

Received: 2020.02.12

Accepted: 2020.03.20

Available online: 2020.05.28

Published: 2020.06.09

# Cerebellar Parieto-occipital Posterior Reversible Encephalopathy Syndrome and Cerebral Metamorphopsia Associated with Asymptomatic Atrial Septum Vegetation and Renal Disease: Case Report

## Authors' Contribution:

Study Design A  
Data Collection B  
Statistical Analysis C  
Data Interpretation D  
Manuscript Preparation E  
Literature Search F  
Funds Collection G

ABCDEF 1,2 **Arash Ghaffari-Rafi**  
BDE 3 **Anthony C. Netzel**  
AE 3 **Madeline Prat**  
DE 3 **Daniel T. Miles**

1 Queen Square Institute of Neurology, University College London, London, U.K.  
2 John A. Burns School of Medicine, University of Hawaii at Mānoa, Honolulu, HI, U.S.A.  
3 Department of Medicine, Tripler Army Medical Center, Honolulu, HI, U.S.A.

**Corresponding Author:** Arash Ghaffari-Rafi, e-mail: [arash.ghaffari-rafi.18@ucl.ac.uk](mailto:arash.ghaffari-rafi.18@ucl.ac.uk)

**Conflict of interest:** None declared

<b>Patient:</b>	<b>Female, 25-year-old</b>
<b>Final Diagnosis:</b>	<b>Posterior reversible encephalopathy syndrome</b>
<b>Symptoms:</b>	<b>Visual disturbances</b>
<b>Medication:</b>	—
<b>Clinical Procedure:</b>	—
<b>Specialty:</b>	<b>Neurology</b>
<b>Objective:</b>	<b>Rare co-existence of disease or pathology</b>
<b>Background:</b>	Posterior reversible encephalopathy syndrome (PRES) is a poorly characterized and enigmatic syndrome. Despite consistently presenting with nervous system vasogenic edema, this malady has been associated with variable triggers, neurological symptoms, and natural history.
<b>Case Report:</b>	The report presents a 25-year old African American female who presented with altered mental status and bilateral cortical blindness. Neuroimaging identified vasogenic edema in the cerebellum, parietal lobe, and occipital lobe. Her PRES was associated with a hypertensive emergency, renal failure, and an atrial septum vegetation (culture-negative endocarditis). All 3 contributing etiologies were addressed, upon which the patient began to recover. During recovery, the patient experienced cerebral metamorphopsia, visualizing her entire environment in the form of a cartoon. After 2 weeks of treatment she recovered to baseline state of health, with vasogenic edema resolved on follow-up neuroimaging.
<b>Conclusions:</b>	This case presents a rarely catalogued phenomena during PRES recovery, cerebral metamorphopsia, along with a new potential association (culture negative atrial septum endocarditis). The report also highlights how PRES recovery patients (with cortical blindness) should be explicitly assessed for cerebral metamorphopsia and Charles Bonnet syndrome – which may distress patients. Lastly, the atypical presentation of cerebellar vasogenic edema in our patient validates existing literature that PRES does not have a uniform picture and is not well served by its current name or proposed diagnostic criteria. Therefore, renaming the disorder to reversible vasogenic edema syndrome and derestricting the diagnostic criteria, may prevent clinicians from being discouraged when faced with diagnosing PRES in the face of atypical findings.
<b>MeSH Keywords:</b>	<b>Endocarditis, Subacute Bacterial • Posterior Leukoencephalopathy Syndrome • Vision Disorders</b>
<b>Abbreviations:</b>	<b>PRES</b> – posterior reversible encephalopathy syndrome; <b>ED</b> – Emergency Department; <b>CKD</b> – chronic kidney disease; <b>VP</b> – ventriculoperitoneal; <b>AKI</b> – acute kidney injury; <b>BID</b> – bis in die; <b>CT</b> – computerized tomography; <b>MRI</b> – magnetic resonance imaging; <b>BUN</b> – blood urea nitrogen

