Case Report / Olgu Sunumu

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When the Pressure Is Not High: A Case of PRES in a Normotensive Child

Basınç Yüksek Değilken: Normotansif Bir Çocukta PRES Olgusu

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiological syndrome due to disturbances in the blood-brain barrier that cause vasogenic oedema. It has been postulated that PRES is caused by disturbances of cerebral autoregulation secondary to acute hypertension, and this is often seen in patients with acute kidney injury. We report a case of PRES in a 6-year-old girl with severe polyserositis and acute kidney injury. She had no neurological symptoms at presentation, and was covered initially for sepsis despite no positive culture. The autoimmune screen was negative. On day 13 of admission, she developed right-sided focal seizures. Clinical examination showed upper motor neuron signs. Pupils were reactive, and consciousness was intact. A contrast-enhanced computed tomography of the brain suggested vasogenic oedema with prominent sulci and ventricles. Subsequently, magnetic resonance imaging of the brain confirmed the diagnosis. She was started on intravenous phenytoin, and no further seizures were observed. A clinical examination one week later showed normal neurological findings. Consequently, phenytoin was gradually tapered, and she remains well with no sequelae. Symptoms of PRES are typically preceded by hypertension or pronounced fluctuations in blood pressure. There are limited reports of PRES in pediatric patients with normal blood pressure. Despite clinical suspicion of an autoimmune disorder, this patient did not fulfil the criteria for systemic lupus erythematosus, and all autoimmune markers were negative. She was later subsequently diagnosed with diffuse large B-cell lymphoma and succumbed to the disease one year later. Furthermore, this patient demonstrates frontal lobe involvement and microhemorrhages, which are absent in adult cohorts but are consistent with previous reports in children with PRES. The clinical and radiological manifestations of PRES remain elusive in the pediatric population and warrant further research.

Öz

Posterior reversibl ensefalopati sendromu (PRES), kan beyin bariyerindeki bozuklukların vazojenik ödeme neden olduğu bir klinik-radyoloji sendromudur. PRES'in akut hipertansiyon nedeniyle serebral otoregülasyonundaki bozukluklardan kaynaklandığı ve bunun sıklıkla akut böbrek hasarı olan hastalarda görüldüğü öne sürülmüştür. Bu çalışmada şiddetli poliseroz ve akut böbrek hasarı olan 6 yaşındaki bir kız çocuğunda PRES olgusu sunulmaktadır. Hasta başvuru sırasında nörolojik semptom göstermiyordu ve kültür sonucu pozitif olmamasına rağmen başlangıçta sepsis tedavisi gördü. Otoimmün tarama negatifti. Hastaneye yatışının 13. gününde sağ taraflı fokal nöbetler gelişti. Klinik muayenede üst motor nöron bulguları saptandı. Göz bebekleri reaktifti ve bilinç bozukluğu yoktu. Kontrastlı bilgisayarlı tomografi bulguları, belirgin sulkus ve ventriküller ile birlikte vazojenik ödem olduğunu gösterdi. Daha sonra yapılan manyetik rezonans görüntüleme ile tanı doğrulandı. Hastaya intravenöz fenitoin tedavisi başlandı ve başka nöbet görülmedi. Bir hafta sonra yapılan klinik muayenede nörolojik açıdan normal olduğu görüldü. Sonuç olarak, fenitoin dozu kademeli olarak azaltıldı. Hasta herhangi bir sekel kalmadan iyi durumda idi. PRES semptomları genellikle hipertansiyon veya belirgin kan basıncı dalgalanmaları ile başlar. Ancak, bu hasta hastanede kaldığı süre boyunca normotansif kaldı. Otoimmün bozukluk şüphesi olmasına rağmen, bu hasta sistemik lupus eritematozus kriterlerini karşılamadı ve tüm otoimmün belirteçler negatifti. Daha sonra yaygın B hücreli lenfoma teşhisi kondu ve bir yıl sonra hayatını kaybetti. Ayrıca, bu hastada frontal lob tutulumu ve mikrokanamalar da görülmektedir. Bu bulgular yetişkin kohortlarında görülmemekle birlikte, PRES'li çocuklarda daha önce bildirilen bulgularla tutarlıdır. PRES'in pediyatrik popülasyondaki klinik ve radyolojik belirtileri hala belirsizdir ve daha fazla araştırma yapılması gerekmektedir.

