International Journal of Contemporary Pediatrics
Sehgal R et al. Int J Contemp Pediatr. 2026 Sep;13(9):1870-1879
https://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20262985

Review Article

Paediatric posterior reversible encephalopathy syndrome: a review of aetiologies, clinical and radiological spectrum, pathophysiology, diagnostic criteria and management

Rachna Sehgal*, Archana Kashyap, Bhavna Anand, Arpita Gupta

Department of Paediatrics, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, India

Received: 10 July 2026
Accepted: 11 August 2026

***Correspondence:**
Dr. Rachna Sehgal,
E-mail: drrachnasehgal6@gmail.com

ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a rare clinico-radiological syndrome postulated to be due to disturbed cerebral autoregulation characterized by acute onset seizures, altered sensorium, headache, visual disturbances, and typical neuroimaging findings of vasogenic oedema in parietooccipital white matter. Although originally described in adults, PRES is increasingly recognized in children, particularly in association with renal disease, hypertension, autoimmune disorders, malignancy, chemotherapy, transplantation, infections, and immune-mediated conditions. Paediatric PRES differs from adult PRES in having a higher frequency of atypical imaging findings involving brainstem, spinal cord, basal ganglia; and broader etiological diversity. Advances in neuroimaging, molecular biology, and endothelial dysfunction research have improved understanding of disease mechanisms. However, diagnostic criteria remain largely clinico-radiological. This review summarizes current evidence regarding diagnosis, typical and atypical radiological findings, aetiologies, tertiary-care experiences, pathophysiological mechanisms, emerging genetic associations, and an algorithmic approach to management.

Keywords: PRES, Diagnosis, Genetics, Management

INTRODUCTION

The first recorded description of posterior reversible encephalopathy syndrome (PRES) is in chapter X of book I, entitled 'Blindness out of the womb' authored by Dr. Nicolaes Pieterszoon Tulp in 1672.[1] In modern literature, it was first described by Hinchey et al in 1996 as reversible posterior leukoencephalopathy syndrome (RPLS) which was as a potentially reversible syndrome characterized by headache, seizures, visual symptoms, altered mental status, and posterior cerebral white matter edema.[2] In 2000, the same condition was rechristened as PRES by Casey et al as it can also involve cerebral cortex.[3]

Since the symptoms of most patients are reversible and the lesions mainly affect the posterior circulation zone of the brain, the most commonly used name in clinical parlance is still PRES. Although termed "posterior" and "reversible," neither posterior localization nor complete reversibility is universal. Paediatric patients frequently demonstrate atypical radiological patterns involving frontal lobes, deep gray nuclei, cerebellum, and brainstem.[4]

This review provides a comprehensive overview and algorithmic approach to childhood PRES, including its pathophysiology, clinic-radiological manifestations, diagnostic evaluation, management strategies, prognostic factors, current areas of uncertainty, and future directions for research and clinical care. Given the expanding spectrum of clinical and radiological presentations associated with this disorder, this review also aims to highlight the similarities and differences in the clinico-radiological features of PRES in children compared with adults.

EPIDEMIOLOGY AND ASSOCIATED OR TRIGGERING CONDITIONS

Although the true incidence of paediatric PRES is unknown, a recent search of database in US estimated that PRES accounted for 0.04% of the hospitalizations.[5]

Reported high-risk groups include the following.

Hypertension

Hypertension is the most common trigger. The mechanisms include autoregulatory failure, hyper-perfusion, blood–brain barrier disruption and endothelial injury. Children may develop PRES at lower blood pressures than adults because cerebral autoregulation is less mature. In children renovascular aetiologies of hypertension are commoner than the essential hypertension.

Renal aetiologies

Renal aetiologies like chronic kidney disease (CKD) where it has shown a dose-response relationship with CKD stage progression, acute glomerulonephritis where both classic posterior as well as diffuse involvement has been documented with associated hypertension, steroid sensitive and steroid resistant nephrotic syndrome in association with hypertension or the use of immunosuppressant agents or high doses of steroid, hemolytic uremic syndrome where PRES is usually associated with precipitating factors such as hypertension, immunosuppressive therapy, CKD, and autoimmune conditions.[6-12]

Rheumatic conditions

Rheumatic conditions like antineutrophil cytoplasmic antibody-related (ANCA) vasculitis, psoriatic arthritis, systemic sclerosis, systemic lupus erythematosus (SLE) with nephritis, and SLE without nephritis have been documented to be linked with PRES with odds ratio of 9.31, 4.61, 6.62, 7.53, and 2.38, respectively.[13] PRES in SLE may result from hypertension, lupus nephritis, endothelial injury, cytokine activation or immunosuppressive drugs. It is critical to differentiate it from neuropsychiatric lupus.

Haematolymphoid malignancies

Haematolymphoid malignancies like acute leukemias, non-Hodgkin lymphoma, Hodgkin lymphoma, and Langerhan's cell histiocytosis.[14] It is reported to occur in recipients of bone marrow transplantation and solid organ transplantation.[15,16] Cytotoxic chemotherapy including platinum-containing drugs (cisplatin, carboplatin), cyclophosphamide, hydroxydaunorubicin/adriamycin, vinca alkaloids (oncovin/vincristine), antimetabolites (methotrexate), prednisone/R-CHOP regimens, and gemcitabine have been reported to be the most commonly used incriminating medications in patients suspected with chemo-associated PRES caused by direct endothelial toxicity.[17] The onset of symptoms occurred after a median of 8 days of chemotherapy. In contrast, immunosuppressive therapy like Gemcitabine-induced PRES is thought to be due to microvascular injury or secondary to thrombotic microangiopathy.[16] Also, certain targeted agents (such as VEGF inhibitors like bevacizumab, sorafenib and sunitinib) rapidly raise systemic blood pressure, exceeding the brain's cerebral autoregulation limit.[16] Tacrolimus and cyclosporine are calcineurin inhibitors indicated usually after solid organ and hematopoietic stem cell transplants. The cumulative incidence of tacrolimus associated-PRES in patients who underwent hematopoietic stem cell transplant was found to be 1.6%.[16] In paediatric oncology populations, PRES incidence ranges from approximately 0.4–0.7%, while much higher rates occur among transplant recipients.

