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Posterior Reversible Encephalopathy Syndrome Associated With Post-Streptococcal Glomerulonephritis in a Young Adult: A Case Report and Literature Review

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Patient: Male, 20-year-old
Final Diagnosis: Posterior reversible encephalopathy syndrome
Symptoms: Convulsions • haematuria • headache
Clinical Procedure: —
Specialty: Neurology

Objective: Rare coexistence of disease or pathology

Background: Posterior reversible encephalopathy syndrome (PRES) is a neuro-radiological disorder characterized by seizures, headache, visual problems, and vasogenic edema visible on imaging scans. While it is most frequently linked to hypertensive crises, eclampsia, autoimmune conditions, and immunosuppressive drugs, its association with post-streptococcal glomerulonephritis (PSGN) is rare, particularly in adults. Typically, PRES associated with PSGN occurs in children and can develop even when blood pressure is mildly elevated.

Case Report: We describe the case of a 20-year-old Sudanese man who presented with a sudden, severe headache, 2 episodes of generalized tonic–clonic seizures, and complete painless bilateral vision loss. Examination showed mild hypertension, periorbital swelling, and pedal edema. Laboratory tests indicated acute kidney injury, nephritic-range proteinuria, microscopic hematuria, low complement C3, and markedly elevated anti-streptolysin (ASO) titers, supporting a diagnosis of PSGN. Brain imaging revealed bilateral parieto-occipital vasogenic edema consistent with PRES. The patient was treated with oral antihypertensives, anticonvulsants, and a short course of steroids. Clinical improvement occurred within 24 h, with progressive vision restoration by day 3 and full recovery by day 5. Renal function normalized within 2 weeks, and no neurological deficits remained.

Conclusions: This case highlights an uncommon presentation of PRES secondary to PSGN in a young adult with mild-to-moderate blood pressure elevation. It emphasizes the importance of early recognition of PRES in patients with recent streptococcal infection and kidney dysfunction, even if they do not have severe hypertension. Rapid diagnosis and prompt treatment are crucial to achieve full neurological recovery and prevent potential complications.

Keywords: Adult • Case Reports • Glomerulonephritis • Hypertension

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a neuro-radiological condition characterized by headache, seizures, visual changes, and vasogenic edema on imaging [1]. PRES results from impaired cerebral autoregulation and endothelial dysfunction, which disrupts the blood–brain barrier (BBB) [2].

PRES is commonly associated with conditions like eclampsia, autoimmune disease, sepsis, organ transplantation, and exposure to cytotoxic or immunosuppressive drugs. However, a rare but important link has been described between PRES and post-streptococcal glomerulonephritis (PSGN), an immune complex-mediated kidney disease characterized by hematuria, edema, hypertension, and renal dysfunction [3]. A combination of laboratory testing and clinical assessment is used to diagnose post-streptococcal glomerulonephritis [4]. Although rare in PSGN, nephrotic-range proteinuria has been observed in adults and can be a sign of more severe glomerulonephritis [5].

PRES often occurs in children with PSGN, but it is rare in adults [6]. Although PRES is frequently associated with severe hypertension, it can also develop with mild blood pressure elevations, especially when renal or immune factors lower the autoregulatory threshold [2,7].

Neuroimaging usually shows parieto-occipital vasogenic edema, best detected with magnetic resonance imaging (MRI), especially fluid-attenuated inversion recovery MRI (FLAIR) and diffusion-weighted imaging sequences, which are more sensitive than computed tomography (CT) [8]. Management centers on controlling blood pressure, managing seizures, and treating the underlying cause [9]. While most patients improve within days to weeks, severe complications such as intracerebral hemorrhage, ischemia, or status epilepticus can occur, highlighting the importance of early recognition to prevent possibly irreversible neurological damage [7,10].

In this report, we describe a case of PRES associated with PSGN occurring in a young man with mild-to-moderate hypertension, emphasizing the importance of clinical vigilance even without severe BP elevation.

