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Reversible cortical blindness in childhood: An unusual case of PRES associated with post streptococcal glomerulonephritis

Juan José Londono-Aranzazu[1], **Andrés Rodríguez**[2], **Nury Isabel Mancilla-Gomez**[2,3]

Abstract

Introduction: Posterior reversible encephalopathy syndrome (PRES) is a rare neurological disorder in children, characterized by distinctive clinical presentations and radiological findings, which may also be associated with renal conditions. We report an unusual case of reversible cortical blindness due to posterior reversible encephalopathy syndrome in a child with post-streptococcal glomerulonephritis (APSGN), highlighting the importance of early recognition and management.

Case presentation: A 9-year-old male with a recent diagnosis of pharyngotonsillitis presented to the hospital with persistent headache and vomiting. These symptoms were followed by transient visual loss and somnolence. Neurological examination revealed dysarthria, incoherent speech, ataxic gait, and cortical blindness. Blood pressure above the 95th percentile for his age led to the diagnosis of hypertensive emergency with cerebral target-organ damage. Antihypertensive therapy was initiated promptly. Laboratory tests demonstrated a positive Group A Streptococcus (GAS) antigen, along with proteinuria and hematuria, while abdominal computed tomography (CT) revealed kidney enlargement. These findings confirmed the diagnosis of post-streptococcal glomerulonephritis. Brain magnetic resonance imaging (MRI) demonstrated findings compatible with hypertensive leukoencephalopathy. Continuous blood pressure monitoring was maintained until optimal ranges were achieved, resulting in complete vision recovery. No neurological deficits were observed at the 20-week follow-up.

Discussion: Patients with posterior reversible encephalopathy syndrome present distinctive symptoms such as epileptic seizures, headaches, and visual disturbances, often mimicking other conditions, underscoring the importance of accurate diagnosis for timely intervention. Imaging findings are essential for confirming the diagnosis and guiding management.

Conclusions: This case highlights the importance of recognizing posterior reversible encephalopathy syndrome in pediatric patients. Appropriate and timely control of blood pressure contributed significantly to complete neurological recovery.

Keywords: Brain edema, Posterior reversible encephalopathy syndrome, Glomerulonephritis, Hypertensive encephalopathy, Cortical blindness, Pediatrics.

Ceguera cortical reversible en la infancia: un caso inusual de PRES asociado con glomerulonefritis postestreptocócica

Resumen

Introducción: el síndrome de encefalopatía posterior reversible (PRES) es un trastorno neurológico poco frecuente en la población pediátrica, caracterizado por manifestaciones clínicas distintivas y hallazgos radiológicos particulares, que puede asociarse con enfermedades renales. Presentamos un caso inusual de ceguera cortical reversible secundaria al síndrome de encefalopatía posterior reversible en un niño con glomerulonefritis postestreptocócica aguda (GNPE), destacando la importancia de su reconocimiento y manejo oportunos.

Presentación del caso: paciente masculino de 9 años con diagnóstico reciente de faringoamigdalitis, quien ingresó al hospital por cefalea persistente y vómito. Posteriormente, presentó pérdida visual transitoria y somnolencia. El examen neurológico reveló disartria, lenguaje incoherente, marcha atáxica y ceguera cortical. La presión arterial por encima del percentil 95 para su edad condujo al diagnóstico de emergencia hipertensiva con daño cerebral como órgano blanco. Se inició de inmediato tratamiento antihipertensivo. Los exámenes de laboratorio mostraron antígeno positivo para Streptococcus del grupo A, así como proteinuria y hematuria. La tomografía computarizada (TC) abdominal evidenció agrandamiento renal, lo que confirmó el diagnóstico de glomerulonefritis postestreptocócica aguda. La resonancia magnética cerebral (RM) mostró hallazgos compatibles con leucoencefalopatía hipertensiva. Se realizó monitoreo continuo de la presión arterial hasta alcanzar rangos óptimos, logrando recuperación visual completa. No se evidenciaron déficits neurológicos durante la evaluación clínica en el seguimiento a 20 semanas.