Burns

The literature is limited to case studies where PRES has been documented to occur in children within days to weeks of suffering from burns, sometimes in association with hypertension, with good clinical recovery.[18,19]

Antibiotic-associated PRES

A recent systematic review found case reports of PRES associated with many antibiotics including metronidazole, linezolid, rifampicin, ciprofloxacin, moxifloxacin, levofloxacin, daptomycin, teicoplanin, ceftriaxone, vancomycin.

Mechanisms remain uncertain but may involve neurotoxicity and endothelial injury for indications like gastrointestinal infection, septic arthritis, pneumonia, infectious endocarditis, urinary tract infection, disseminated infection. Onset of PRES from drug intake ranged from 1 hour to 2-3 weeks.[20]

Vaccination-associated PRES

Rare cases have been reported after measles vaccination in children though a systematic review of vaccine adverse event reporting system (VAERS) database of USA showed association of reversible cerebral vasoconstriction and PRES with COVID 19 vaccinations in adults.

The evidence is thus limited to case reports and pharmacovigilance databases. Causality is not established. Potential mechanisms include immune activation and cytokine-mediated endothelial dysfunction.[21,22]

Infection associated PRES

PRES has been documented to occur in association with both viral and bacterial infections like rotavirus, COVID 19, influenza, human herpes 6, severe gram-negative sepsis, bacterial meningitis and leptospirosis.[23-27]

Other drugs

Case reports show association of several drugs with PRES like monoclonal antibodies (rituximab, infliximab, alemtuzumab), immunomodulatory cytokines (interferon-α, interleukin-2), growth factors (granulocyte-stimulating factor, erythropoietin), intravenous immunoglobulins, blood transfusion, intravenous contrast agents, carbamazepine, epinephrine.[28]

Metabolic conditions

It includes hypomagnesemia, hyponatremia, and acute intermittent porphyria.

CLINICAL MANIFESTATIONS

Seizures and altered sensorium are the most common presenting feature in children. Status epilepticus may occur. In a cohort of children with hematolymphoid malignancies, the presenting complaints were seizures (100%), altered sensorium (81%), headache (75%), and visual disturbances (15%). Hypertension at time of diagnosis was noted in 50 (96%) patients.[14]

In children with SLE documented acute-onset headache, vomiting, seizures, abnormalities in visual perception, quadriparesis with spasticity in limbs and hypertonia in all the limbs with extensor plantar response.[29,30]

On the other hand, in a study on antibiotic associated PRES, the clinical features included altered mental status (92%), seizures (58.3%), headache (50%), visual disturbances (16% blurred vision, bilateral visual loss (1%), nausea (16%), others (each reported once: vertigo, meningism, fatigue, myoclonus, dysarthria, dysphagia, and tetanus).[20]

RADIOLOGICAL FINDINGS

Posterior reversible encephalopathy syndrome manifests typically as bilateral vasogenic edema within the occipital and parietal regions (70-90% of cases) appearing hyperintense on T2/FLAIR on MRI brain and hypoattenuating on CT scan (Table 1 and Figure 1).[31]

Despite its name, however, posterior reversible encephalopathy syndrome is known to involve other areas and atypical patterns as shown in Table 2.[4,32] Both cortical and subcortical locations are affected.

Ischemic stroke, intracerebral haemorrhage, and subarachnoid haemorrhage are associated with posterior reversible encephalopathy syndrome in ~11%, ~10% and 7% of cases respectively.[33] The presence of contrast enhancement does not correlate with the clinical outcome. Clinical-radiological dissociation may be encountered whereby the radiological features may be more severe compared to the clinical features. SWI may be used to detect petechial haemorrhage. MR angiography may demonstrate vasculopathy with vessel irregularity consistent with focal vasoconstrictions/vasodilatation or diffuse vasoconstriction. Perfusion imaging can show regional hyper perfusion or hypoperfusion. MRS may document elevated lactate occasionally.[31]

Table 1: Typical MRI brain findings in posterior reversible encephalopathy syndrome.

Findings	Typical pattern
Characteristic findings include bilateral symmetrical T2/FLAIR hyperintensities in parieto-occipital subcortical white matter with diffusion facilitation suggestive of vasogenic oedema	Parieto-occipital > posterior frontal > temporal regions

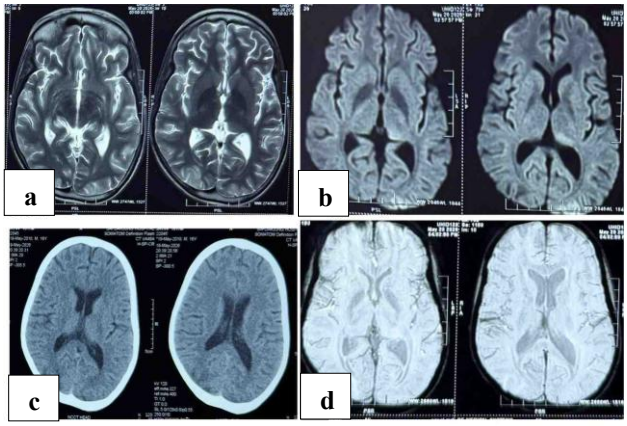

Figure 1: MRI brain showing features of typical PRES; (a and b) parietooccipital white matter T2 and FLAIR hyperintensities, (c) parietooccipital hypoattenuation, and (d) normal SWI.