Case Report

A 20-year-old Sudanese man with no known chronic medical conditions arrived at the emergency department with concerns of a severe headache and sudden bilateral visual loss. He reported that the symptoms started 48 hours before admission (Table 1), beginning with a sudden, intense occipital headache. The pain was described as throbbing and dull-aching, radiating to the frontal area, and persisted throughout the day. It was not relieved by over-the-counter analgesics. He denied experiencing symptoms such as vomiting, photophobia, phonophobia, or neck stiffness.

Approximately 8 hours after the headache began, he noticed blurring of vision in his right eye, which quickly worsened to complete, painless visual loss. The peripheral field was affected first, then the central field. Over the next few hours, the same occurred in his left eye. By the time he sought medical help, he reported complete blindness with no light perception in either eye. Despite the severity of his vision loss, he remained alert and oriented, and did not experience weakness, speech difficulty, or loss of consciousness. The patient subsequently developed generalized tonic–clonic seizures and was diagnosed with encephalitis by a local physician. He received intravenous acyclovir; however, there was no clinical improvement. He was then referred to Khartoum Hospital. During transfer,

Table 1. Structured chronological timeline of clinical presentation, key diagnostic findings, and outcome.

Time	Event
2 weeks prior	Pustular skin infection treated with antibiotics
48 h before admission	Sudden severe occipital headache
8 h later	Rapid right eye visual loss → progressed to bilateral blindness
Before referral	Developed seizures; IV acyclovir started (no improvement)
During transfer	Two generalized tonic–clonic seizures
On admission	BP 147/86 mmHg; papilledema; edema; AKI; proteinuria; hyponatremia (125 mmol/L); elevated ASO/anti-DNase B; low C3
Same day	CT brain suggestive of PRES
Day 4	MRI revealed radiological features of PRES
2-week follow-up	Complete clinical and laboratory recovery

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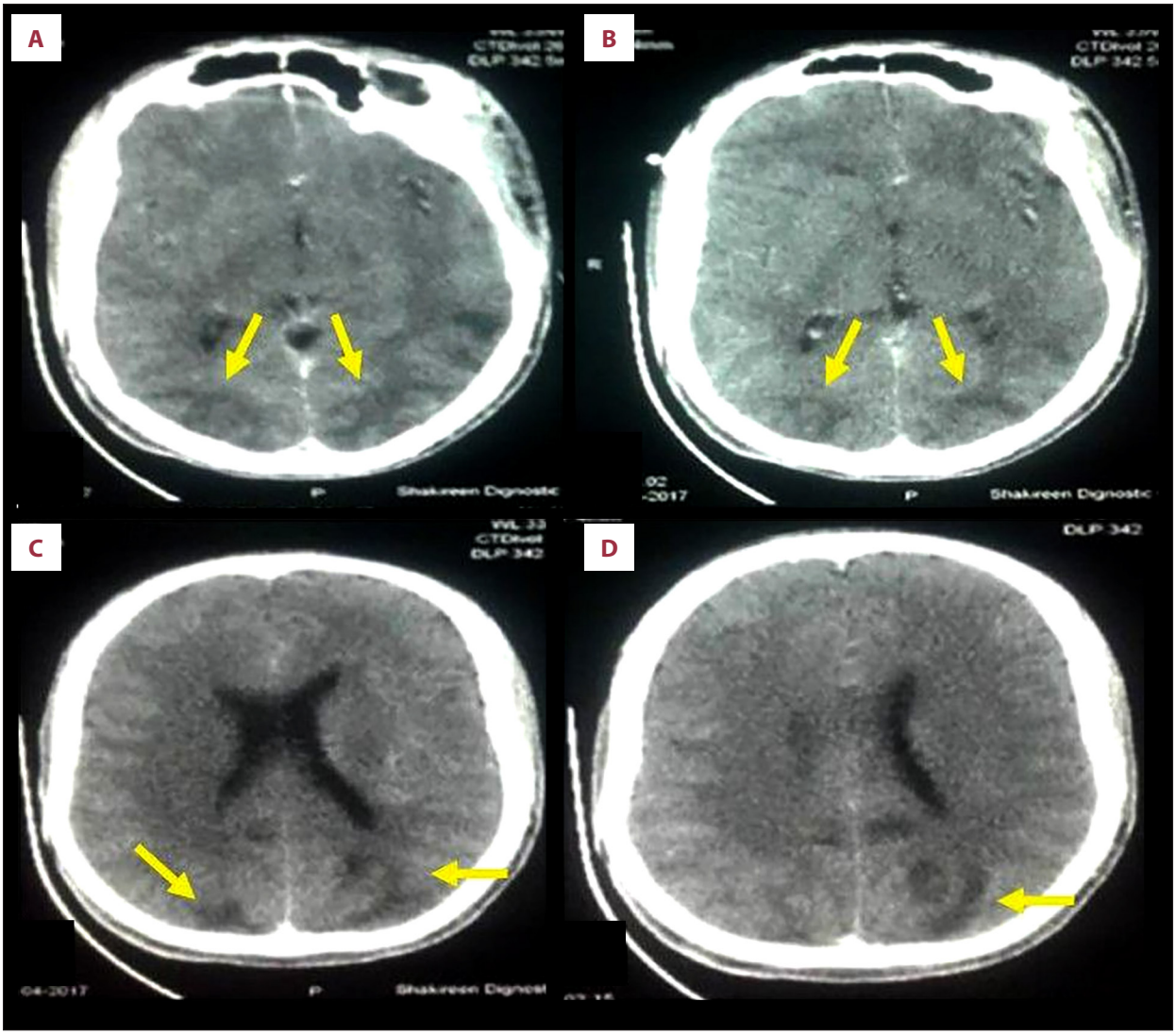