Discusión: los pacientes con síndrome de encefalopatía posterior reversible presentan síntomas característicos como crisis epilépticas, cefalea y alteraciones visuales, que a menudo simulan otras condiciones, lo que resalta la importancia de un diagnóstico preciso para una intervención oportuna. Los hallazgos por imágenes son esenciales para confirmar el diagnóstico y orientar el manejo.

Conclusiones: este caso subraya la importancia de reconocer el síndrome de encefalopatía posterior reversible en pacientes pediátricos. El control adecuado y oportuno de la presión arterial contribuyó de manera significativa a la recuperación neurológica completa.

Palabras clave: edema cerebral, síndrome de encefalopatía posterior reversible, glomerulonefritis, encefalopatía hipertensiva, ceguera cortical, pediatría.

[1] Universidad Libre, Cali, Colombia

[2] Hospital Universitario Santa Clara E.S.E, Bogotá, Colombia

[3] Universidad Nacional de Colombia, Bogotá, Colombia

Correspondence/Correspondencia:
Juan José Londono-Aranzazu, Universidad Libre, seccional Cali, Cra. 37a #3-29, Cali, Colombia.
E-mail: juanj-londonoa@unilibre.edu.co

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare neurological disorder in the pediatric population that features distinctive clinical manifestations and specific radiological findings, it was initially described in 1996 by Hinchey et al. (1). Its clinical presentation is variable but typically includes acute-onset symptoms such as visual disturbances, headaches, vomiting, epileptic seizures, and altered consciousness (2). This syndrome is associated with multiple conditions that cause moderate to critically high blood pressure, including systemic disorders, obstetric conditions (3), autoimmune diseases (4), renal disorders (5), and oncological or hematological conditions (6-8), among others (9,10).

From a radiological perspective, PRES is characterized by a typical pattern of bilateral and symmetrical vasogenic edema, predominantly affecting the territories of the posterior cerebral circulation, particularly the subcortical white matter, and most frequently involving the parieto-occipital regions (10). Approximately 70% of patients with PRES require intensive care unit management. While most recover within two weeks, neurological sequelae and permanent PRES have been reported (11).

The aim of this case is to describe the clinical and neuroradiological features of a patient and its association with post-infectious glomerulonephritis (APSGN) caused by group A Streptococcus.

Case presentation

We present the case of a 9-year-old male patient from a rural area in Antioquia, Colombia, with a challenging biopsychosocial background, including a history of forced displacement due to armed conflict one month prior. The patient had a recent diagnosis of pharyngotonsillitis, established 12 days before the onset of symptoms. He presented with a global headache, acute vision loss, and persistent vomiting lasting approximately 10 hours.

On physical examination, the patient was in fair general condition with marked pallor. Neurological assessment revealed somnolence, dysarthria, incoherent speech, and ataxic gait. Muscle strength and sensation were preserved, with no signs of meningeal irritation. Fundoscopic examination showed no papilledema, and the pupils were isochoric and reactive. However, he exhibited progressive visual acuity loss, evolving into cortical blindness.

Blood pressure at admission (223/140 mmHg) was significantly above the 95th percentile for his age, confirming a hypertensive emergency with central nervous system target-organ damage. Antihypertensive treatment was initiated promptly, beginning with an oral antihypertensive agent, and was carefully managed to ensure a gradual reduction in blood pressure, thereby minimizing potential risks associated with rapid decreases. Table 1 displays the initial paraclinical findings, which include leukocytosis predominantly due to neutrophilia. Additionally, mild metabolic acidosis was recorded.

In the context of central nervous system involvement, a non-contrast cranial computed tomography (CT) scan was performed, which revealed no significant abnormalities. Cerebrospinal fluid (CSF) analysis showed an opening pressure of 22 mmHg. The FilmArray Meningitis/Encephalitis Panel PCR tested negative for pathogens, and blood cultures were also negative. Considering the recent infectious history, a rapid test for Streptococcus pyogenes and anti-streptolysin O (ASO) titers were performed, both yielding positive results. Complement component C3 levels were mildly decreased. These findings supported the diagnosis of concomitant APSGN.

