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CLINICORADIOLOGICAL FINDINGS AND OUTCOME OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN CHILDREN: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background and Objective:
Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiological syndrome characterised by altered sensorium, seizures, visual impairment and behavioural changes. The objective of the study was to evaluate clinicoradiological findings and outcome of PRES in children.

Methods:
A prospective cross-sectional study was conducted at department of pediatric neurology, the Children's Hospital Lahore from October 2020 to September 2023. Clinical and demographic data were colllected on a structured proforma. Investigations comprised complete blood count, biochemistry, urinalysis, complement levels, inflammatory markers, immune panel, lipid profile and abdominal ultrasonography. All patients underwent MRI brain. Outcome was categorized as complete recovery or persistence of neurological deficits and/or residual radiological abnormalities at six months. Data were analyzed using SPSS version 23.

Results:
Thirty patients enrolled with male predominance (53.3%; male-to-female ratio of 1.14:1) and mean age of 10.86 ± 2.22 years. Hypertension was universal followed by seizures (93.3%), headache (83.3%), altered sensorium/aphasia (80%), visual impairment (70%), pyramidal signs (70%), peripheral edema (66.7%) and motor deficits (43.3%). Renal disorders accounted for 83.3% cases, most commonly post-infectious glomerulonephritis (43.3%). MRI revealed reversible vasogenic Edema on T2-weighted/FLAIR sequences, predominantly in occipital (100%) and parietal lobes (90%). 6.7% patients had ischemic changes correlating with visual loss.

Conclusion:
PRES in children presents with diverse neurological manifestations associated with hypertension secondary to renal diseases. MRI plays a pivotal diagnostic role. Early recognition with timely management is essential to prevent deficits.

Key words: Posterior reversible encephalopathy syndrome, Hypertension, Renal disorders, Vasogenic edema, Magnetic resonance imaging

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is an uncommon but increasingly recognized neurological disorder in children. It is frequently associated with acute hypertension due to renal disorders and the use of immunosuppressive or chemotherapeutic agents. Although first described in adults, pediatric PRES has distinct clinical and radiological features that emphasize immediate attention. In United States, the estimated incidence is about 0.04% of pediatric hospitalizations.[1]

The syndrome typically presents with a spectrum of neurological manifestations including seizures, focal neurological deficits such as hemiparesis, visual disturbances, headaches and altered sensorium. Signs of raised intracranial pressure may also be observed.[2]

The underlying pathophysiology remains incompletely understood. Howeve, disruption of cerebral autoregulation and endothelial dysfunction triggered by hypertension or exposure to toxic agents are believed to result in reversible vasogenic edema, particularly in watershed areas of the brain. Magnetic resonance imaging (MRI) plays a pivotal role in diagnosis. Classical findings include bilateral, symmetrical

white matter hyperintensities in the parieto-occipital regions on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences, though anterior circulation territories may also be involved. These radiological features when correlated with clinical suspicion establish the diagnosis of PRES.[3-5]

Management in children focuses on supportive care, strict blood pressure control and treatment of underlying etiologies. Neuroprotective measures and seizure control with antiseizure medications are frequently required. Compared with adults, pediatric patients demonstrate a higher incidence of seizures and multi-organ involvement.[5,6] Despite this, outcomes are generally favorable if timely recognition and management are achieved. Poor prognostic indicators include altered consciousness at presentation, delayed diagnosis, prolonged hospitalization and MRI evidence of hemorrhagic or ischemic changes. [7,8]

In resource-limited settings, PRES remains under-recognized and under-reported in pediatric cohorts. The presence of acute neurological symptoms in association with hypertension should raise strong clinical suspicion. Early diagnosis and prompt therapeutic interventions are essential to prevent significant morbidity and mortality. This study was undertaken to delineate the etiological factors, clinical spectrum and neuroimaging features of PRES in children at our center as well as to assess hospital-based outcomes. Establishing such data will contribute to better understanding, timely recognition and the development of standardized management strategies for pediatric PRES.