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/923441>



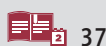
2614



1



2



37



## Background

First characterized in 1996, posterior reversible encephalopathy syndrome (PRES), also known as reversible posterior leukoencephalopathy, is vaguely defined by a diversity of neurologic symptoms, variable onset (hours to weeks), and neuroimaging findings [1,2]. The etiologies are numerous, with arterial hypertension, post-transplant immunosuppressive medications, autoimmune disorders, renal disease, and eclampsia being the leading conditions associated with PRES diagnosis [1,2]. The incidence of symptoms includes the following: encephalopathy in 28% to 92% of cases, disorders of consciousness in 67% to 90%, acute arterial hypertension or blood pressure fluctuations in 61% to 80%, epileptic seizures in 70% to 74%, visual disturbances in 20% to 67%, headache in 26% to 53%, and focal neurological signs in 5% to 15% [3]. Meanwhile, brain regions are involved in the following proportions: 83.3% to 98.7% parietooccipital, 54.2% to 78.9% posterior-frontal, 16.7% to 68.4% temporal, 30.3% thalamus, 12.5% to 34.2% cerebellum, 4.2% to 18.4% brainstem, and 11.8% basal ganglia [4–6]. Hence, the term PRES is a misnomer, as the occipital lobe is not always impacted and encephalopathy not always present. Therefore, cataloging unique cases such as this one – a 25-year-old career hypertensive with cerebellum involvement and a new atrial septum vegetation, presenting with cerebral metamorphopsia during recovery – aids in characterizing this enigmatic syndrome.

## Case Report

The patient was a 25-year old African American female presenting to the emergency department (ED), after being found on the floor at home verbally incomprehensible, with complete bilateral vision loss and altered mental status. She has a complicated medical history, but no past total blindness. Prior, she was fully independent and at baseline state of health. When found, the patient was suspected to not have been eating well or taking her medications for 4 days, including antihypertensives.

Her past medical history includes systemic hypertension of unknown cause, stage 4 chronic kidney disease (CKD) secondary to poorly controlled hypertension, idiopathic intracranial hypertension with ventriculo-peritoneal (VP) shunt, and hypertensive retinopathy with optic neuropathy (loss of peripheral vision in right eye; loss of central vision in left eye). This is the patient's third admission for acute kidney injury (AKI) on CKD. For her hypertension, she takes 12.5 mg carvedilol twice daily and 60 mg nifedipine twice daily. Her family history is unremarkable. Her social history is notable for medication non-compliance.

Admission physical examination was remarkable for a blood pressure of 168/98 mmHg and pulse of 100 beats per minute.

The patient was incomprehensible, minimally verbal, and obtunded. Neurological examination was notable for total bilateral vision loss and altered mental status, but no other focal deficits were found. Once the patient was able to verbalize to pain, she emphasized left sided rib pain with coughing.

## Investigations

Due to the altered mental status, bilateral complete vision loss, and presence of a VP shunt, prompt head computerized tomography (CT) was conducted. The CT demonstrated hypodensities in the cerebellum and hyperdensities extending to the cortex in the posterior occipital lobe. The hypodensity of the cerebellum, also known as the “dark cerebellar sign,” and the hyperdensities of the occipital lobe, represent diffuse parenchymal edema or infarction [7].

Subsequently, brain magnetic resonance imaging (MRI) without gadolinium was performed to assess for vasogenic edema. MRI demonstrated T2/FLAIR hyperintensity, T1 hypointensity, and no restricted diffusion, in the cerebellum, posterior parietal and occipital lobes bilaterally – findings consistent with vasogenic edema (Figure 1).