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Abstract

Keywords: Posterior reversible encephalopathy syndrome, magnetic resonance imaging, posterior leukoencephalopathy syndrome

Öz

Anahtar Kelimeler: Posterior reversibl ensefalopati sendromu, manyetik rezonans görüntüleme, posterior lökoensefalopati sendromu

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a rare clinico-radiological condition that may occur in adult and pediatric patients.[1] It was first described by Hinchey et al.[2] in 1996 as a reversible neurological condition presenting with headache, altered consciousness, and seizures, associated with white matter oedema on radiological imaging. The exact mechanism of PRES is still not fully understood.[3] However, the postulated mechanism underlying it involves perturbed cerebrovascular autoregulation and endothelial dysfunction, resulting in vasogenic oedema that most commonly affects the parieto-occipital white matter.[3]

The common precipitating factors among pediatric patients include hypertension, renal disease, systemic infection, autoimmune conditions, and oncological disease or immunosuppressive therapy.[1,4] It has been increasingly recognized in the pediatric population, which has prompted growing interest in its pathophysiology, presentation, and management.[4] Management of PRES is supportive and involves treating the underlying cause. Despite its typically reversible nature, delayed recognition and treatment can lead to irreversible neurological damage or fatal outcomes, occurring in as many as 16% of cases.[2]

Case Report

A previously healthy 6-year-old girl presented with focal seizures on day 14 of admission to the intensive care unit (ICU). She initially presented with respiratory distress, serositis, and multi-organ failure, requiring mechanical ventilation. Neuroimaging revealed vasogenic oedema consistent with PRES. Informed consent was obtained from the patient's parents.

A 6-year-old girl of Mediterranean origin presented to the emergency department with a three-month history of breathlessness, lethargy, and constipation. Over the preceding year, she had experienced weight loss, reduced appetite, polyarthralgia, and intermittent vasculitic rashes, but had not sought medical attention due to financial constraints. She was born at term as part of a monochorionic monoamniotic twin pair to consanguineous parents. Her twin sister had been diagnosed with systemic lupus erythematosus at the age of five, and there was a strong family history of autoimmune diseases and malignancies.

On examination, she was cachectic and in severe respiratory distress, requiring immediate intubation. She was hypoxic, pale, and exhibited muscle wasting. Respiratory assessment revealed bilateral crepitations and reduced air entry. Abdominal examination showed gross ascites and a large palpable mass. Neurologically, she was conscious with normal tone, power, and reflexes; cranial nerve function was intact. Notably, she was normotensive and had normal renal function at presentation.

She was intubated, and bilateral chest drains and peritoneal drains were inserted. Contrast-enhanced computed tomography (CECT) of the thorax, abdomen, and pelvis revealed bilateral adnexal masses with features suggestive of ovarian malignancy, including omental caking, peritoneal thickening, and lymphadenopathy. Additional findings included bilateral pleural effusions, ascites, and enlarged kidneys. She was treated for serositis complicated by multi-organ failure and investigated for an underlying malignancy or an autoimmune disorder. Despite extensive imaging and fluid analysis, cytology and immunophenotyping were inconclusive, and autoimmune screening was negative (Tables 1 and 2). Surgical exploration of the abdominal mass was not performed because the patient was critically ill.

On day 13 of ICU admission, she developed focal tonic seizures affecting the right upper limb that lasted one hour and necessitated loading and maintenance doses of phenytoin. During this time, she received mechanical ventilation without haemodynamic support. Neurological examination showed bilateral hypertonicity of the lower limbs, hyperreflexia, and clonus, while the pupils remained reactive and equal. Neurological examination of the upper limbs was normal. Her level of consciousness could not be reliably assessed because she was intubated and sedated.

At this time, her blood pressure remained between the 50th and 95th percentiles, with no marked variability. Trends in her systolic and diastolic blood pressure are shown in Figure 1.