METHODS

After obtaining approval from the Institutional Review Board (IRB), a cross-sectional study was conducted in the department of pediatric neurology, the Children's Hospital Lahore, a tertiary care referral center from October 2020 to September 2023. The estimated incidence of PRES in the pediatric population is 0.4 per 100,000 per year. Using the OpenEpi version 3 sample size calculator and standard statistical formula, a total sample size of 30 patients was determined, with a confidence level of 80% and confidence limits of 5%.

$n = [DEFF*Np(1-p)]/ [(d2/Z21-\alpha/2*(N-1)+p*(1-p)]$

Children aged six months to 18 years presenting with acute neurological symptoms such as seizures, altered consciousness, visual disturbances, encephalopathy and hypertension were evaluated. The diagnosis of PRES was established on the basis of clinical presentation confirmed by brain MRI findings consistent with vasogenic edema, particularly in the parieto-temporo-occipital regions. MRI protocols included T1-weighted (T1W), T2-weighted (T2W), fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI) with apparent diffusion coefficient (ADC) mapping. Imaging was reviewed independently by two senior radiologists to minimize bias. Patients with alternative diagnoses including central nervous system (CNS) infections, acute disseminated encephalomyelitis (ADEM) or stroke were systematically excluded through cerebrospinal fluid (CSF) analysis and distinct neuroimaging features.

Informed consent was obtained from parents or guardians. Demographic details, presenting complaints, past medical history, physical findings and blood pressure recordings were documented on a structured proforma. Blood pressure was measured on multiple occasions and defined as hypertension when systolic or diastolic readings equaled or exceeded the 95th percentile for age, sex and height.[9]

To evaluate underlying etiologies, routine investigations included serum electrolytes (sodium, potassium, calcium, magnesium), renal and liver function tests, serum albumin, total proteins, lipid profile and 24-hour urinary protein quantification. Immunological markers such as complement levels (C3, C4), antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were obtained where clinically indicated. Urine analysis and abdominal ultrasonography were performed in all patients. Targeted tests such as 24-hour urinary porphyrins or whole-body angiography were reserved for selected cases. In suspected CNS infections, lumbar puncture with CSF examination was performed and patients with confirmed infections were excluded.

All patients were followed at two weeks, three months and six months after discharge. Follow-up assessments included neurological examination to determine resolution or persistence of deficits. Repeat MRI scans (T1W, T2W, FLAIR) were performed at three to six months to document resolution of vasogenic edema or persistence of residual lesions. Outcomes were classified as favorable if both clinical recovery and radiological resolution were achieved. Persistence of neurological deficits or radiological abnormalities beyond six months was considered unfavorable.

Data were analyzed using SPSS version 23. Continuous variables such as age were expressed as mean ± standard deviation while categorical variables such as sex and clinical

features were presented as frequencies and percentages. Comparative analysis was performed between etiological factors, clinical presentations, radiological features and outcomes. The chi-square test was applied to evaluate associations, with a p-value < 0.05 considered statistically significant.

RESULTS

Among 30 children diagnosed with PRES, 16 (53.3%) were males, yielding a male-to-female ratio of 1.14:1. The mean age of the cohort was 10.86 ± 2.22 years. The majority of the cases, 18 patients (60%), were older than 10 years, with a noted predominance of females within this subgroup as shown in table 1.

In evaluating patients with PRES, renal disorders with deranged renal function were identified in 22 patients (73%), whereas 8 patients (27%) had non-renal etiologies with preserved renal function. Consequently, the clinical features were analyzed by categorizing patients into two groups; those with renal disorders and those without. Hypertension was present in all patients (100%). Fever was significantly more frequent among those with renal disorders (19, 63%) compared to non-renal cases (3, 10%) ($p = 0.016$). Seizures were the most common manifestation, reported in 28 patients (93%), with no group differences ($p = 0.531$). Headache occurred in 25 (83%) and visual impairment in 21 (70%) patients, both without significant differences between groups. Altered sensorium and speech loss were observed in 24 patients (80%), again showing no statistical variation. Abdominal distension was reported only in renal cases (8, 27%), nearing significance ($p = 0.055$). Peripheral edema was significantly higher in renal patients (20, 67%) compared to non-renal group ($p < 0.001$).