The patient was transferred to the intensive care unit (ICU) two days after symptom onset due to neurological deterioration characterized by somnolence, irritability, and cortical blindness, along with focal seizures involving the right upper limb. These seizures progressed to status epilepticus, necessitating continuous intravenous midazolam infusion, phenytoin administration, and endotracheal intubation. On day 4 after symptom onset, prolonged video-electroencephalographic monitoring (12 hours) revealed diffuse encephalopathic slowing with loss of age-appropriate background rhythms and no electrographic ictal discharges. Intracranial angiography and orbital CT showed no significant abnormalities, and visual evoked potentials were within normal limits.

The management of high blood pressure was complex, requiring multiple adjustments to achieve stabilization. A stepwise approach to antihypertensive therapy was adopted, ultimately involving the use of four antihypertensive agents, including intravenous medications. As shown in Table 2, on the fifth day of the clinical course, laboratory tests indicated a decline in renal function, accompanied by mild hematuria

Table 1. Paraclinical findings at admission

Paraclinical assessment	Result	References values
BG	181	70-110
CBC	WCB: 20,48 x 10^3 RBC: 4,3 x 10^3 Hb: 14 PLT: 340 x 10^3	WBC: 4,2 - 9, x 10^3 RBC: 4,6 x 10^3 Hb: 13,7 - 17,5 PLT: 163 - 337 x 10^3
CRP	0,32	0 – 1
LDH	288	140 - 240
BUN	15,2	9 - 20
SC	0,39	0,6 - 1,3
SE	Na: 135 K: 3, 76 CL: 103 P: 6,1 Ca: 9,6 MG: 1,9	Na: 137 - 145 K: 3,5 – 5,1 CL: 98 - 107 P: 2,5 - 4,5 Ca: 8,4 -10,2 MG: 1,6 - 2,3
LFT	AST: 23 ALT: 13 PT: 13,6 aPTT: 33,3 INR: 1,2	AST: 14 – 36 ALT: 0 – 35 PT: 11,4 PTT: 30,2
hs-TnI	0,012	< 0,034
TFT	FT4: 1,2 TSH: 2,76	FT4: 0,8-1,8 TSH: 0,4-4,5
Others	Alb: 3 C3: 18 C4: 26 ANA: Reactive (1:80) ANCA: No reactive VDRL: No reactive RSPT: Positive ASO: 200 VBG: Mild metabolic acidosis LCR: Glucose 62 (mg/dL), Proteins < 10 mg/dL, Colorless, Clear odor, Fresh red blood cell counts 0 cells/µL, Culture: negative, KOH: Negative, Oligoclonal bands: negative.	Alb: 3 – 5 C3: 80 – 150 C4: 17 – 51

Note. Abbreviations: BG: Blood glucose (mg/dL); CBC: Complete blood count; WBC: White blood cells (cells/mm3); RBC: Red blood cells (cells/mm3); Hb: Hemoglobin (g/dL); PLT: Platelets (cells/mm3); CRP: C reactive protein (mg/dL); LDH: Lactate dehydrogenase (U/L); BUN: Blood urea nitrogen (mg/dL); SC: Serum creatinine (mg/dL); SE: Serum electrolytes; Na: Sodium (mmoL/L); K: Potassium (mmoL/L); CL: Chloride (mmoL/L); P: Phosphorus (mg/dL); Ca: Calcium (mmol/dL); Mg: Magnesium (mg/gL); LFT: Liver function test; AST: Aspartate aminotransferase (UI/L); ALT: Alanine aminotransferase (UI/L); PT: Prothrombin time (seconds); aPTT: Activated partial thromboplastin time (seconds); INR: International normalized ratio; hs-TIn: Troponin I high sensibility (ng/ml); TFT: Thyroid function tests; FT4: Free thyroxine (ng/dL); TSH: Thyroid stimulating hormone (µIU/mL); Alb: albumin (g/dL); C3: Complement 3 (mg/dL); C4: Complement 4 (mg/dL); ANA: Antinuclear antibody; ANCA: antineutrophil cytoplasmic antibodies; VDRL: Venereal disease research laboratory test; RSPT: Rapid Streptococcus pyogenes test; ASO: anti-streptolysin O (IU/mL); VBG: Venous blood gas; LCR: Cerebrospinal fluid analysis; KOH: Potassium hydroxide.

Source: Own elaboration.

and persistent proteinuria. Abdominal CT imaging revealed enlarged kidneys.