At this point, her medications included intravenous meropenem, dexmedetomidine, fentanyl, and clonidine. She had acute kidney injury with hyperkalemia and hypophosphatemia a week prior to the seizure; the kidney injury was attributed to tumor lysis syndrome. However, her renal profile and electrolyte levels normalized two days prior to the seizure. There was no evidence of other end-organ failure at this point. All cultures were sterile. Autoimmune

Table 1. Results of pleural and peritoneal fluid

Parameter	Pleural fluid	Peritoneal fluid	Remarks
pH Total protein (g/L) LDH (U/L)	7.8 40.84 3861	N/A	Serum: Protein: 70.13 LDH: 2663
Gram stain Appearance WBC (x10^6/L) RBC (x10^6/L) Mononuclear cell (x10^6/L) Polymorphonuclear (x10^6/L) Total count WBC (x10^6/L) Culture and sensitivity	Nil Cloudy 6965 12000 6037 928 7800 No growth	Nil Slightly cloudy 2419 5000 2264 155 2444 No growth	
AFB Mycobacterium C&S	Negative No growth	Negative No growth	
Cytology	No atypical cells	No atypical cells	
Immunophenotyping	Inconclusive results	Blood tap sample. 7% cluster of small sized B cells lacking surface light chains.	

N/A: Not applicable, Nil: None detected, WBC: White blood cell, RBC: Red blood cell, AFB: Acid fast bacilli, C&S: Culture and sensitivity

Table 2. Investigation results

Investigations	Results	Reference range	Interpretation
Complement C3 Complement C4	1.34 g/L 0.24 g/L	0.82-1.85 g/L 0.15-0.53 g/L	Normal Normal
ANA screening and immunofluorescence	Negative		
Anti-mycoplasma antibody	Negative		
Coombs test	Negative		
Antiphospholipid antibody panel	Negative		
ENA	Negative		
Rheumatoid factor	Negative		
Immunoglobulin level Immunoglobulin G Immunoglobulin A Immunoglobulin M	 3.87 g/L 2.86 g/L 0.99 g/L	 0.52-16.31 0.21-2.82 0.47-2.4	 Low Elevated Normal
Alpha feto-protein	2.457 ng/mL	0-8.1	Normal
Beta human chorionic gonadotropin	<2 mIU/mL	0-6	Normal
CA-125	1 300 U/mL	0-35	Elevated
CEA	<0.5 ng/mL	0-5.1	Normal
Parathyroid hormone	3.04 pmol/L	1.58-6.03	Normal
25-hydroxyvitamin D	17.63 ng/mL	20-40	Low
Procalcitonin	17.05 ng/mL	0.03-0.1	Elevated

ANA: Antinuclear antibody, ENA: Extractable nuclear antigen, CA: Cancer antigen, CEA: Carcinoembryonic antigen

and viral screenings were negative. An electroencephalogram was not available in our setting at the time. A CECT scan of the brain demonstrated white matter hypodensities in the bilateral superior fronto-parietal lobes with effacement of the adjacent sulci, suggesting vasogenic oedema (Figure 2A-B).

Magnetic resonance imaging (MRI) of the brain was performed the next day. The MRI demonstrated symmetrical abnormal signal intensities in the bilateral superior fronto-parietal lobes, which were hypointense on T1-weighted images and hyperintense on T2/fluid-attenuated inversion recovery (FLAIR) sequences (Figure 3A-C). No cortical thinning or enhancement was observed after gadolinium administration (Figure 3D).

Diffusion-weighted imaging and apparent diffusion coefficient mapping showed facilitated diffusion, consistent with vasogenic oedema (Figure 3E-F). Additionally, multiple foci of blooming artefacts were observed on the gradient-recalled echo sequence within adjacent sulci, more pronounced on the right than on the left, suggestive of microhemorrhages (Figure 3G). These changes involved the subcortical and juxtacortical regions, with effacement of the grey-white matter junction (Figure 3H). The brainstem, cerebellum, and

spinal cord appeared normal; no hydrocephalus, midline shift, or venous sinus thrombosis were detected.

Anti-epileptic medication was initiated and successfully tapered off within four weeks. A week later, she was extubated and had a normal neurological examination. No follow-up brain imaging was performed due to financial constraints. She received intravenous hydrocortisone 4 mg/kg four times daily for two weeks, followed by a weaning regimen of oral prednisolone for the subsequent two weeks. During admission, she received multiple courses of antibiotics, as shown in Table 3. Consequently, a repeat CECT of the abdomen showed a significant reduction in the size of the adnexal mass. Thus, she was discharged after six weeks and followed up by the pediatric rheumatology team.

Upon discharge, her neurological examination was normal, with no motor or sensory deficits. She regained full independence in her activities of daily living and had no difficulties with feeding, urination, or bowel movements. A year later, she presented again with a left lumbar mass and constitutional symptoms, and was eventually diagnosed with diffuse large B-cell lymphoma (DLBCL). She underwent multiple courses of chemotherapy but succumbed to the disease's aggressive nature; throughout the illness she exhibited no neurological symptoms. This case illustrates an atypical presentation of PRES in a critically ill child with a suspected malignancy who had no initial hypertension, highlighting the importance of early neuroimaging for unexplained seizures.