Figure 1. Axial unenhanced CT brain images (**A-D**) at different levels demonstrate bilateral, ill-defined hypoattenuation predominantly involving the subcortical white matter of the occipital lobes (yellow arrows), consistent with vasogenic edema. The remainder of the brain parenchyma appears preserved.

he experienced 2 separate generalized tonic–clonic seizures, each lasting approximately 2 minutes.

Two weeks before this presentation, he developed multiple painful pustular skin lesions resembling small abscesses on his lower limbs and forearms. A dermatologist examined him and prescribed topical and oral antibiotics. The lesions began to heal on their own, with minimal scarring. He denied having a fever, hematuria, weight loss, or joint pain. He also reported no prior similar episodes and had never been diagnosed with hypertension, diabetes, renal disease, or any neurological disorders. His social history was unremarkable. He did not smoke, drink alcohol, or use illicit drugs. There was no history of exposure to nephrotoxic agents, herbal medications, or industrial chemicals. He lived with his family in a crowded urban environment and had limited access to healthcare services. There is no family history of hypertension, kidney disease, or neurological disorders.

On physical examination, he appeared anxious but was conscious and oriented to time, place, and person. He had puffiness around the eyes and bilateral pedal edema. Vital signs on admission showed elevated blood pressure, with an initial reading of 147/86 mmHg and subsequent measurements remaining at similar levels. Pulse rate was 80 beats per minute and regular, temperature 36.5°C, respiratory rate 18 breaths per minute, and oxygen saturation 99% on room air. Neurological examination revealed no light perception in both eyes; pupils were equal and reactive to light, and fundoscopic exam showed bilateral grade 2 papilledema. Other cranial nerves