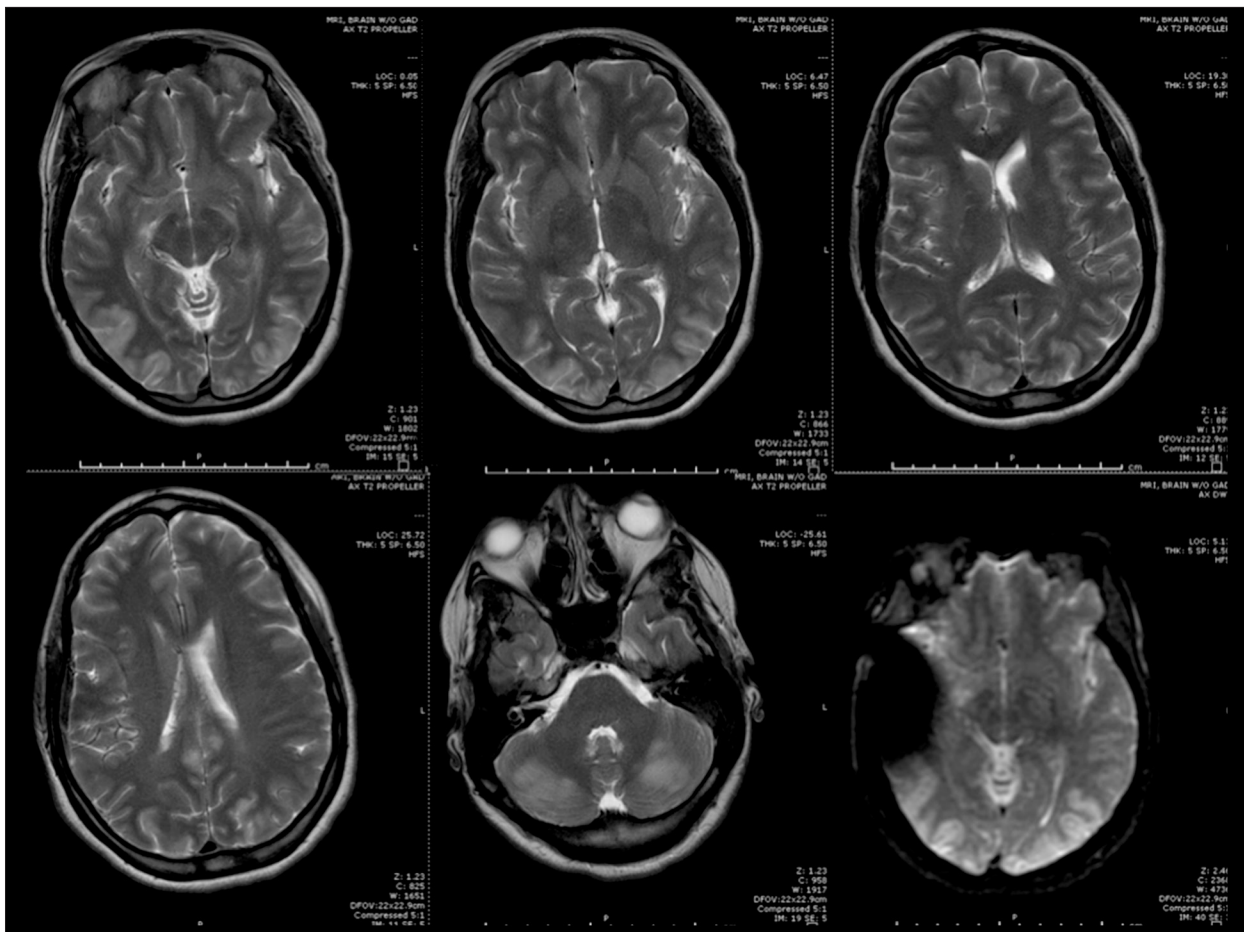
A complete metabolic panel and blood count were notable for a blood urea nitrogen (BUN) of 92.0 mg/dL and a creatinine of 17.56 mg/dL. For her left rib pain, an echocardiogram for pericarditis was conducted, but only presented a right-sided atrial septum vegetation; subsequent blood cultures were all negative (indicating culture-negative endocarditis).