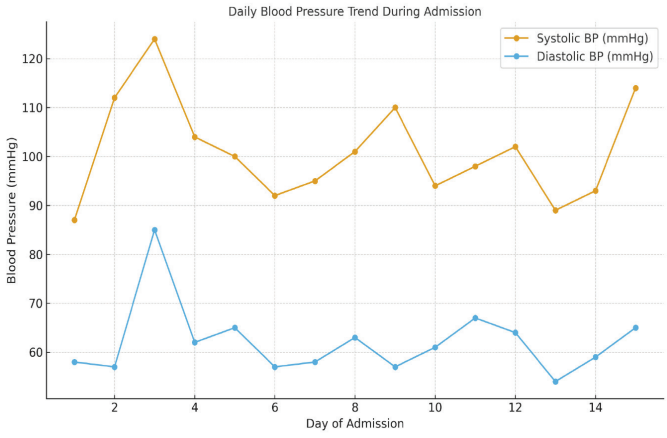

Figure 1. Blood pressure trend during PICU stay

PICU: Pediatric intensive care unit, BP: Blood pressure

Discussion

We report a case of PRES in a pediatric patient despite normal blood pressure and resolved acute kidney injury. It is an increasingly recognized clinico-radiological entity in the pediatric population, characterized by diverse etiologies, variable clinical presentations, and potential reversibility with timely intervention.[1,5]

As outlined in the introduction, the pathophysiology of PRES is thought to involve impaired cerebrovascular autoregulation.[1,2] Impaired cerebrovascular autoregulation, combined with endothelial dysfunction, is considered the primary pathophysiological mechanism leading to transient vasogenic edema of the brain parenchyma.[1,2] In this model, severe hypertension exceeds the limits of autoregulation, overwhelming endothelial capacity and impairing compensatory vasoconstriction. The resulting hyperperfusion allows plasma and macromolecules to leak from cerebral vessels, producing edema.[1,6,7]

While hypertension is a well-established trigger, particularly in the context of renal disease, PRES can also arise in normotensive or hypotensive states, as illustrated by the case reported by Adhikari et al.[5] in which hypovolemic shock was the precipitating factor. In our case, the patient developed PRES during a prolonged ICU stay, with contributing factors including systemic

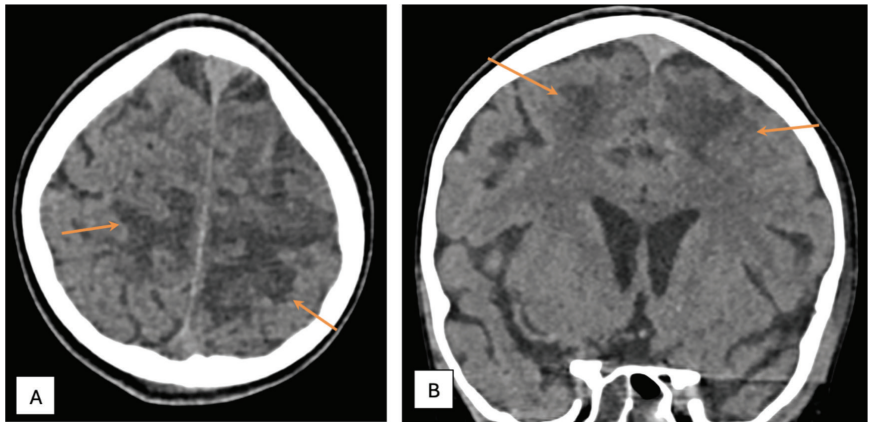

Figure 2. Contrast-enhanced CT brain in axial **(A)** and coronal **(B)** views showed symmetrical white matter hypodensities at bilateral superior fronto-parietal lobes (orange arrows). The adjacent sulci are effaced