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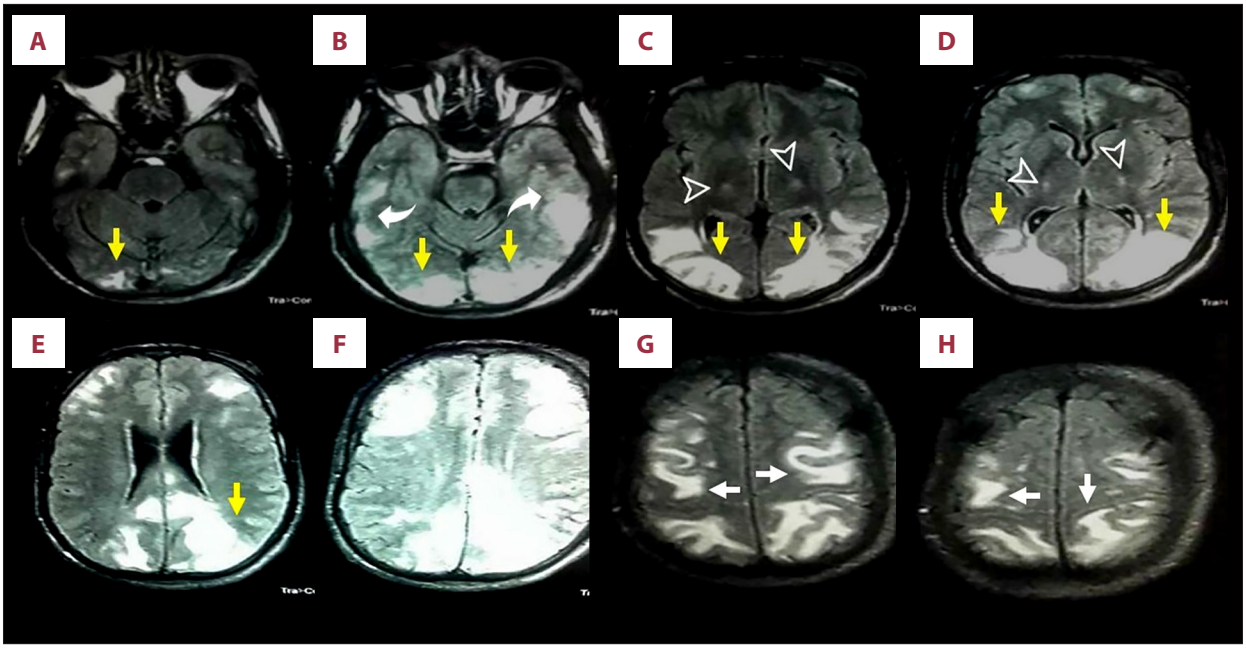

Figure 2. Axial T2-weighted and FLAIR MRI images of the brain (**A-H**) demonstrate the previously noted subcortical white matter abnormalities in both occipital lobes (yellow arrows), now extending to the parietal (white arrows) and inferior temporal lobes (white curved arrows). Additional bilateral basal ganglia hyperintense foci are observed (arrowheads). These changes are relatively symmetrical and correspond to increased T2/FLAIR signal intensity, in keeping with vasogenic edema. The remainder of the brain parenchyma appears unremarkable.

were intact. Motor and sensory exams indicated normal tone, with power 5/5 in all 4 limb muscle groups, intact sensations, and symmetric reflexes, including plantar flexion bilaterally. Cerebellar signs could not be assessed due to visual loss, and no meningeal signs were present. Skin examination revealed multiple healing pustular lesions with post-inflammatory hyperpigmentation on the legs and forearms.

The patient's laboratory tests showed normal white blood cell count, hemoglobin, and platelets. Renal function tests revealed elevated urea at 70 mg/dL and creatinine at 1.7 mg/dL, suggestive of acute kidney injury. Electrolytes were mostly normal, except for mild hyponatremia at 125 mmol/L. Urinalysis indicated 3+ proteinuria, granular casts, microscopic hematuria, and red blood cell casts. His 24-h urine protein was 6.88 g/day. The ASO titer was significantly elevated at >400 IU/mL, along with an increased anti-DNase B titer of 732 IU/mL (normal <300 IU/mL). CRP was 22 mg/L, and ESR was 35 mm/h, both mildly elevated. ANA and ANCA were negative, which suggests that autoimmune and post-staphylococcus-associated glomerulonephritis were unlikely. Complement levels showed C3 mildly decreased to 0.7 g/L (normal 0.9-1.8 g/L), while C4 was normal at 0.3 g/L (normal 0.1-0.4 g/L).