Brain magnetic resonance imaging (MRI) was performed on day 8 after symptom onset, revealing predominantly hyperintense focal lesions in the parieto-occipital regions, with flattening of the gyri and effacement of the sulci on both T2-weighted and FLAIR sequences. These findings were consistent with a diagnosis of hypertensive leukoencephalopathy (Figure 1). Following confirmation with contrast-enhanced MRI, antihypertensive management was continued with strict blood pressure monitoring to maintain values within optimal ranges.

The patient showed an initial improvement in central vision, achieving complete recovery of visual acuity 11 days after symptom onset. By day 18, he was free of proteinuria and hematuria, and his blood pressure remained below the 90th percentile with adequate control using two oral antihypertensive medications. After clinical stabilization, he was discharged on day 26. Given the patient's social context, a multidisciplinary follow-up plan was implemented, including support from social work and psychology. Follow-up was coordinated through pediatric neurology, nephrology, and ophthalmology.

A follow-up brain MRI was performed 15 weeks post-discharge, revealing isolated punctate hyperintensities in the subcortical regions of both hemispheres on FLAIR and T2 sequences, consistent with sequelae, with no parenchymal abnormalities in the periventricular areas. No neurological deficits were observed at the 20-week follow-up.

Discussion

This case illustrates a rare but well-documented presentation of PRES in a previously healthy pediatric patient who developed APSGN with acute and severe elevation in blood pressure following an episode of pharyngotonsillitis. It is crucial to consider this pathology when a hypertensive emergency presents accompanied by target organ damage in the brain, as prompt diagnosis and appropriate therapeutic intervention are key to preventing potential sequelae.

PRES is characterized as a clinico-radiological syndrome presenting with neurological features that occur in decreasing order of frequency, including epileptic seizures, headaches, nausea, altered mental status, and visual disturbances. The hallmark of PRES is the reversibility of symptoms with appro-

Table 2. Paraclinical follow-up data

Paraclinical assessment	2 Days post symptoms onset	5 Days post symptoms onset	9 Days post symptoms onset	Discharge	Reference values
CBC	WBC 15 x 103 RBC: 4,85 x 103 Hb: 12.4 g/dL PLT: 281 x 103	WBC 11,17 x 103 RBC: 4,74 x 103 Hb: 13 g/dL PLT: 303 x 103	WBC 10,18 x 103 RBC: 4,2 x 103 Hb: 13 g/dL PLT: 439 x 103	WBC 9,5 x 103 BC: 5 x 103 Hb: 11,8 g/dL PLT: 365 x 103	WBC: 4,2-9, x 103 RBC: 4,6 x 103 Hb: 13,7-17,5 g/dL PLT: 163-337 x 103
BUN	18	13	11,2	12	7-20
Serum creatinine	0,4	0,42	0,41	0,5	0,6-1,3
Urine creatinine	-	60	13,8	33	-
Proteinuria	-	79	40	13	<12
P/C	-	1,3	2,8	0,39	<0,2
Urinalysis	-	Yellow urine with clear appearance, pH of 7,5. P: 30, Hb: 0.15 (Eu 95% and Dys 5%). Negative for leukocyte esterase, nitrites and glucose. But ketones 30 mg/dL.	Yellow urine with clear appearance, pH of 7, 5. P: 15. Hb: 0.75 (Eu 98% and Dys 2%). Negative for leukocyte esterase, nitrites, glucose, ketones, and bilirubin.	Yellow urine with clear appearance. pH of 7,5. P: 30. Hb: 0,06. Negative for leukocyte esterase, nitrites, glucose, ketones, and bilirubin.	-

Note. Abbreviations: CBC: Complete blood count; WBC: White blood cells (cells/mm3); RBC: Red blood cells (cells/mm3); Hb: Hemoglobin (mg/dL); PLT: Platelets (cells/mm3); P/C: Protein/creatinine ratio; BUN: Blood urea nitrogen (mg/dL); SC: Serum creatinine (mg/dL); UC: Urine creatinine (mg/dL); P: Protein (mg/dL); Eu: eumorphic; Dys: Dysmorphic.

