications like hemorrhage or infarction occur. Deficits may include residual motor or sensory impairments, visual field deficits, and cognitive impairment. These sequelae are often linked to the severity of the initial episode and any delays in treatment. Persistent deficits have a substantial impact on quality of life, particularly in pediatric patients, where they may interfere with development and daily functioning[2].

8.5 Chronic Cognitive and Behavioral Changes

Cognitive deficits and behavioral changes have been reported as long-term complications in PRES, particularly in pediatric patients. These may include difficulties with memory, attention, and executive functioning, which can interfere with academic performance and social interactions. Although these deficits are generally mild, they can be challenging to detect and manage, especially in children who may not fully communicate or recognize their symptoms[9]. Persistent cognitive and behavioral changes may require ongoing cognitive therapy and rehabilitation.

8.6 Risk of Recurrence

Recurrence of PRES is rare but possible, especially if patients remain exposed to underlying risk factors, such as chronic hypertension, immunosuppressive therapy, or renal disease. Recurrence may be more common in patients with autoimmune diseases, such as systemic lupus erythematosus, who are on long-term immunosuppressive treatment[8]. The risk of recurrence necessitates vigilant monitoring and modification of predisposing factors when possible.

It has been suggested that the accumulation of fluid, toxins and metabolic waste may promote the onset of PRES in patients with CRF. This may be due to volume-dependent hypertension caused by fluid retention and vascular endothelial injury resulting from the accumulation of toxins and metabolic waste[18].

9. Materials and Methods

The present study was designed as a retrospective cohort study at the Institute of Child Health and Hospital for children, Egmore from the period December 2022

to December 2023 for 1year. The children aged upto 12 years of age attending the outpatient department and admitted in the wards with clinical features of posterior reversible encephalopathy syndrome were considered for the study. Written informed consent was obtained from all the patients at the time of admission.

Totally 30 children were clinically suspected for PRES and were confirmed by Assistant professor and Professor of Radiology at institute of child health.

A total of 30 children (22 males and 8 females) of age 7.69±3.24 years (Mean±SD) were included in the study.

10. Results (Including Observations)

In the above period, 30 children (Age: 7.69±3.24 years; Male: 22, Female: 8) were admitted. Etiology of PRES included 13.3% (n=4) of central, 53.3% (n=16) were PRES secondary to renal complications such as crescentic glomerulonephritis, post infective glomerulonephritis, rapidly progressive glomerulonephritis, chronic kidney disease and 33.5% (n=10) were secondary to other complications like snake bite (acute kidney injury), systemic lupus erythematosis, chemotherapeutic agents, factor 7 deficiency. Clinical presentation included seizures (n=15 [50%]), altered mental states (n=12 [37.5%]) and headache (n=14 [43.75%]), blurring of vision (n=2) [6.6%]. MRI signatures suggested common involvement of parieto-occipital regions (n=22 [68.75%]). 30 children had hypertension (n=22 [68.75%] Atypical presentation included frontal lobe (n=4 [13.3%]), temporal lobe (n=2[6.6%]), brainstem involvement (n=2) [6.6%]. 90% [n=27] cases improved, 10% cases [n=3] had fatality as shown in figure 1.

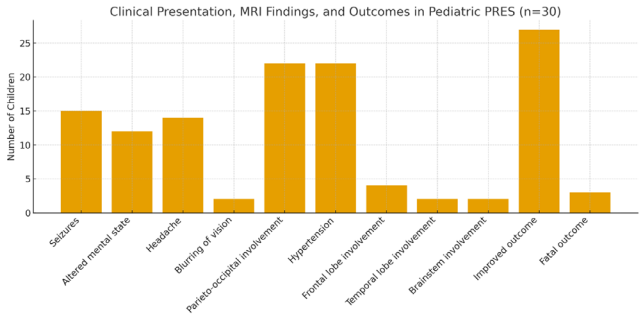

Figure 1. The distribution of clinical features, MRI involvement, and outcomes among the study population.

11. Discussion

We further propose that the following future strategies to enhance early detection, refine risk assessment, and develop standardized treatment protocols to improve patient outcomes.