Immediately after control of seizures, a CT brain scan showed bilateral, poorly defined areas of hypoattenuation, mainly affecting the subcortical white matter of the occipital lobes, consistent with vasogenic edema. These changes were most evident on axial non-contrast images and are typical of posterior reversible encephalopathy syndrome (PRES). The rest of the brain tissue appeared intact, with no signs of hemorrhage, mass effect, or midline shift (**Figure 1**). Because of the difficult accessibility to MRI, 4 days later, MRI of the brain, including axial T2-weighted and FLAIR sequences, confirmed the previously mentioned subcortical white matter abnormalities in both occipital lobes, extending into the parietal and inferior temporal lobes. Additionally, bilateral hyperintense spots were observed within the basal ganglia. These signal changes were mostly symmetrical and showed increased T2/FLAIR signal intensity, indicating vasogenic edema. The remaining brain tissue appeared normal, with no evidence of hemorrhage, diffusion restriction, or mass effect (**Figure 2**). Renal ultrasound revealed normal kidney size, with increased echogenicity, and no obstruction. Echocardiography showed no left ventricular hypertrophy, with preserved systolic function.

Our patient was diagnosed with posterior reversible encephalopathy syndrome (PRES) secondary to acute post-streptococcal glomerulonephritis and mild blood pressure elevation based on clinical profile and the evidence of recent Group A β-hemolytic Streptococcus (GAS). He was started on amlodipine 10 mg and valsartan 160 mg daily. The Nephrology team initiated methylprednisolone 1 g infusion daily for 5 days, followed by oral prednisolone 20 mg/day tapered over 1 month.

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Although posterior reversible encephalopathy syndrome was considered the cause of the seizures, the seizure threshold might have been lowered by concomitant hyponatremia (125 mmol/L). A 100 mL intravenous bolus of hypertonic saline (3% NaCl) was administered over 10 min, with serum sodium levels closely monitored. The goal of the correction was to raise the level by 4 to 6 mmol/L. Carbamazepine 200 mg twice a day was started for seizure control because other drugs were unavailable. Serum sodium levels were adjusted concurrently and were monitored throughout the hospitalization, with no clinically significant worsening seen.

At follow-up after 2 weeks, the patient's blood pressure had improved significantly, and results of a fundus examination were normal. Renal function tests showed: blood urea 26 mg/dL, serum creatinine 0.8 mg/dL, sodium 138 mmol/L, potassium 3.52 mmol/L, phosphorus 2.9 mg/dL, calcium 8.0 mg/dL, and uric acid 3.8 mg/dL, with no neurological sequelae. Unfortunately, because of the patient's financial situation, follow-up neuroimaging was not feasible.

Discussion

PRES is a neuro-radiological condition associated with various conditions, such as preeclampsia and lupus nephritis, but rarely with PSGN [1]. Here, we report a case of an unusual association between PRES and PSGN in an adult, characterized by subtle blood pressure changes, emphasizing the importance of early recognition of PRES in atypical situations and timely management to improve outcomes and prevent complications.

The primary theory of PRES pathophysiology is cerebral hyperperfusion, caused by elevated blood pressure [11]. However, 15% to 20% of patients have low to normal blood pressure, which leads to endothelial dysfunction and loss of autoregulation caused by endotoxins or exotoxins [12]. Our patient had blood pressure readings of 140-155/82-100 mmHg. This level of hypertension is considered mild-to-moderate compared to levels usually associated with PRES, indicating that additional mechanisms besides pressure alone contributed to the development of the condition.