## Differential diagnosis

Due to the patient's neurologic signs and symptoms, neuroimaging findings, and eventual clinical course (vasogenic edema resolution on repeat MRI), PRES was diagnosed. However, the underlying etiology is likely multifactorial, involving the atrial septum vegetation (culture-negative endocarditis), hypertension, and renal failure.

## Treatment

The patient was initially in the intensive care unit to control her blood pressure and altered mental status. She received neurological examination checks every 2 hours and had her VP shunt interrogated by the neurosurgery team (unremarkable assessment). The patient's blood pressure was controlled with labetalol and hydralazine; then she was placed on clevidipine to slowly reduce the blood pressure by 25% within 6 hours. With time, the patient was eventually transitioned to a daily regimen of 12.5 mg carvedilol twice daily (by mouth), 20 mg lisinopril daily (by mouth), and 30 mg nifedipine twice daily (by mouth), all to maintain her blood pressure.



**Figure 1.** Vasogenic edema in the cerebellum, parietal lobe, and occipital lobe. The magnetic resonance imaging (MRIs) without gadolinium, taken on admission, demonstrate T2/FLAIR hyperintensity, T1 hypointensity, and no restricted diffusion in the cerebellum along with the bilateral posterior parietal and occipital lobes. Additionally, the gray and white matter differentiation is preserved with no abnormal signal intensities within the brain parenchyma.

Regarding her renal function, the patient received a tunneled catheter and subsequently began dialysis treatment; she also began taking sevelamer carbonate. For her atrial septal vegetation, she was begun on an empiric antibiotic regimen for culture-negative endocarditis (vancomycin and ceftazidime).

### Outcome and follow-up

Over several days as blood pressure stabilized, the patient regained baseline cognition (becoming verbally fluent and oriented to person, place, and time) and vision.

She first began to see light, but then noted visual distortion, by visualizing all objects and shapes as “cartoons” – a form of cerebral metamorphopsia; no hallucinations were present, ruling out Charles Bonnet syndrome. Other than noting her entire environment appeared as if a cartoon, she was unable to further characterize her visual distortions, and no further visual deficits were noted (even after formal neurologist assessment).

Eventually, after 1-week from admission, the metamorphopsia resolved and she regained baseline vision. Outpatient follow-up MRI 2 weeks later was unremarkable, including for vasogenic edema (Figure 2).

### Discussion

#### Pathophysiology

Two theories, the toxic and hyperperfusion, predict PRES pathogenesis – our case supports both [3,8]. The toxic theory presumes toxic molecules trigger pro-inflammatory cytokine release, which causes endothelial dysfunction with eventual blood pressure elevation [8]. Toxins compromise endothelial adhesion molecules, leading to vascular leakage [8]. Endothelial compromise then triggers further damage, via release of pro-inflammatory mediators and vasoactive substances (e.g., nitric oxide, thromboxane A2 or endothelin-1) [2,9]. These substances





Epidemiologic data both endorses and refutes the hyperperfusion theory, for despite most patients experiencing hypertension or rapid blood pressure fluctuations at syndrome onset, roughly 30% have normal pressures [21,22]. Rather, those with normal blood pressure support the idea that PRES is cerebrovascular autonomic dysfunction disorder, rather than a hypertensive disorder; instead, hypertension likely plays the role promoting the autonomic dysfunction.

The role of autonomics in PRES is further bolstered by the anatomic distribution of superior cervical ganglion sympathetics, which significantly innervate the anterior circulation relative to the posterior circulation [2]. This anatomic distribution would account for the 83.3% to 98.7% cases involving the parieto-occipital region [4–6].

However, why some experience PRES in atypical regions is unknown. One potential reason could be individual anatomic variation in cerebrovascular autonomics. On the other hand, certain patients may have had a progressive event which permitted for more widespread autonomic dysfunction. For instance, our patient had a history of recurrent hypertensive crises, which may have established a baseline dysfunction in her autonomic cerebrovascular regulation, in turn predisposing to vasogenic edema in brain regions atypical for an individual not chronically afflicted by that stressor (i.e., hypertension). Our patient's history of hypertensive emergencies may have predisposed gradual development of more dispersed autonomic dysfunction than normal, leading to vasogenic edema in the cerebellum (a region afflicted in only 12.5% to 34.2% of patients) [4–6]. Chronic hypertension could gradually induce vessel architectural changes (hyaline deposition) making them less amenable to autonomic influences.