Source: Own elaboration.

priate management, which typically occurs within the first two weeks (11). Due to its potential for reversibility, early recognition is crucial, particularly in pediatric patients presenting with risk factors and nonspecific neurological symptoms.

In patients with APSGN, as observed in the present case, severe complications have been documented, including hypertensive emergencies (21.5%), encephalopathy (4.6%), congestive heart failure (12.3%), retinopathy (1.5%), and PRES in 2.9% of cases (12). The incidence of PRES in pediatric patients with renal abnormalities is estimated to range between 4% and 9%; however, some studies report rates as high as 29.4%, with a higher prevalence observed in male patients, as seen in this case (11,13).

Furthermore, it has been demonstrated that PRES associated with APSGN predominantly manifests in pediatric patients with blood pressure readings exceeding the 95th percentile for age (14). Early recognition of PRES within this context is crucial, as diagnosis is often challenging due to the nonspecific nature of symptoms, which may include both neurological and renal manifestations, often mimicking a variety of other conditions (15,16). Prompt initiation of antihypertensive therapy is essential to reduce blood pressure safely. The target blood pressure should be tailored according to the initial levels, and, in accordance with established guidelines for severe symptomatic acute hypertension, it is generally recommended to reduce the mean blood pressure by no more than 20–25% within the first 6 to 9 hours to mitigate the risk of adverse outcomes (17).

In pediatric patients with PRES, clinical presentations are notably heterogeneous, as documented in Table 3. The most prevalent features include epileptic seizures, reported in approximately 88–95% of cases. Treatment options include phenytoin, phenobarbital, and, in cases of refractory status epilepticus, continuous midazolam infusion (14,17,18). These seizures may manifest as generalized tonic-clonic episodes, accounting for approximately 72.1% of cases, and as relapsing seizures in 64.7%. In some instances, seizures may progress to status epilepti-

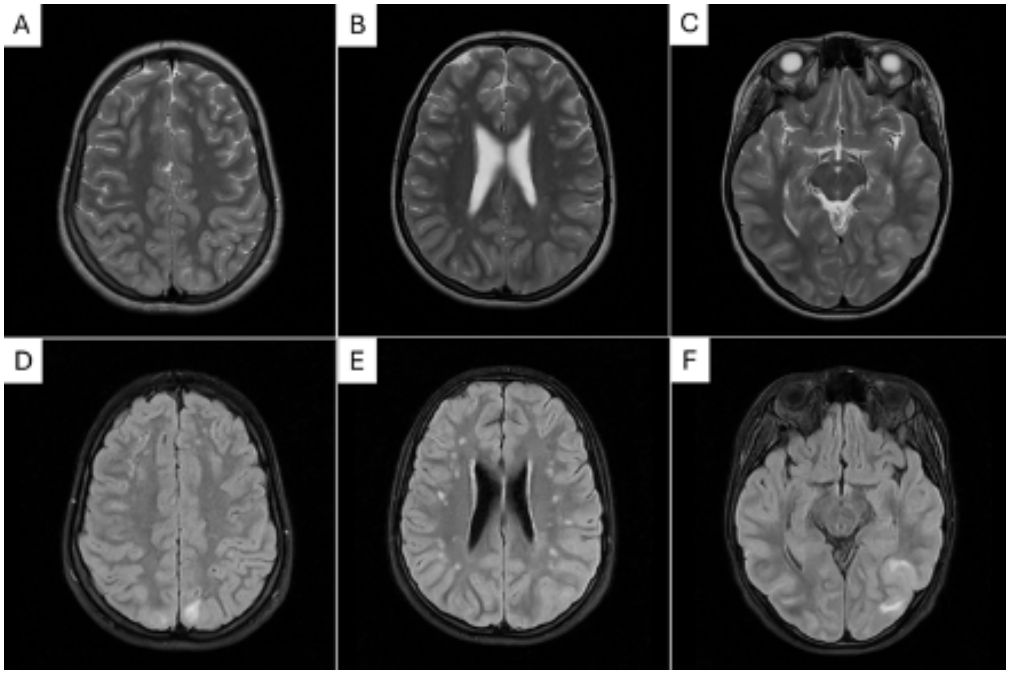

Figure 1. Brain MRI performed on day 8

Note. (A - C) Axial cuts in the T2 sequence show hyperintense areas in the bilateral cortico-subcortical substance, affecting the frontal, parietal, and occipital lobes, along with punctate foci in the corona radiata and semioval centers. (D - F) Axial cuts in the T2-FLAIR sequence reveal asymmetric enhancement predominantly in the occipital gyri, associated with flattening of the gyri and effacement of the cortical sulci, findings suggestive of vasogenic edema secondary to PRES.