OTHER INVESTIGATIONS

Electroencephalogram

Electroencephalogram alterations are non-specific and range from global slow activity, normal, bifrontal cortical dysfunction, bilateral occipital sharp waves and slow waves bursts, tempo-parietal theta focus).[20,34]

CSF

Increased CSF proteins, cerebrospinal fluid pleocytosis may be seen with PRES.[20]

Serum findings

These are usually not specific. Low serum magnesium was reported during the first 48 hour after onset reported in a cohort of patients with PRES of varying etiology.[35] Another study found that 84.8% of 79 patients with PRES of different aetiologies had decreased serum albumin levels. However, serum albumin concentration was not

associated with the type of edema (pure vasogenic versus vasogenic with cytotoxic components).[36]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of childhood PRES is broad because its clinical manifestations and neuroimaging findings overlap with several neurological disorders.[28]

Infectious encephalitis and autoimmune or paraneoplastic encephalitis should be considered in children presenting with acute encephalopathy, seizures, and altered mental status, particularly when fever or inflammatory markers are present. Intracranial neoplasms, including lymphoma, gliomatosis cerebri, metastatic disease, and post-transplant lymphoproliferative disease, may produce focal neurological deficits and white matter abnormalities that mimic PRES.

Vascular disorders such as cerebral venous thrombosis, top of the basilar syndrome, ischemic stroke involving watershed or posterior cerebral artery territories, and primary angiitis (vasculitis) of the central nervous system should also be excluded, as they may present with acute neurological deficits and characteristic imaging abnormalities. Seizure-related disorders, including ictal and post-ictal states, particularly following status epilepticus, can demonstrate transient cortical and subcortical MRI changes resembling PRES. White matter disorders such as acute disseminated encephalomyelitis (ADEM), progressive multifocal leukoencephalopathy (PML), toxic leukoencephalopathy, osmotic demyelination syndrome, and severe subcortical leukoaraiosis may also have overlapping radiological features.

In addition**,** inherited and metabolic disorders, including cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) and mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS) syndrome, should be considered, particularly in children with recurrent neurological episodes or suggestive family history.

Differentiation of PRES from these conditions relies on careful correlation of the clinical presentation, blood pressure status, underlying systemic disease, laboratory investigations, cerebrospinal fluid analysis when indicated, and characteristic magnetic resonance imaging findings of reversible vasogenic edema, most commonly affecting the bilateral parieto-occipital regions.[28]

PATHOPHYSIOLOGY

The hallmark of PRES is vasogenic oedema resulting from perturbation of cerebral vascular autoregulatory pathway. As the vascular sympathetic nerve fibres in the posterior circulation of the brain are less distributed and easy to expand, therefore vasogenic cerebral oedema is more likely to appear in parietooccipital regions.[19] There are three main mechanisms postulated to be responsible for this phenomenon namely hypoperfusion, hyperperfusion and endothelial injury as shown in Figure 2.[19,34]

Molecular and genetic mechanisms

Key molecular mediators of the posterior reversible encephalopathy syndrome include vascular endothelial growth factor (VEGF) that increases vascular permeability, TNF-α that promotes endothelial activation, IL-1β involved in inducing blood brain barrier (BBB) dysfunction, IL-6 postulated to be major mediator in inflammatory PRES. Moreover, endothelin-1 mediates vasoconstriction. Also, nitric oxide dysregulation is believed to lead to impaired autoregulation. Complement activation is of special importance in SLE-associated PRES.[37]

Genetic susceptibility remains incompletely understood. Candidate pathways include endothelial genes namely VEGFA and eNOS (NOS3), complement genes **(**CFH, CFI), cytokine polymorphisms involving IL6 and TNFA. Also, BBB regulation mediated by MMP-9, Claudin proteins and Occludin pathways. Future genomic studies may identify predisposition to endothelial injury and PRES development.

Biomarkers

The assessment of clinical biomarkers in PRES is inadequately studied with only two studies on specific biomarkers namely, serum neurofilaments light, a marker of neuroaxonal injury (Fang X) or copeptin, a precursor of AVP in an attempt to identify an optimal therapeutic window for *vaptan* (AVP receptor antagonist) treatments.[38]

Proposed diagnostic criteria

There are no universally accepted diagnostic criteria for paediatric PRES. However, few experts have suggested the use of diagnostic criteria encompassing combination of reversible neurological deficits with typical neuroimaging.[39,40] Some authors perceive that the clinicoradiological reversibility criteria could prevent the inclusion of atypical cases of PRES and artificially restrict the field of the syndrome.[36] Based on the literature review following diagnostic criteria are proposed as tabulated in Table 3.

A review of several paediatric cohorts with PRES in different geographical areas is summarised in Table 4.

ALGORITHMIC APPROACH TO MANAGEMENT

Therapeutic approaches

The management of PRES is multimodal and hinges on prompt recognition and combines supportive strategies with cause specific therapeutic modalities. The primary

goal is to stabilize the patient, prevent further damage, and manage complications. Therapy is tailored based on the severity of symptoms and the underlying cause, with a focus on both acute and long-term management.