Likewise, she may simply have less cerebellar sympathetics than average.

### Cerebral metamorphopsia

Most unique about this case is the association between PRES-induced cortical blindness with cerebral metamorphopsia (where the patient reports her surroundings appearing as a cartoon). Only one other case of PRES-induced metamorphopsia had been reported, the patient with cortical blindness observed people and objects changing sizes [24]. Although rarely reported, cerebral metamorphopsia is likely more common than suspected in PRES, as it potentially follows a similar pathogenesis with Charles Bonnet syndrome, which was found to be more prevalent than previously thought (as most patients are not asked about visual hallucinations or distortions) [25]. Hence, patients who experience any amount of visual loss from PRES, should be assessed for visual distortions and hallucinations during recovery, for these visual abnormalities may be distressing but resolved with reassurance.

**Table 1.** Proposed PRES diagnostic criteria – modification of recommendations by Fugate et al. (2010) [15].

1. Onset of new neurologic symptoms, regardless of acuity
2. Evidence of vasogenic edema on neuroimaging
3. Resolution of the vasogenic edema on follow-up neuroimaging

Lastly, increased cerebral blood flow, a physiologic change seen in PRES patients, has been linked to cerebral metamorphopsia in the literature, this likely caused our patient's visual distortion [26,27].

### Diagnosis

PRES lacks formalized diagnostic criteria. However, one proposed guideline (which should be considered for formal adoption, with modification), diagnoses PRES based on acute neurologic symptoms, focal vasogenic edema on neuroimaging, and clinical/radiographical reversibility [15]. Yet, 3 issues exist: not all cases involve acute symptom onset, focal vasogenic edema, or are fully reversible [1,2,28,29].

Moreover, regarding laboratory studies, only general trends have been uncovered. For instance, 85% of patients have decreased serum albumin, while some experience hypomagnesemia in the first 48 hours, and others present with elevated lactate dehydrogenase, creatine, or liver function parameters [3,14,30–32]. Although our patient's BUN and creatinine levels were elevated, no other trend was observed. Thus, the disparity in laboratory studies between patients indicates laboratory results cannot be used in PRES diagnosis, rather only to elucidate etiology [3,14]. Likewise, lumbar puncture is non-specific, although it can show elevated cerebrospinal fluid (CSF) albumin and CSF/serum albumin quotient (supporting blood-brain barrier disruption) [33,34].

Arguably the most important of the diagnostic criteria is identification of vasogenic edema on neuroimaging – a consistent finding in nearly all PRES patients – along with subsequent edema regression and neurologic symptom resolution [3]. For suspected PRES, due to time restriction, CT should be conducted first, and once the patient is stabilized a T2-weighted or fluid-attenuated inversion recovery (FLAIR) MRI should be ordered (since MRI is more sensitive for hyperintense lesions) [10].

Overall, adopting the diagnostic criteria set forth by Fugate et al. (2010), with slight modification to accommodate the diversity of PRES presentations, appears reasonable [15]. In particular, we propose the criteria be adjusted to involve the following components: 1) onset of new neurologic symptoms, regardless of acuity, 2) evidence of vasogenic edema on neuroimaging, and 3) resolution of the vasogenic edema on follow-up neuroimaging (Table 1).

We excluded any timeframe for symptom presentation, as there have been cases of PRES with onset ranging from hours to weeks [1,2]. Moreover, we do not specify if the vasogenic edema is focal, diffuse, or restricted to a particular nervous system region – as prior cases include diffuse whole-brain edema to atypical spinal cord involvement [1,2]. Finally, we excluded the requirement of clinical symptom resolution (as some cases have noted irreversibility of symptoms), instead requiring vasogenic edema resolution on follow-up neuroimaging (a more consistent finding) [1,2].