Motor deficits vwere noted in 13 (43%), and pyramidal signs such as hypertonia, hyperreflexia and extensor plantar responses were significantly more common in renal patients (18, 60% vs. 3, 10%; $p = 0.032$). Cranial nerve involvement (2, 7%) and brainstem dysfunction (2, 7%) were seen exclusively in the non-renal group. Cerebellar signs were uncommon (3, 10%).

Table 1 demonstrates clinical and demographic features of the study population.

Table 1: Clinical & Demographic Features of Patients with Posterior Reversible Encephalopathy Syndrome

Characteristics	Renal disorders with deranged renal function tests (n =22) n (%)	Non renal disorders with normal renal function tests (n = 8) n (%)	All (n=30)	P-value
Patient's sex			**0**	
Male	12 (40)	4 (13)	16 (53)	0.574
Female	10 (33)	4 (13)	14 (47)	
Patient's age group				
5 to 10 years	11 (37)	1 (3)	12 (40)	0.099
More than 10 years (>10-15 years)	11 (37)	7 (23)	18 (60)	
Sign & Symptoms				
Fever	19 (63)	3 (10)	22 (73)	0.016
Seizures	20 (67)	8 (27)	28 (93)	0.531
Headache	18 (60)	7 (23)	25 (83)	0.595
Visual impairment	17 (57)	4 (13)	21 (70)	0.161
Speech loss	19 (63)	5 (17)	24 (80)	0.300
Hypertension	22 (73)	8 (27)	30 (100)	-
Abdominal distension	8 (27)	0	8 (27)	0.055
Peripheral Edema	20 (67)	0	20 (67)	0.000
Motor deficit	11 (37)	2 (7)	13 (43)	0.407
Cranial nerve involvement	0	2 (7)	2 (7)	0.064
Pyramidal signs	18 (60)	3 (10)	21 (70)	0.032
Cerebellar signs	2 (7)	1 (3)	3 (10)	1
Brainstem dysfunction	0	2 (7)	2 (7)	0.064
Respiratory failure	0	1 (3)	1 (3)	0.267
Altered sensorium	19 (63)	5 (17)	24 (80)	0.3

Male: Female.....1.14:1
Mean age ....10.86 years ± 2.22

All patients underwent detailed laboratory evaluation to determine etiologies of PRES. Low hemoglobin was found in 12 patients (40%) while leukocytosis with neutrophil predominance was present in 20 (67%). Electrolyte analysis revealed hypokalemia in three (10%) and hyperkalemia in 27 (90%). Low serum albumin was noted in 18 (60%). Dyslipidemia with elevated cholesterol and triglycerides occurred in three (10%). Raised ASO titers were detected in five (17%) and ANA and anti-dsDNA positivity in five (17%) each. Low complement (C3, C4) levels were observed in 18 (60%), elevated CRP in 7 (23%), renal parenchymal disease in 22 (73%) and cardiac dysfunction in 1 (3%) as shown in table 2. Brain MRI with multiple sequences (T1W, T2W, FLAIR, DWI, ADC) revealed lesions in the form of hyperintensities in all patients, consistent with PRES. Lesions predominantly involved posterior brain regions, especially watershed areas appreciated as hyperintense signals on T2W/FLAIR in the occipital lobes of all patients (30, 100%), parietal lobes in 27 (90%) and temporal lobes in 20 (66.7%) as shown in table 2 and figure 1. Additional involvement included the frontal lobes in 10 (33.3%) and cerebellar hemispheres in 15 (50%). DWI and ADC sequences were largely unremarkable, though small ischemic foci in occipital, parietal, and frontal regions were detected in 2 patients (6.7%). These two patients were having persitent visual loss due to this ischmeic damage ocurred due to PRES.