Role of corticosteroids

Corticosteroids are a double-edged sword in PRES. They are reported to be involved in both to inducing and treating the condition. Corticosteroids may precipitate PRES through hypertension and thereafter hyperperfusion mediated cerebral injury. They may help in the management of PRES by a multipronged action of enhancing endothelial stability, anti-inflammatory properties and improving blood-brain barrier integrity thereby mitigating vasogenic edema formation.[45]

A recent case report of a patient with PRES who presented with aphasia, hemiparesis, generalized seizure, and prolonged loss of consciousness, highlighted this dilemma. The initial malignant hypertension at presentation was normalized, but the clinical status deteriorated continuously. After adding steroids to the therapy, there was rapid clinical and radiological recovery, suggesting that this could have been a useful therapeutic measure.[46]

An algorithmic management of PRES is summarised in Figure 3.

Table 2: Atypical MRI findings in posterior reversible encephalopathy syndrome.

Findings	May be present in	
	Unusual locations	**Unusual patterns**
Atypical findings are substantially more common in children than adults and occur in 60–80% of paediatric cases	Frontal lobes, cerebellum, basal ganglia, thalamus, cvPRES central variant of PRES involving brainstem; corpus callosum, or spinal cord (PRES-SCI) with sparing of parietooccipital regions	Asymmetric lesions, unilateral involvement, diffusion restriction, haemorrhage, microhaemorrhage, contrast enhancement, cortical involvement

Table 3: Proposed diagnostic criteria for paediatric PRES.

S. no.	Diagnostic criteria
1	Acute to subacute onset neurological features including at least one of seizures, confusion, altered sensorium, headache, visual disturbance ranging from cortical blindness to visual field deficits and hallucinations, focal neurological deficits.
2	Clinical context of associated trigger factors or comorbidities like acute hypertension, renal dysfunction, exposure to causative drugs, autoimmune disease, haematological malignancies.
3	Resolution following treatment
4	Neuroimaging suggestive of (focal) vasogenic edema predominantly involving parieto-occipital regions, posterior white matter, cortical-subcortical junction.
5	Partial/complete reversibility of clinical and/or radiological findings
6	Exclusion of acute ischemic stroke, CNS vasculitis, infectious encephalitis, ADEM, toxic leukoencephalopathy

Table 4: Comparison of few cohorts with paediatric posterior reversible encephalopathy syndrome.

Centre	Northern India[41]	Southern India[42]	Turkish cohort[43]	Italian cohort[44]
No of patients/ duration of study	32/5 years	10 children	5 children/5 years	111 children/8 years
Male: female	1: 2.2	1.5:1	2:3	Female preponderance
Mean age (years)	10.6	6.2	10±4.58	4 in younger cohort
Aetiologies	Hypertension: 81.2%, renal disease: 62.5%, chemotherapy-related: 15.6%	Hypertension: 100%, acute lymphoblastic leukemia (n=5), post-HSCT patients (n=2), Burkitt's lymphoma (n=1) Intracranial non germinomatous germ cell tumor (n=1), metastatic Ewing sarcoma (n=1)	Acute lymphocytic leukemia (n=2), Henoch-Schönlein purpura (n=1), systemic lupus erythematous (n=1), acute poststrepto-coccal glomerulo-nephritis (n=1)	Arterial hypertension (61%), cytotoxic/ immunosuppressant medications (19%), sepsis (7%), preecla-mpsia/eclampsia (6%), multiple organ dysfunction (1%)

Continued.

Centre	Northern India[41]	Southern India[42]	Turkish cohort[43]	Italian cohort[44]
Radiological profile	Atypical MRI findings: 62.5%, frontal lobe involvement: 56%	Bilateral lesions 100%, typical parieto-occipital lesions 70%, atypical involvement in corpus callosum, basal ganglia, thalamus, and hippo-campal regions 30%	Typical parieto-occipital lesions in 100%	80% atypical MRI patterns
Outcome	Vasogenic edema generally resolves completely; restricted diffusion (cytotoxic oedema) or haemorr-hage on initial MRI can occasionally predict poorer neurological recovery	Clinical resolution occurred in all patients. Six got repeat MRIs on follow-up, which showed complete resolution of findings of PRES.	Complete clinical recovery in all patients	Infants presented with higher rates of status epilepticus and ICU admissions compared to older children. Overall, 80–90% favourable outcome rate