CT: Computed tomography

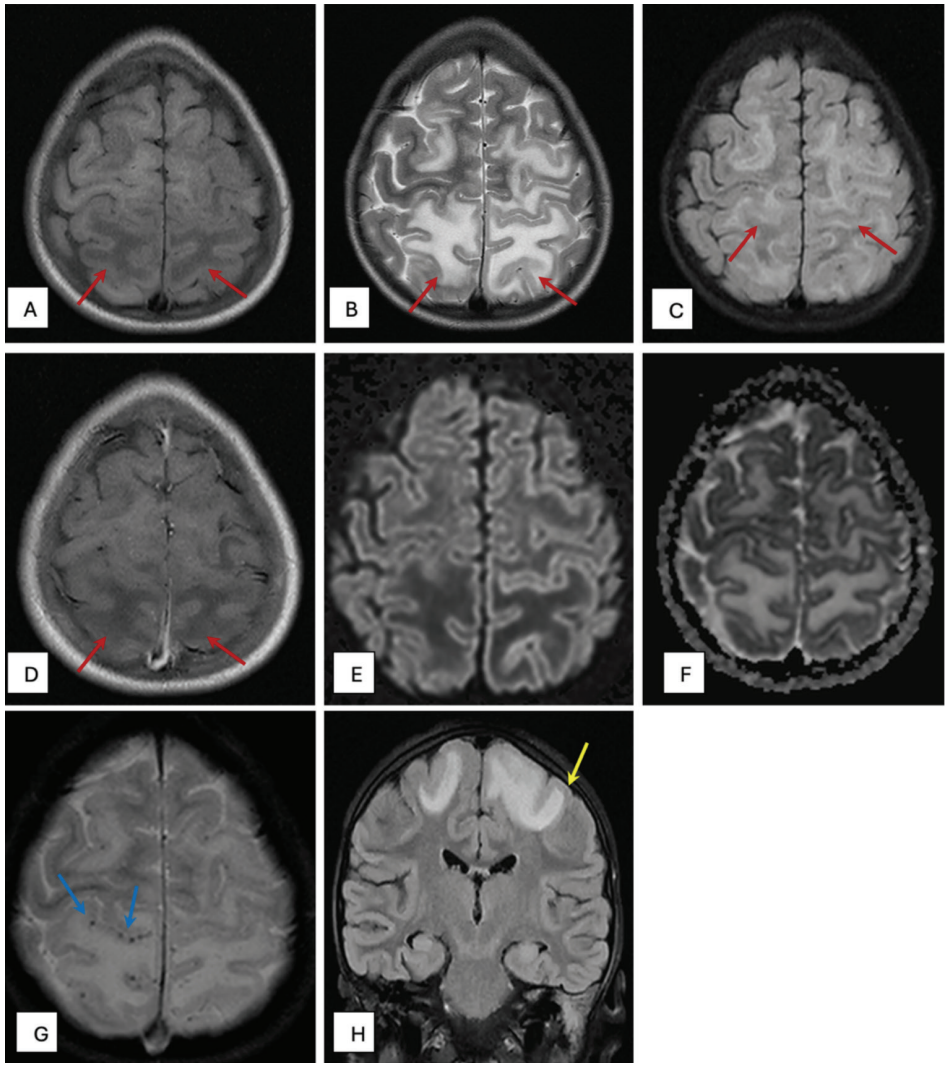

Figure 3. MRI brain in axial views revealed symmetrical white matter abnormal signal intensities at bilateral fronto-parietal lobes (red arrows), which returned hypointense signal on T1 **(A)**, hyperintense on T2 **(B)**, not suppressed on FLAIR **(C)** and no enhancement post contrast **(D)**. ADC/DWI sequences **(E,F)** showed no restricted diffusion to suggest acute infarct. Blooming artefacts on GRE suggestive of microhaemorrhages were present (blue arrow) **(G)**. The T2 weighted image in the coronal view **(H)** showed abnormal white matter signal distribution at the juxtacortical and subcortical region (yellow arrow)

MRI: Magnetic resonance imaging, FLAIR: Fluid-attenuated inversion recovery, ADC: Apparent diffusion coefficient, DWI: Diffusion-weighted imaging, GRE: Gradient-recalled echo