Table 2. Laboratory Investigations in Patients With PRES				
Investigations	**Renal disorders with deranged renal function tests (n =22) n (%)**	**Non renal disorders with normal renal function tests (n = 8) n (%)**	**All (n=30) n (%)**	**P-value**
Complete blood count				
(Hb) Low	12 (40)	0	12 (40)	0.01
High count with predominant neutrophils (WBC)	20 (67)	0	20 (67)	0
Urine complete examination	21 (70)	0	21 (70)	0.032
(hematuria, proteinuria) Serum electrolyte levels				
Hypokalemia	3 (10)	0	3 (10)	0.545
Hyperkalemia	19 (63)	8 (27)	27 (90)	
Serum albumin level (Low)	18 (60)	0	18 (60)	0
Serum cholesterol and triglyceride level (High)	3 (10)	0	3 (10)	0.545
ASO titer (Raised)	5 (17)	0	5 (17)	0.287
ANA (Positive)	2 (7)	3 (10)	5 (17)	0.102
Anti-dsDNA (Positive)	2 (7)	3 (10)	5 (17)	0.102
C3 and C4 levels (Low)	15 (50)	3 (10)	18 (60)	0.21
CRP level (High)	7 (23)	0	7 (23)	0.143
Imaging Studies				
USG KUB (Renal parenchymal disease)	19 (63)	3 (10)	22 (73)	0.016
Echocardiography (Cardiac dysfunction)	1 (3)	0	1 (3)	1
Follow-up MRI requirement			30	
All lesions disappear	19 (63)	5 (17)	24 (80)	0.175
Residual lesions persist	3 (10)	3 (10)	6 (20)	
MRI brain findings as hyperintensities on T2-weighted and FLAIR sequence				
Bilateral occipital area	22(74)	8(26)	30 (100)	1
Bilateral temporal areas	15 (50)	5 (17)	20 (67)	1
Bilateral parietal areas	22 (73)	5 (17)	27 (90)	0.014
Bilateral frontal areas	9 (30)	1 (3)	10 (33)	0.21
Cerebellar involvement	13 (43)	2 (7)	15 (50)	0.215
Diffusion weighted images (DWI)	2 (7)	0	2 (7)	1

Evaluation of underlying disorders showed acute glomerulonephritis (AGN) as the most common cause affecting 13 patients (43%). Systemic lupus erythematosus (SLE) was seen in 5 (17%), systemic vasculitis in 3 (10%) and chronic kidney disease in 4 (13%). Less frequent causes included acute intermittent porphyria (2, 7%), atypical nephrotic syndrome (2, 7%) and post-chemotherapy PRES (1, 3%). Overall, renal disorders predominated as contributors to pediatric PRES.