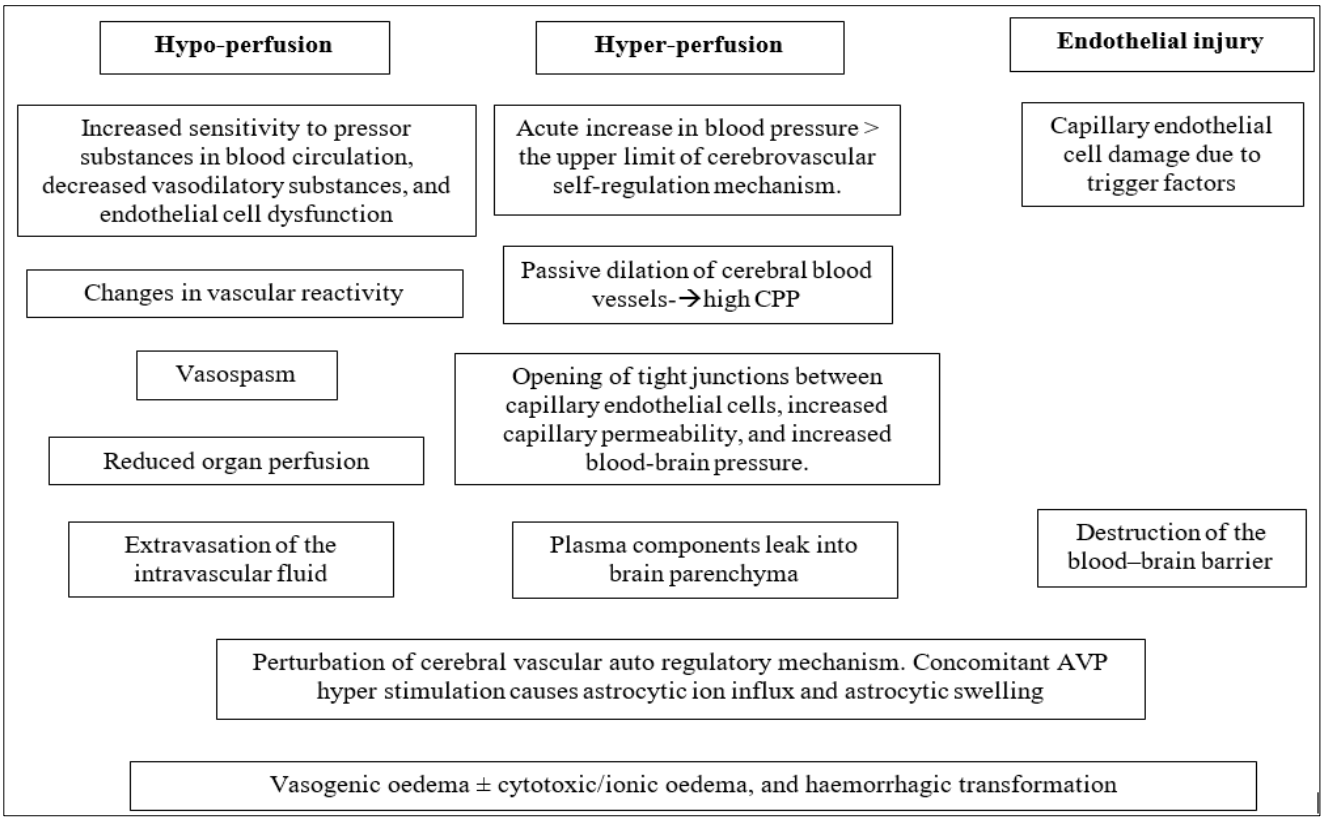

Figure 2: Postulated mechanisms for posterior reversible encephalopathy syndrome.

OUTCOME AND PROGNOSIS

The prognosis of PRES is primarily determined by its underlying aetiology and is generally favourable, with the exception of uncommon cases complicated by intracranial hemorrhage.[47] Most paediatric patients achieve complete clinical recovery, and mortality remains low. However, deaths have been reported, particularly among patients with end-stage renal disease and underlying malignancies, which are recognized as the strongest predictors of mortality.[48,49]

In a cohort of children with hemato-lymphoid malignancies, neurological sequelae were observed in 19% of patients, while one patient died due to PRES.[14] Similarly, most cases of drug-associated PRES, including those related to antibiotic therapy, demonstrated complete or near-complete clinical recovery, although a small proportion showed persistent radiological abnormalities despite clinical improvement.[20]

Gewirtz et al reported that 75–90% of patients experienced complete recovery within one week, whereas 10–20% developed persistent neurological deficits.[50]