Table 3. Antibiotic summary

Antibiotic	Dose	Mode of administration	Duration (days)
Ceftriaxone	50 mg/kg twice daily	Intravenous	1 day
Azithromycin	15 mg/kg once daily	Intravenous	1 day
Piperacillin-tazobactam	112.5 mg/kg three times daily	Intravenous	2 days
Meropenem	20mg/kg twice daily	Intravenous	7 days
Vancomycin	25 mg/kg three times daily	Intravenous	7 days
Fluconazole	12 mg/kg once daily	Intravenous	5 days
Ceftazidime	25 mg/kg three times daily	Intravenous	5 days
Amikacin	25 mg/kg on day 1 then 18mg/kg twice daily	Intravenous	5 days
Ampicillin-sulbactam	200 mg/kg+100mg/kg three times daily	Intravenous	4 days
Linezolid	10 mg/kg three times daily	Intravenous	3 days
Nystatin	250.000 units three times daily	Oral	9 days

infection, mechanical ventilation, and preceding acute kidney injury. This aligns with the literature, in which critical illness and systemic inflammation are increasingly recognized as potential contributors to PRES pathogenesis, alongside renal dysfunction, autoimmune diseases, cytotoxic agents, and sepsis.[6-10]

Beyond hypertension, endothelial dysfunction is a critical contributing factor.[6,8,11] This can stem from a direct toxic effect of immunosuppressive or chemotherapeutic agents, or from conditions involving systemic inflammation or sepsis.[8,9,11] In some cases of chronic anemia, rapid, large-volume blood transfusions have been hypothesized to induce PRES by disrupting vascular autoregulation, resulting in hyperperfusion and acute vascular endothelial dysfunction, which can cause cerebral damage.[12] Other potential factors include reperfusion injury and, in some cases, microthrombosis associated with immunosuppressive therapy, such as systemic corticosteroids, which this patient was receiving.[1,13,14]

Given the multifactorial nature of PRES in critically ill patients, as seen in our case, accurate and timely diagnosis is essential, particularly via neuroimaging.[3,4,6] MRI is the preferred modality because its sensitivity and specificity for detecting characteristic vasogenic edema, particularly in posterior subcortical regions, are superior to those of CT scans.

The hallmark radiological feature on MRI is white matter edema, which typically appears as hyperintense signal on T2-weighted and FLAIR sequences, most commonly involving the occipital and parietal lobes. However, pediatric PRES frequently involves atypical MRI findings in regions beyond the posterior circulation, including the frontal and temporal lobes, cerebellum, brainstem, basal ganglia, thalamus, and corpus callosum.[15] Some authors suggest that these "atypical" features in adults may be considered "typical" in children.[6,7,15]

This patient's MRI showed vasogenic edema and microhemorrhages. Microhemorrhages are increasingly reported in this condition and are believed to result from blood-brain barrier disruption due to impaired cerebral autoregulation, often triggered by acute elevations in systemic blood pressure.[6,8] This leads to hyperperfusion, increasing hydrostatic pressure within cerebral capillaries and resulting in leakage of fluid into the brain parenchyma (vasogenic edema). Subsequently, this leads to the extravasation of red blood cells, which appears as petechial hemorrhages.[11] Unfortunately, follow-up imaging was not available for this patient, rendering confirmation of complete resolution of PRES difficult.

PRES in DLBCL arises via two pathways: as a paraneoplastic or metabolic presentation at diagnosis (notably with hypercalcemia and labile blood pressure) and as a toxicity of lymphoma therapy, most often during or shortly after chemotherapy initiation.[16-18] This patient developed PRES as the first manifestation of DLBCL, without hypertension or prior exposure to cytotoxic chemotherapy, similar to a reported case of DLBCL presenting with PRES in an adult with hypercalcemia and no hypertension.[17] Corticosteroid therapy and acute kidney injury were likely contributing factors, both recognized risk factors for endothelial dysfunction.

As highlighted in pediatric and oncologic cohorts, PRES may occur in normotensive patients and can precede the diagnosis of malignancy. In similar cases, clinicians should maintain a high index of suspicion for underlying hematological disease and pursue early, aggressive investigation to enable timely diagnosis and management.

Conclusion

PRES may rarely present as initial manifestation of an underlying hematologic malignancy in children. This case emphasizes the need for thorough evaluation and close observation in pediatric patients with PRES who lack an obvious precipitating factor. Early identification of the underlying cause may alter management and improve outcomes.

Ethics

Informed Consent: Informed consent was obtained from the patient's parents.

Footnotes

Authorship Contributions

Concept: A.M.K., Design: A.B.N.M., A.M.K., Data Collection or Processing: A.J.A.B.N., A.B.N.M., A.M.K., Analysis or Interpretation: A.J.A.B.N., A.B.N.M., A.M.K., Literature Search: A.J.A.B.N., A.B.N.M., A.M.K., Writing: A.J.A.B.N., A.M.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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