Source: Own elaboration.

cus, with reported rates ranging from 13.2% to 52% (11,17,18). Other frequently observed manifestations encompass altered consciousness, reported in 38% to 63.2% of cases; headaches, present in 6.31% to 85%; and visual disturbances, occurring in 12.6% to 56% of patients (11,17,18). Additionally, renal abnormalities have been documented in approximately 25% of these cases, with a concomitant initial presentation involving renal and other neurological or systemic alterations occurring in 4% of patients (18).

Imaging correlation plays a crucial role in the diagnosis of PRES. In pediatric populations, the sensitivity of initial computed tomography (CT) remains limited, with detection rates as low as 46%. Conversely, brain MRI offers a sensitivity of up to 98% and is considered the gold standard for diagnosis (17). MRI enables early identification of hallmark features, such as cortico-subcortical hyperintensities on T2-weighted and FLAIR sequences, which can be detected from the first day of presentation. These radiological findings typically reflect vasogenic edema and indicate the potential reversibility of the condition within the first week, predominantly affecting posterior regions, especially the parieto-occipital lobes (19). This imaging modality facilitates timely diagnosis and allows for the prompt initiation of targeted treatment, which is essential for optimal patient outcomes.

A wide spectrum of atypical presentations of PRES has also been documented, including involvement of less common regions such as the frontal lobes, basal ganglia, corpus callosum, cerebellum, and brainstem (20). Additionally, hemorrhagic changes and findings of restricted diffusion on diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) sequences may be observed. These atypical features are particularly relevant in pediatric patients, where they have been reported in 61–82% of cases. Notably, involvement of the superior frontal sulcus has emerged as a distinctive characteristic in this population (19,21).

PRES generally has a favorable prognosis when management of elevated blood pressure is initiated

Table 3. Clinical, neurological, and radiological characteristics reported in pediatric patients with PRES in different international studies

References	Population	Demographic characteristics	Neurological manifestations (%)	MRI Changes (%)	Outcome
Koller et al. (2024) (22)	2 cases of Press in patient with renal involvement.	Ages 15 and 10, Male (100%).	Headache (100%), nausea (50%), vomiting (50%), seizures (100%), altered mental status (50%), visual disturbances (50%).	Hyperintensity on T2, FLAIR symmetrically distributed in the bilateral cortical-subcortical hemispheres; frontal lobe, parietal, occipital, post-central gyru and cerebellar (50%).	Alive, no sequelae, no recurrences.
Tomarelli et al. (2024) (7)	A case of PRES with acute glomerulonephritis and hemolytic anemia.	4 years old, male.	Headache, nausea, vomiting, status epilepticus.	Brain MRI Day 5: Bilateral symmetric occipital subcortical hyperintensity. No diffusion restriction.	Alive, no sequelae, no recurrences.
Ekinci et al. (2023) (11)	68 Pediatric Patients with PRES.	Mean age 9.3 years; 66.2% male.	Altered consciousness (80%), seizures (60%).	Parieto-occipital (72.1%), typical lesions (29.4%), atypical lesions (70.6%), supratentorial (76.5%).	Complete recovery (80.9%), death (13.2%), neurological deficit (5.9%), recurrence (4.4%).
Castellano-Martinez A et al. (2022) (16)	2 pediatric patients with PRES as onset of acute post-infectious glomerulonephritis.	Ages 9 and 11 years. Male (100%).	Generalized tonic-clonic seizures (100%), somnolence (50%), headache (100%), blurry vision (50%).	Symmetrical areas of cortico- subcortical hyperintensity in the temporo-occipital and frontoparietal regions of both cerebral hemispheres.	Full neurological recovery (100%), normal renal function (100%), persistent microscopic hematuria in 1 patient (50%).
Orlando et al. (2022) (18)	52 Patients with Post-Infectious Glomerulonephritis.	Age: ≤18 years (96%); male (61.5%).	Neurological manifestations (71%), In severe hypertension: headache-nausea (85%), altered consciousness (71%), seizures (90%), visual disturbances (54%).	Severe changes (2%), moderate (4%), mild (94%). In severe hypertension: posterior changes (58%), diffuse (41%).	Complete clinical resolution 3-16 weeks (90%). Radiological resolution (100%).
Darwish (2020) (14)	28 pediatric patients with PRES.	Mean age 6 ± 2.2, 58.3% male.	Seizures (100%), status epilepticus (20.8%), headache (62.5%), visual disturbances (37.5%).	Hyperintensity on T2, FLAIR, and ADC (100%), hyperintense DWI (75%), parieto-occipital (62.5%), frontal cortico-subcortical (33.3%), periventricular (4.2%).	Normal neurological exam at 2, 6, and 12 months (100%), complete brain MRI resolution at 2 months (87.5%), 6 months (95.8%).