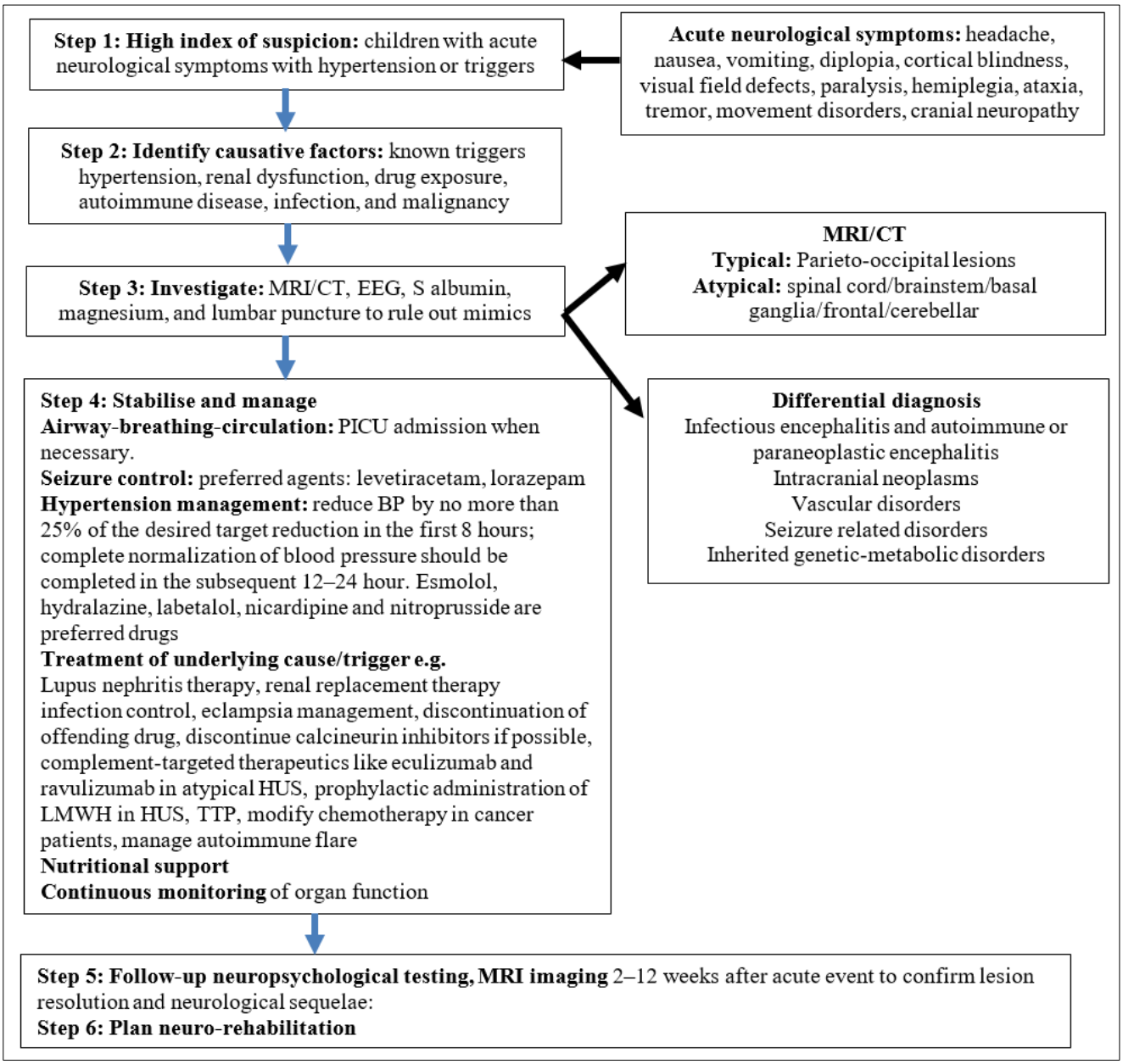

Figure 3: Algorithmic management of paediatric PRES.

Several clinical and radiological factors have been associated with an unfavourable prognosis. Clinical predictors include severe encephalopathy, hyperglycemia, malignancy, delayed correction of the precipitating factor, multiple comorbidities, elevated C-reactive protein (CRP), coagulopathy, and laboratory abnormalities such as low cerebrospinal fluid glucose and elevated serum creatinine, uric acid, and lactate dehydrogenase (LDH) levels.[49]

A six-year retrospective cohort study of non-oncologic paediatric patients with PRES also identified nephrotic syndrome, focal neurological deficits, prolonged hospitalization, and the need for mechanical ventilation as significant predictors of poor outcomes.[10]

Neuroimaging findings also have important prognostic value. Diffusion restriction and the presence of intracerebral or subarachnoid haemorrhage on initial MRI were identified as independent predictors of adverse outcomes in multivariate analyses.[48,51] In addition, atypical MRI features, including corpus callosum involvement and progressive cerebral oedema with diffusion abnormalities, have been associated with poorer neurological outcomes.[48]

NEUROREHABILITATION AND FOLLOW-UP

The extent of long-term neurological sequelae following PRES is variable. While many patients experience complete neurological recovery, others require prolonged rehabilitation and continued clinical surveillance. Patients who develop severe neurological complications during the

acute phase are at increased risk of persistent deficits, including chronic epilepsy, hemiparesis, visual impairment, cognitive dysfunction, and behavioral or attention disorders. In children, these neurocognitive and behavioral impairments may adversely affect academic achievement and social functioning.

Management of persistent neurological deficits requires a multidisciplinary rehabilitation approach involving physical, occupational, speech, and cognitive therapies to improve motor function, daily living skills, communication, and cognitive performance. Pharmacological treatment may also be required for the control of residual symptoms such as seizures and muscle spasticity.

Long-term follow-up should include periodic neuropsychological evaluation, ongoing monitoring and management of underlying comorbidities, and optimal blood pressure control to facilitate early identification and treatment of chronic complications.