A focused literature search was performed in PubMed, Scopus, and Google Scholar using the terms PRES and PSGN. The study included English-language case reports and series describing PRES associated with PSGN, excluding cases of other infection-related glomerulonephritis. Separate analyses for pediatric and adult cases revealed neurological symptoms, like seizures and visual disturbances, alongside hypertension and renal impairment, including proteinuria and hematuria. Most cases of PRES due to PSGN were found in pediatric patients, although a recent report documented a rare adult case confirmed by renal pathology (**Table 2**) [6,13-16].

PRES is typically triggered by skin infections rather than streptococcal pharyngitis. In those reports, macroscopic hematuria often prompted renal evaluation, leading to the diagnosis of PSGN [17]. Our case was initially misdiagnosed as encephalitis based on the seizures, absence of hematuria, and subtle blood pressure changes. This underscores the importance of maintaining high clinical suspicion in patients, especially those with a history of infection and new neurological symptoms, with or without elevated blood pressure.

Infection-related glomerulonephritis can result from post-streptococcal GN, active staphylococcal infection, or other bacterial causes, particularly in adults, and distinguishing them is clinically important [18]. In our case, the elevated ASO, increased anti-DNase B titer, and negative ANA and ANCA, which suggest autoimmune and post-staphylococcus-associated glomerulonephritis, are unlikely. Alternative causes of PRES were evaluated, ruling out before referral autoimmune diseases or vasculitis. Despite modestly elevated blood pressure, it did not approach the range associated with hypertensive encephalopathy. This leads to the conclusion that PSGN-associated endothelial dysfunction was a probable contributor.

A slight decline in C3 and nephrotic-range proteinuria were present in our case. Nephrotic-range proteinuria in adults with PSGN has been noted and can indicate more severe glomerulonephritis [5].

Treatment mainly targets controlling blood pressure and addressing the root cause. Guidelines suggest lowering the mean arterial pressure by 25% in the first few hours, then gradually returning to normal [19]. In our case, this was achieved with intravenous hydralazine, followed by oral amlodipine and valsartan. Seizures were managed with oral carbamazepine. Similar treatment successes have been reported in other cases using medications such as nifedipine, furosemide, nicardipine, and labetalol [17].

In our case, the Nephrology team prescribed a 1 g methylprednisolone infusion daily for 5 days, which, based on our observations, improved the clinical findings. However, when blood pressure control and supportive treatment are being administered at the same time, it is impossible to conclusively determine the independent contribution of steroids. Therefore, the role of corticosteroids in PRES is a matter of controversy. A systematic review showed the positive effect of steroids in managing PRES cases, which was attributed to their ability to reduce the inflammatory endothelial injury that underlies vasogenic edema and to stabilize the permeability of the BBB. This was most notably observed in cases with underlying inflammatory or autoimmune diseases [19]. It may also have a role in PSGN patients by reducing systemic inflammatory cytokines and immune complexes that can worsen endothelial dysfunction.

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Table 2. Summary of reported PRES cases associated with PSGN, including clinical features, imaging findings, management, and outcome.