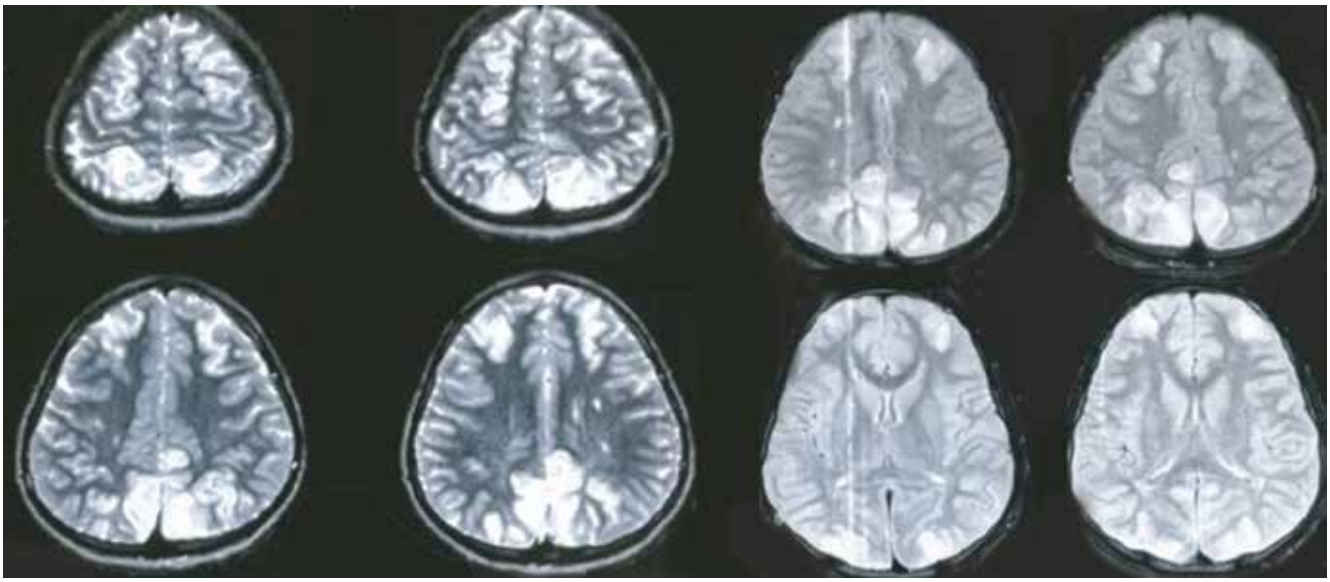

Figure 1. Magnetic resonance imaging; (a) T2W sequence showing hyperintensities in bilateral parietal, frontal and occipital areas. (b) FLAIR sequence showing hyperintensities in both anterior and posterior areas of bilateral parietal, frontal and occipital areas.

Patients were treated according to underlying conditions with antihypertensive therapy, seizure control and neuroprotective measures. Clinical monitoring emphasized recovery of consciousness, seizure remission, visual improvement and blood pressure control. Follow-up MRI performed three to six months later showed resolution of hyperintensities in 24 patients (80%) while six (20%) had persistent lesions. Outcomes were favorable in most, though two patients exhibited persistent visual deficits due to occipital ischemic injury with diffusion restriction on initial imaging.

DISCUSSION

PRES in children is a clinico-radiological entity caused by dysregulated hemodynamics and reversible vasogenic edema in posterior watershed regions, often triggered by hypertension secondary to renal disorders, chemotherapy or toxins causing endothelial injury.[10]

In the present study, we evaluated clinical symptoms, radiological features and underlying disorders causing PRES and compared our findings with existing literature. In our cohort, PRES was more common in males and in older children with a mean age of 10.83 years. However, when stratified by age, females showed a higher prevalence beyond 10 years. This observation aligns with previous reports where PRES has similarly been described as more frequent in females among older children with a comparable mean age of approximately 11.8 years. Similarly D N Gera et al compared age and gender in children with PRES and reported comparable results.[11] This difference may be explained by hormonal changes during puberty as well as the higher frequency of autoimmune conditions including renal disorders in females of this age group. [6,12]

Among clinical presentation of PRES our cohort, the most striking feature was hypertension documented in all children. This observation is consistent with reports from other centers where hypertension has been described in the majority of pediatric cases with prevalence ranging from 90% to nearly 100%.[13-15] Other clinical manifestations in decreasing order of frequency included headache, altered sensorium, seizures and visual disturbances. Similar patterns of clinical presentation have been reported in multiple studies in a comparable order of frequency. Given the considerable overlap of these signs and symptoms with

other neurological disorders, a high index of suspicion is essential particularly in the presence of concurrent cortical dysfunction and hypertension to ensure timely recognition and evaluation of PRES.[15]