Source: Own elaboration.

gradually and appropriately, with clinical and radiological resolution occurring within 3 to 16 weeks in approximately 70-90% of cases (13,18,21). Nonetheless, despite limited data in the literature regarding long-term outcomes, residual brain damage has been documented through neurological assessments and follow-up brain MRI. Although most patients typically exhibit reversibility of MRI abnormalities at the time of discharge, outcomes can vary, and persistent abnormalities may be evident in a minority of cases, estimated to range from 12.5% to 38.5% (21). These findings highlight the critical

importance of ongoing neurological and radiological surveillance.

In this case, the patient achieved complete resolution of cortical blindness by day 11 and recovery of renal function by day 18. A follow-up brain MRI at 15 weeks revealed minor sequelae, while no neurological deficits were observed during clinical evaluation at 20 weeks.

Conclusions

This case underscores the fundamental importance of early recognition and prompt management of PRES in pediatric patients, especially in the context of hypertensive emergencies associated with APSGN. Strict surveillance and adequate blood pressure control were essential for achieving complete neurological recovery, including the resolution of cortical blindness. Additionally, this case highlights the value of a multidisciplinary approach that integrates clinical evaluation, laboratory findings, and brain MRI, considered the gold standard in diagnosing this condition, ensuring timely and guided treatment. The favorable outcome reinforces that PRES is a reversible condition when identified and treated promptly, emphasizing the importance of vigilant monitoring and comprehensive care in similar cases.

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Ethical implications. The authors confirm that this study complies with ethical standards and ethical approval was granted under Acta No. 447, dated November 25, 2024, by the Ethics Committee of the Hospital Universitario Santa Clara. Informed consent was obtained from the patient's legal guardian for the publication of this case report and accompanying images. Institutional ethical approval for experimental interventions was not required, as this manuscript describes a single case report based on clinical observations.

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References

1. 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. https://doi.org/10.1056/NEJM199602223340803
2. Parasher A, Jhamb R. Posterior reversible encephalopathy syndrome (PRES): Presentation, diagnosis and treatment. Postgrad Med J. 2020;96(1140):623–628. https://doi.org/10.1136/postgradmedj-2020-137706
3. Gómez Jordan S, Polo Trujillo M, Cuellar Lobo M, Cárdenas Castro V, Meza Cely N. Neuropsychiatric manifestations of posterior reversible encephalopathy syndrome (PRES) after a severe obstetric complication. Acta Neurol Colomb. 2024;40(3):e1806. https://doi.org/10.22379/anc.v40i3.1806
4. Baisya R, Kumar Devarasetti P, Narayanan R, Rajasekhar L. Posterior reversible encephalopathy syndrome in juvenile lupus: A case series and literature review. Lupus. 2022;31(5):606–612. https://doi.org/10.1177/09612033221088207