11.1 Advances in Early Detection and Imaging Techniques

Early detection of PRES is crucial to prevent complications such as hemorrhage and infarction. MRI remains the preferred modality for diagnosing PRES, especially T2-weighted and FLAIR sequences that highlight the characteristic vasogenic edema[13]. However, Diffusion-Weighted Imaging (DWI) and Susceptibility-Weighted Imaging (SWI) are emerging as essential tools, helping to differentiate vasogenic from cytotoxic edema and detect microhemorrhages that may signal more severe or atypical presentations of PRES[17]. Future imaging advancements may involve automated AI-based tools that can identify early radiological signs of PRES, offering quicker diagnostics in emergency settings.

11.2 Biomarker Development for Early Diagnosis

The search for reliable biomarkers that could indicate PRES before the onset of neurological symptoms is ongoing. Biomarkers of endothelial dysfunction, including circulating endothelial cells and markers of blood-brain barrier integrity, are promising for early PRES identification, as PRES is often triggered by endothelial injury[6]. Inflammatory markers and cytokine profiles are also being studied to identify patients at risk, particularly in those undergoing immunosuppressive therapies or with autoimmune disorders, where endothelial dysfunction plays a significant role.

11.3 Risk Stratification and Preventative Monitoring:

Risk stratification models can aid clinicians in identifying individuals at high risk for PRES, enabling close monitoring and preemptive management. For instance, patients with severe hypertension, eclampsia, renal disease, or undergoing chemotherapy are known to be at higher risk. Regular blood pressure monitoring

and neurologic assessments could help mitigate the likelihood of progression in high-risk individual[1]. Machine learning models may further aid in identifying risk patterns and in customizing monitoring protocols.

11.4 Timely Intervention and Management Protocols

Although hypertension management remains central to treating PRES, the development of standardized treatment protocols will help ensure consistent and effective care. Current guidelines suggest gradual blood pressure reduction to prevent further cerebral insult, and newer antihypertensive agents are under investigation to optimize blood pressure control with minimal side effects[2]. Additionally, the cessation or dose modification of triggering agents such as immunosuppressants or cytotoxic drugs should be standardized, with protocols providing guidance on tapering or substituting medications safely.

11.5 Long-Term Follow-Up and Neurorehabilitation

Future strategies also emphasize the importance of long-term follow-up and neurorehabilitation, particularly for pediatric and young adult patients who may experience cognitive or behavioral complications. Regular cognitive assessments, coupled with early neurorehabilitation, could help mitigate these impacts. Integrating these follow-ups into treatment guidelines would provide patients with comprehensive care beyond the acute management of PRES[8].

New imaging techniques, such as vessel wall imaging and arterial spin labeling, are proving useful in reversible cerebral vasoconstriction syndrome and are giving new insights into the pathophysiology of this condition[19].

11.6 Education and Awareness in Clinical Practice

Increasing awareness among clinicians about the diverse presentations of PRES is essential to reducing misdiagnosis or delays. Since PRES often occurs in critical care settings, targeted training for healthcare providers on recognizing early symptoms such as headache, confusion, and visual disturbances will improve diagnosis and outcomes. Educational initiatives for clinicians and caregivers about the risks, particularly for patients in high-risk categories (e.g., hypertensive, renal impairment), can enhance vigilance and prompt intervention.

Because of a difference in the pathomechanism of PRES, children with normal Blood Pressure (BP) may be missed for the diagnosis, resulting in unnecessary investigations and long-term complications from delayed treatment[3].

12. Summary and Conclusion

Posterior Reversible Encephalopathy Syndrome (PRES) presents significant diagnostic and management challenges, especially due to its wide-ranging clinical presentation and the potential for irreversible complications if not treated promptly. The present study demonstrated that parieto-occipital lobe had the commonest involvement in PRES. Blurring of vision improved in both cases of snake bite and factor 7 deficiency. PRES should be considered in differential diagnosis in children with complaints of seizures, headache and altered mental states.

In our study, Parietooccipital lobe had the commonest involvement. Blurring of vision improved in both cases of snake bite and factor 7 deficiency. chronic kidney disease was the commonest cause of PRES.

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