Author/ year	Age/ sex	Neurological manifestations	Renal findings	Blood pressure at presentation	Neuroimaging findings	Management	Outcome
Castellano-Martinez A [13]/2022 first case	11 yo male	Somnolence, seizures, headache, and blurry vision.	Hematuria and acute kidney injury	160/100 mmHg (95th percentile)	Symmetrical areas of cortico-subcortical hyperintensity in the temporo-occipital and frontoparietal regions of both cerebral hemispheres	Antihypertensive and anticonvulsant	Complete recovery
Castellano-Martinez A [13]/2022 second case	9 yo male	Seizures and headache	Hematuria and acute kidney injury	170/110 mmHg (95th percentile)	Symmetrical areas of cortico-subcortical hyperintensity in the temporo-occipital and frontoparietal regions of both cerebral hemispheres	Antihypertensive and anticonvulsant	Significant improvement with persistent microscopic hematuria
Bazhu [14]/ 2022	14 yo female	Headache, dizziness, seizures, and vomiting	Hematuria (3+), proteinuria and azotemia (urea 7.33 mmol/L, serum creatinine 168 µmol/L)	150/90	High-intensity signals in the bilateral frontal, parietal, and occipital lobes	Antihypertensive and anticonvulsant	Significant improvement
Kharbat AF [15]/ 2022	8 yo male	Seizures, blurred vision, headache	Hematuria and renal dysfunction	155-192/ 114-132	Subcortical edema of various brain regions, including occipital, temporal, and parietal cortices	Antihypertensive and anticonvulsant drugs	Improved with stable blood pressure
Harikrishna GV [16] /2023	15 yo male	Headache, visual disturbances, and involuntary movements of all 4 limbs	Proteinuria and hematuria	150/100 mmHg (99th percentile as per the Indian Pediatric Association)	Symmetrical enhancement of superficial and deep watershed areas, predominantly in the occipital and temporal regions	Antihypertensive and anticonvulsant	Complete resolution

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Table 2 continued. Summary of reported PRES cases associated with PSGN, including clinical features, imaging findings, management, and outcome.

Author/ year	Age/ sex	Neurological manifestations	Renal findings	Blood pressure at presentation	Neuroimaging findings	Management	Outcome
Han Q [6]/ 2025	22 yo female	Headache, consciousness disturbance, and seizures	Hematuria (3+), proteinuria (3+) and azotemia (urea 14.2 mmol/L, serum creatinine 187.8 µmol/L)	132/84	Multiple edemas in the cortex and subcortical areas of the bilateral cerebral hemispheres	Antihypertensive and anticonvulsant	Complete recovery
Presented case	20 yo male	Headache, loss of vision and seizures	3+ proteinuria, granular casts, microscopic hematuria, red blood cell casts and acute kidney injury	147/86	Bilateral, poorly defined hypoattenuation mainly involving the subcortical white matter of the occipital lobes	Antihypertensive, anticonvulsant, and steroids	Complete recovery

However, since steroids have also been linked in the literature to causing PRES by inducing hypertension and loss of autoregulation [20], it is very important to carefully evaluate each case. Our patient's response suggests that, in carefully selected cases of PSGN-associated PRES with prominent inflammatory features and after strict blood pressure control, a short course of steroids may be reasonable, while recognizing the need for controlled studies and the potential for harm in certain situations.

PRES is generally reversible when recognized and treated promptly. Our patient showed clinical improvement starting on day 1, with vision returning by day 3 and full recovery by day 5. Other reports have demonstrated clinical and radiologic resolution within 2 to 6 weeks [16,21].

The main limitation of this report is the lack of an electroencephalogram (EEG) to document seizure burden and follow-up imaging to verify complete radiological resolution. This case highlights the importance of being aware of PRES as an uncommon yet significant complication of PSGN, especially in patients showing signs like acute hypertension, seizures, and cortical blindness. Early clinical suspicion, along with prompt recognition, imaging, and aggressive treatment, is essential to prevent permanent neurological damage and ongoing cortical blindness.

Conclusions

We describe the case of a 20-year-old Sudanese man who developed PRES in the context of PSGN, despite only modestly elevated blood pressure. This underscores an uncommon but clinically significant link between PRES and PSGN. Clinicians should maintain a high suspicion for PRES even without severe hypertension, especially in patients with recent streptococcal infection and renal involvement; however, careful exclusion of alternative causes is essential. Early recognition and prompt management of hypertension and seizures are vital to achieving full clinical and radiological recovery and preventing irreversible neurological damage.

Department and Institution Where Work Was Done

Department of Neurology, Wad Madani Teaching Hospital, Madani, Sudan.

Consent to Participate

The patient provided written informed consent before manuscript submission.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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