5. 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):1793–1800. https://doi.org/10.1007/s11255-017-1684-x
6. Sommers KR, Skiles J, Leland B, Rowan CM. Posterior reversible encephalopathy syndrome: Incidence and clinical characteristics in children with cancer. J Pediatr Hematol Oncol. 2022;44(2):54–59. https://doi.org/10.1097/MPH.0000000000002153
7. Tomarelli RG, Arriagada SD, Donoso FA. Síndrome de encefalopatía posterior reversible (PRES) y anemia hemolítica: Dos complicaciones graves e inhabituales de glomerulonefritis aguda post estreptocócica. Andes Pediatr. 2024;95(5):600–607. https://doi.org/10.32641/andespediatr.v95i5.4705
8. Kiermasz A, Zapała M, Zwiernik B, Stręk-Cholewińska A, Machnikowska-Sokołowska M, Mizia-Malarz A. Posterior reversible encephalopathy syndrome in children with malignancies: A single-center retrospective study. Front Neurol. 2023;14:1261075. https://doi.org/10.3389/fneur.2023.1261075
9. Geocadin RG. Posterior reversible encephalopathy syndrome. N Engl J Med. 2023;388(23):2171–2178. https://doi.org/10.1056/NEJMra2114482
10. Ando Y, Ono Y, Sano A, Fujita N, Ono S. Posterior reversible encephalopathy syndrome: A review of the literature. Intern Med. 2022;61(2):135–141. https://doi.org/10.2169/internalmedicine.7520-21
11. Ekinci F, Yildizdas D, Horoz OO, Gul Mert G, Kaya O, Bayram I, et al. Pediatric posterior reversible encephalopathy syndrome: Age-related clinico-radiological profile and neurologic outcomes. Pediatr Int. 2023;65(1):e15562. https://doi.org/10.1111/ped.15562
12. Bhat IA, Ashraf M, Sheikh WS, Ahmed P, Yousuf V. Clinical spectrum of acute post-streptococcal glomerulonephritis in children: A kashmir experience. Cureus. 2024;16(7):e63902. https://doi.org/10.7759/cureus.63902
13. Cordelli DM, Marra C, Ciampoli L, Barbon D, Toni F, Zama D, et al. Posterior reversible encephalopathy syndrome in infants and young children. Eur J Paediatr Neurol. 2021;30:128–133. https://doi.org/10.1016/j.ejpn.2020.10.009
14. Darwish AH. Posterior reversible encephalopathy syndrome in children: A prospective follow-up study. J Child Neurol. 2020;35(1):55-62. https://doi.org/10.1177/0883073819876470
15. Faille LD, Fieuws S, Van Paesschen W. Clinical predictors and differential diagnosis of posterior reversible encephalopathy syndrome. Acta Neurol Belg. 2017;117(2):469–475. https://doi.org/10.1007/s13760-017-0750-6
16. Castellano-Martinez A, Roldán-Cano V, Morales-Arandojo P, Rodriguez-González M. Posterior reversible encephalopathy syndrome as a debut of postinfectious glomerulonephritis. An Pediatr (Barc). 2022;96(5):452–454. https://doi.org/10.1016/j.anpede.2021.06.012
17. Chen TH. Childhood posterior reversible encephalopathy syndrome: Clinicoradiological characteristics, managements and outcomes. Front Pediatr. 2020;8:585. https://doi.org/10.3389/fped.2020.00585
18. 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):833–841. https://doi.org/10.1007/s00467-021-05244-z
19. Bazhu S, Shi Y, Ren S, Shu L, Li Q. Posterior reversible encephalopathy syndrome secondary to acute post-streptococcal glomerulonephritis in a child: A case report from the Tibetan plateau. BMC Neurol. 2022;22:225. https://doi.org/10.1186/s12883-022-02750-x
20. Albach SM, Stein D. Posterior reversible encephalopathy syndrome in children and adolescents. Curr Hypertens Rep. 2024;26(8):349-354. https://doi.org/10.1007/s11906-024-01303-6
21. Brady E, Parikh NS, Navi BB, Gupta A, Schweitzer AD. The imaging spectrum of posterior reversible encephalopathy syndrome: A pictorial review. Clin Imaging. 2018;47:80–89. https://doi.org/10.1016/j.clinimag.2017.08.008
22. Koller AR, Man A, Muntean C. Posterior reversible encephalopathy syndrome, not so uncommon in pediatric patients with renal involvement: A case series. J Crit Care Med (Targu Mures). 2024;10(1):96–102. https://doi.org/10.2478/jccm-2024-0004