Comprehensive investigations are warranted in cases of diagnostic uncertainty as identification of the underlying etiology is crucial for appropriate management. In our cohort, renal disorders emerged as the most common etiological factor in the pediatric population. Among these, post-infectious glomerulonephritis was the leading cause followed by lupus nephritis, chronic kidney disease presenting with hypertensive emergency and atypical nephrotic syndrome. Comparable findings have been reported by Mustafa Komur et al. who, in their 12-years experience, identified acute hypertension as the principal precipitating factor for PRES with approximately 76% of cases attributable to renal diseases leading to systemic hypertension.[12] Our observations similarly underscore renal disorders as the predominant causative factor in pediatric PRES.

In addition to renal disorders, several less common etiologies have been reported as precipitating factors for PRES. Among these, chemotherapy for acute leukemias is a recognized cause with the underlying pathophysiology attributed primarily to the direct toxic effects on the cerebral vasculature rather than to hypertension. In our cohort, one case was attributed to post-chemotherapy PRES. Similar findings have been documented in previous studies where chemotherapeutic agents represented one of the most frequently identified non-renal precipitants.[15,16] Other rare associations observed in our series included acute intermittent porphyria and systemic vasculitis such as Takayasu arteritis. These uncommon disorders have likewise been described in the literature as potential etiologies of PRES in children.[17,18]

Radiological features remain central to the diagnosis of PRES. MRI provides the most reliable assessment. The hallmark findings of PRES are hyperintense lesions on T2-weighted and FLAIR sequences most commonly affecting the bilateral occipital lobes within the posterior circulation in the watershed areas of brain. While the posterior regions are classically involved, less frequent patterns may extend to the parietal, temporal and even frontal lobes highlighting that PRES is not confined to the posterior circulation alone.6 Similar radiological patterns were observed in our pediatric cohort. Most radiological abnormalities in PRES should resolve without causing structural injury to the brain. However, ischemic changes characterized by hyperintensities on DWI with corresponding hypointensities on ADC maps indicating restricted diffusion may result in permanent and irreversible cerebral damage.
In our cohort, we examined the association between radiological features and clinical outcomes, taking into account variables including age, seizure occurrence, altered sensorium, underlying etiology and imaging characteristics. Among these, diffusion restriction in the occipital areas among two patients reflecting ischemic injury emerged as the sole factor correlated with persistent visual impairment. Similar observations have been made by Diego J and his colleagues who studied the radiological findings and clinical presentations with the outcome. In their work, apart from diffusion restrictions, other factors like status epilepticus and altered sensorium were also associated with neurological deficit and nonreversibility of clinical condition.[19]

PRES in children has variable clinical presentation with wide range of disorders responsible for the precipitation of the disorder. Good clinical knowledge with high index of suspicion can help to identify these patients timely and prompt intervention can be done to minimize the neurological deficits.

This study has some limitations. This is a single center study and for the results to be more generalized we should conduct multicenter study in order to recognize various risk factors leading to the development of PRES. All possible etiologies should be investigated by involvement of different pediatric specialties including neurology, nephrology, oncology, rheumatology and general pediatrics at various centers.

CONCLUSION

Posterior reversible encephalopathy syndrome is a reversible clinicoradiological syndrome with hypertension being the commonest precipitating factor. Although various underlying disorders can lead to PRES in children, renal disorders are amongst the most prevalent. Early recognition, timely investigation and prompt intervention to control the underlying precipitating disorder especially hypertension can reduce the complications.

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Authors' contribution:
Zia ur Rehman Muhammad: Concept, Design, Data analysis, manuscript writing
Shaila Ali: Data collection, manuscript writing
Shumaila Rafiq; Data Analysis, Manuscript writing
Javeria Raza Alvi; Data Analysis, Manuscript writing
Tipu Sultan; data interpretation, manuscript revision

All the authors have approved the final version to be published and agree to be accountable for all aspects of the work.