## Etiology

PRES has been associated with countless etiologies, including: hypertensive encephalopathy, eclampsia, thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, immunosuppressants, anti-neoplastic agents, high-dose steroids, intravenous immunoglobulins, renal disease, cirrhosis, pheochromocytoma, hypercalcemia, hypomagnesemia, erythropoietin therapy, large blood transfusion, acute intermittent porphyria, systemic lupus erythematosus, polyarteritis nodosa, Behcet's disease, contrast medium exposure, scorpion poison, stimulant abuse, digitoxin intoxication, and *Averrhoa carambola* [35]. Unique to our case is the potential multifactorial origin with chronic hypertension, culture-negative endocarditis, and renal disease.

In our patient case, the hypertension had been worked up prior, but no etiology was identified, including renal artery stenosis, polyarteritis nodosa, or pheochromocytoma. Although her hypertension was associated with PRES, her past episodes of hypertensive crisis did not yield a diagnosis of PRES – indicating a potential gradual development in cerebrovascular dysfunction.

Moreover, confounding her PRES pathogenesis was the new atrial septum vegetation, resulting in a culture-negative endocarditis diagnosis. The endocarditis could have provided an enhanced pro-inflammatory state, which would fall in line with the toxic theory for PRES. However, due to the empiric treatment for all these etiologies carried out simultaneously, the vegetation cannot be classified as a causative association or a confounding incidentaloma.

## Treatment

For treatment, due to the myriad of etiologies, each PRES case needs personalized management to address the suspected trigger. Other than the recommendation to assess for vasogenic edema resolution via neuroimaging, no standardized treatment exists in the literature [3]. In our case, although several suspect etiologies exist, addressing the hypertension and renal failure provided greatest benefit, as neurologic function

improved subsequently. Prior studies have also highlighted that addressing hypertension is vital for most cases of PRES [3,36].

The subacute endocarditis treatment was begun later (due to the vegetation being identified further in the hospital course), but still temporally correlated with cortical blindness resolution. For the altered mental status, although not conducted in our case, utilization of an electroencephalography has been recommended for identifying a non-convulsive epileptic state, an occasional encephalopathy cause in PRES [37].

## Conclusions

Overall, PRES rises from the perfect storm of cerebrovascular autonomic dysfunction and endothelial damage triggered by a host of pro-inflammatory mediators (toxins). Our case supports both the hyperperfusion and toxic theories underlying this syndrome, by associating several etiologies with the disorder, including renal failure, hypertension, and atrial septal vegetation (culture-negative endocarditis). However, to better understand the pathogenesis of PRES and determine patient predisposition, future research could involve conducting a genome-wide association study to determine the significance of genes impacting the inflammatory or autonomic pathways.

Moreover, our case highlighted an uncatalogued form of cerebral metamorphopsia (visualization of the environment as a cartoon) linked to PRES recovery. Cerebral metamorphopsia might be more common than suspected, thus clinical research should quantify how many patients during PRES recovery, after vision loss, experience cerebral metamorphopsia or Charles Bonnet syndrome.

In addition, our case supported that PRES has a vast diversity of presentations, hence experts should consider renaming the syndrome to a less restrictive title, such as “reversible vasogenic edema syndrome.” Therefore, atypical imaging findings should not dissuade the diagnosis of PRES in the appropriate clinical scenario. Along the same lines, due to the diversity of presentations, a less restrictive diagnostic criteria should be adopted to facilitate clinicians who encounter PRES throughout their practice. Our report proposes utilizing a modified version of the Fugate et al. (2010) diagnostic criteria (Table 1). In summary, this report provides not only a novel characterization of PRES, but also a brief review of the syndrome.

## Conflict of interests

None.