CONCLUSION

Paediatric PRES is a heterogeneous clinico-radiological syndrome increasingly recognized across nephrology, rheumatology, oncology, intensive care, infectious disease, and transplant settings. MRI remains the cornerstone of diagnosis, with atypical imaging patterns occurring far more frequently in children than adults. Endothelial dysfunction and BBB disruption constitute the central pathophysiological framework integrating hypertension, autoimmune disease, infection, cancer therapy, and immune-mediated injury. Early recognition, prompt blood pressure control, withdrawal of offending agents, and treatment of the underlying disorder are critical for favourable outcomes. The development of a standardized diagnostic and prognostic algorithm integrating clinical presentation, underlying aetiology, serum and cerebrospinal fluid biomarkers, and neuroimaging features across diverse comorbid conditions would facilitate future research and improve clinical decision-making. Further experimental studies are needed to elucidate the underlying pathophysiological mechanisms of PRES, with the aim of identifying reliable laboratory and imaging biomarkers as well as potential therapeutic targets to enhance functional recovery. In addition, well-designed paediatric-specific studies are essential to better define the risk factors for adverse outcomes and to develop effective neuroprotective strategies for children with PRES.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Ganzevoort W, Boterbloem K, van der Post J, Bleker O. 'Blindness out of the womb', a historical account of the first report of posterior reversible encephalopathy syndrome (PRES): choose your title well or your findings will be neglected. BJOG. 2020;127(1):37-8.
2. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334(8):494-500.
3. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: utility of FLAIR imaging in the detection of cortical and subcortical lesions. Am J Neuroradiol. 2000;21:1199-206.
4. Sharma S, Gupta R, Sehgal R, Aggarwal KC. Atypical presentation of posterior reversible encephalopathy: in a child with bilateral grade IV vesicoureteric reflux. J Trop Pediatr. 2014;60(4):331-3.
5. Thavamani A, Umapathi KK, Puliyel M, Super D, Allareddy V, Ghori A. Epidemiology, Comorbidities, and Outcomes of Posterior Reversible Encephalopathy Syndrome in Children in the United States. Pediatr Neurol. 2020;103:21-6.
6. He M, Gillespie A. Posterior Reversible Encephalopathy Syndrome in Chronic Kidney Disease: Incidence, Outcomes, and Risk Factors in a National Cohort. Kidney Med. 2025;7(7):101022.
7. Orlando C, Milani GP, Simonetti GD, Goeggel Simonetti B, Lava SAG, Wyttenbach R, et al. Posterior reversible leukoencephalopathy syndrome associated with acute postinfectious glomerulonephritis: systematic review. Pediatr Nephrol. 2022;37(4):833-41.
8. Yamada A, Atsumi M, Tashiro A, Hiraiwa T, Ueda N. Recurrent posterior reversible encephalopathy syndrome in nephrotic syndrome: case report and review of the literature. Clin Nephrol. 2012;78(5):406-11.
9. Youssef D, Fawzy F. Posterior reversible encephalopathy syndrome in a child with steroid sensitive nephrotic syndrome. Arab J Nephrol Transplant. 2012;5(3):163-6.
10. Zhang Y, Zhou J, Chen Y. Posterior reversible encephalopathy syndrome in a child with steroid-resistant nephrotic syndrome: a case report and review of literature. Int J Clin Exp Pathol. 2014;7(7):4433-7.
11. Karpman D, Loos S, Tati R, Arvidsson I. Haemolytic Uraemic Syndrome. J Intern Med. 2017;281:123-48.
12. Tonkovic U, Bogicevic M, Manzar A, Andrejic N, Sic A, Atanaskovic M, et al. Neurological Manifestations of Hemolytic Uremic Syndrome: A Comprehensive Review. Brain Sci. 2025;15(7):717.
13. Manadan A, Kambhatla S, Gauto-Mariotti E, Okoli C, Block JA. Rheumatic diseases associated with posterior reversible encephalopathy syndrome. J Clin Rheumatol. 2021;27:4.
14. Ramanathan S, Subramani V, Kembhavi S, Prasad M, Roy Moulik N, Dhamne C, et al. Clinical features, predictors and outcome of posterior reversible

encephalopathy syndrome (PRES) in children with hematolymphoid malignancies. Childs Nerv Syst. 2022;38(9):1689-98.
15. Teive HA, Brandi IV, Camargo CH, Bittencourt MA, Bonfim CM, Friedrich ML, et al. Reversible posterior leucoencephalopathy syndrome associated with bone marrow transplantation. Arq Neuropsiquiatr. 2001;59(3-B):784-9.
16. Kaur G, Ashraf I, Peck MM, Maram R, Mohamed A, Crespo DO, et al. Chemotherapy and Immunosuppressant Therapy-Induced Posterior Reversible Encephalopathy Syndrome. Cureus. 2020;12(10):e11163.
17. How J, Blattner M, Fowler S, Wang-Gillam A, Schindler SE. Chemotherapy-associated Posterior Reversible Encephalopathy Syndrome: A Case Report and Review of the Literature. Neurologist. 2016;21(6):112-7.
18. Wake K, Imataka G, Ohnishi T, Kikuchi J, Hoshiyama E, Ono K, et al. A Case-Study of a Child with Reversible Posterior Leukoencephalopathy Syndrome (RPLS) Associated with Severe Burns throughout the Body. Iran J Public Health. 2020;49(7):1390-1.
19. He X, Shi D, Long C, Yang J, Tian J. Posterior reversible encephalopathy syndrome in a child after burns: A case report and literature review. SAGE Open Med Case Rep. 2023;11:2050313X231157988.
20. Barba L, Carrubba C, Spindler K, Weise CM, Sachs T, Foschi M, et al. Posterior reversible encephalopathy syndrome associated with antibiotic therapy: a case report and systematic review. Neurol Sci. 2024;45(9):4151-9.
21. Aydin K, Elmas S, Guzes EA. Reversible posterior leukoencephalopathy and Adie's pupil after measles vaccination. J Child Neurol. 2006;21(6):525-7.
22. Srichawla BS, Fang T, Kipkorir V, Garcia-Dominguez MA. Reversible cerebral vasoconstriction syndrome and posterior reversible encephalopathy syndrome following vaccination: analysis of the VAERS database and systematic review. Ann Med Surg (Lond). 2023;86(3):1251-60.
23. Sano H, Yamazaki T, Kudo E, Hatakeyama K, Ito T. Posterior reversible encephalopathy syndrome associated with mild COVID-19 infection in a 9-year-old child: A case report and literature review. IDCases. 2023;31:e01699.
24. Palliyaguru T, Ruwanpathirana P, Aravinthan M, Priyankara D, Weeratunga P. Posterior reversible encephalopathy syndrome in severe leptospirosis: A case report. IDCases. 2025;41:e02330.
25. Christofidis C, Anastasiou A, Krasnikova E, Mandros C, Potolidis E. Posterior reversible encephalopathy syndrome as a complication of bacterial meningitis. Hippokratia. 2019;23(3):131-4.
26. Lee DJ, Kim JS. Rotavirus Infection-Associated Posterior Reversible encephalopathy Syndrome. Ann Child Neurol. 2019;27(2):60-2.
27. Ito Y, Toyama K, Honda A, Nakazaki K, Arai S, Kurokawa M. Posterior reversible encephalopathy syndrome concurrent with human herpesvirus-6B encephalitis after allogeneic hematopoietic stem cell transplantation. J Infect Chemother. 2019;26(2):265-8.
28. Chen TH. Childhood Posterior Reversible Encephalopathy Syndrome: Clinicoradiological Characteristics, Managements, and Outcome. Front Pediatr. 2020;8:585.
29. Mani SH, Shivaprasad SM, Umesh L. A rare presentation of lupus nephritis flare up with posterior reversible leucoencephalopathy. J Clin Diagn Res. 2016;10:4.
30. Bhangu JK, Javed K, Manshahia PK, Nahar S, Kanda S, Chatha U, et al. The Association of Hypertension With Posterior Reversible Encephalopathy Syndrome in Systemic Lupus Erythematosus Patients: A Systematic Review. Cureus. 2023;15(12):e50620.
31. Radiopaedia. Posterior reversible encephalopathy syndrome. 2026. Available at: https://radiopaedia.org/articles/posterior-reversible-encephalopathy-syndrome-1. Accessed on 06 July 2026.
32. Bartynski W, Boardman J. Distinct Imaging Patterns and Lesion Distribution in Posterior Reversible Encephalopathy Syndrome. AJNR Am J Neuroradiol. 2007;28(7):1320-7.
33. Kaufmann J, Buecke P, Meinel T, Beyeler M, Scutelnic A, Kaesmacher J, et al. Frequency of ischaemic stroke and intracranial haemorrhage in patients with reversible cerebral vasoconstriction syndrome (RCVS) and posterior reversible encephalopathy syndrome (PRES) - A systematic review. Eur J Neurol. 2024;31(5):e16246.
34. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. Lancet Neurol. 2015;14:914-25.
35. Chardain A, Mesnage V, Alamowitch S, Bourdain F, Crozier S, Lenglet T, et al. Posterior reversible encephalopathy syndrome (PRES) and hypomagnesemia: a frequent association? Rev Neurol. 2016;172(6–7):384-8.
36. Gao B, Yu BX, Li RS, Zhang G, Xie HZ, Liu FL, et al. Cytotoxic edema in posterior reversible encephalopathy syndrome: correlation of MRI features with serum albumin levels. AJNR Am J Neuroradiol. 2015;36(10):1884-9.
37. Largeau B, Bergeron S, Auger F, Salmon Gandonnière C, Jonville-Béra AP, Ehrmann S, et al. Experimental Models of Posterior Reversible Encephalopathy Syndrome: A Review From Pathophysiology to Therapeutic Targets. Stroke. 2024;55(2):484-93.
38. ClinicalTrial.Gov. The Arginine Vasopressin aXis as a Potential Therapeutic Target for Posterior REverSible Encephalopathy SyndromE (XPRESSE). 2022. Available at: https://clinicaltrials.gov/ct2/show/NCT04950270. Accessed on 15 June 2026.

39. Fischer M, Schmutzhard E. Posterior reversible encephalopathy syndrome. J Neurol. 2017;264(8):1608-16.
40. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. Mayo Clin Proc. 2010;85(5):427-32.
41. Gupta V, Bhatia V, Khandelwal N, Singh P, Singhi P. Imaging Findings in Pediatric Posterior Reversible Encephalopathy Syndrome (PRES): 5 Years of Experience From a Tertiary Care Center in India. J Child Neurol. 2016;31(9):1166-73.
42. Gaddam S, Kodandapani R, Mani N, Jayaraman D, Abirami BN. Posterior reversible encephalopathy syndrome in immunocompromised children - A single-center study from South India. J Neurosci Rural Pract. 2024;15(2):365-9.
43. Emeksiz S, Kutlu NO, Çaksen H, Alkan G, Yıkmaz HŞ, Tokgöz H. Posterior reversible encephalopathy syndrome in children: a case series. Turk Pediatri Ars. 2016;51(4):217-20.
44. de Laat P, Te Winkel ML, Devos AS, Catsman-Berrevoets CE, Pieters R, van den Heuvel-Eibrink MM. Posterior reversible encephalopathy syndrome in childhood cancer. Ann Oncol. 2011;22(2):472-8.
45. Srichawla BS, Kaur T, Singh H. Corticosteroids in posterior reversible encephalopathy syndrome: Friend or foe? A systematic review. World J Clin Cases. 2025;13(12):98768.
46. Meyer M, Niemöller U, Stein T, Schmetsdorf S, Arnold A, El-Sheik M, et al. Positive Effect of Steroids in Posterior Reversible Encephalopathy Syndrome. Case Rep Neurol. 2019;11(2):173-7.
47. Gavrilovici C, Miron I, Voroneanu L, Bădărau S, Stârcea M. Posterior reversible encephalopathy syndrome in children with kidney disease. Int Urol Nephrol. 2017;49(10):1793-800.
48. Triplett JD, Kutlubaev MA, Kermode AG, Hardy T. Posterior reversible encephalopathy syndrome (PRES): diagnosis and management. Pract Neurol. BMJ Publishing Group Ltd. 2022.
49. Hinduja A. Posterior reversible encephalopathy syndrome: clinical features and outcome. Front Neurol. 2020;11:71.
50. Gewirtz AN, Gao V, Parauda SC, Robbins MS. Posterior reversible encephalopathy syndrome. Curr Pain Headache Rep. 2021;25:19.
51. Nada MG, Libda YI, Gohary MM, Dessouky R. Pediatric posterior reversible encephalopathy syndrome: Can MR imaging features predict outcomes in non-oncologic patients? Eur J Radiol. 2024;170:111214.

Cite this article as: Sehgal R, Kashyap A, Anand B, Gupta A. Paediatric posterior reversible encephalopathy syndrome: a review of aetiologies, clinical and radiological spectrum, pathophysiology, diagnostic criteria and management. Int J Contemp Pediatr 2026;13:1870-9.