## References:

1. Hinchey J, Chaves C, Appignani B et al: A reversible posterior leukoencephalopathy syndrome. *N Engl J Med*, 1996; 334(8): 494–500
2. Fugate JE, Rabinstein AA: Posterior reversible encephalopathy syndrome: Clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol*, 2015; 14(9): 914–25
3. Fischer M, Schmutzhard E: Posterior reversible encephalopathy syndrome. *J Neurol*, 2017; 264(8): 1608–16
4. McKinney AM, Short J, Truwit CL et al: Posterior reversible encephalopathy syndrome: Incidence of atypical regions of involvement and imaging findings. *Am J Roentgenol*, 2007; 189(4): 904–12
5. Ni J, Zhou LX, Hao HL et al: The clinical and radiological spectrum of posterior reversible encephalopathy syndrome: A retrospective series of 24 patients. *J Neuroimaging*, 2011; 21(3): 219–24
6. Bartynski WS, Boardman JF: Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. *Am J Neuroradiol*, 2007; 28(7): 1320–27
7. Huisman TA, Kubat SH, Eckhardt BP: The “dark cerebellar sign”. *Neuropediatrics*, 2007; 38(3): 160–63
8. Bartynski WS: Posterior reversible encephalopathy syndrome, part 2: Controversies surrounding pathophysiology of vasogenic edema. *Am J Neuroradiol*, 2008; 29(6): 1043–49
9. Bartynski WS, Boardman JF, Zeigler ZR et al: Posterior reversible encephalopathy syndrome in infection, sepsis, and shock. *Am J Neuroradiol*, 2006; 27(10): 2179–90
10. Bartynski WS: Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. *Am J Neuroradiol*, 2008; 29(6): 1036–42
11. Bartynski WS, Tan HP, Boardman JF et al: Posterior reversible encephalopathy syndrome after solid organ transplantation. *Am J Neuroradiol*, 2008; 29(5): 924–30
12. Masetti R, Cordelli DM, Zama D et al: PRES in children undergoing hematopoietic stem cell or solid organ transplantation. *Pediatrics*, 2015; 135(5): 890–901
13. Wu Q, Marescaux C, Wolff V et al: Tacrolimus-associated posterior reversible encephalopathy syndrome after solid organ transplantation. *Eur Neurol*, 2010; 64(3): 169–77
14. Mayama M, Uno K, Tano S et al: Incidence of posterior reversible encephalopathy syndrome in eclamptic and patients with preeclampsia with neurologic symptoms. *Am J Obstet Gynecol*, 2016; 215(2): 239.e1-5
15. Fugate JE, Claassen DO, Cloft HJ et al: Posterior reversible encephalopathy syndrome: Associated clinical and radiologic findings. *Mayo Clin Proc*, 2010; 85(5): 427–32
16. Dooley MA, Hogan SL: Environmental epidemiology and risk factors for autoimmune disease. *Curr Opin Rheumatol*, 2003; 15(2): 99–103
17. Lassen NA: Regulation of cerebral circulation. *Acta Anaesthesiol Scand Suppl*, 1971; 45: 78–80
18. Meng L, Gelb AW: Regulation of cerebral autoregulation by carbon dioxide. *Anesthesiology*, 2015; 122(1): 196–205
19. Kontos HA, Wei EP, Navari RM et al: Responses of cerebral arteries and arterioles to acute hypotension and hypertension. *Am J Physiol*, 1978; 234(4): H371–83
20. MacKenzie ET, Strandgaard S, Graham DI et al: Effects of acutely induced hypertension in cats on pial arteriolar caliber, local cerebral blood flow, and the blood-brain barrier. *Circ Res*, 1976; 39(1): 33–41
21. Strandgaard S, Olesen J, Skinhoj E, Lassen NA: Autoregulation of brain circulation in severe arterial hypertension. *Br Med J*, 1973; 1(5852): 507–10
22. Lee VH, Wijdicks EF, Manno EM, Rabinstein AA: Clinical spectrum of reversible posterior leukoencephalopathy syndrome. *Arch Neurol*, 2008; 65(2): 205–10
23. Feske SK: Posterior reversible encephalopathy syndrome: A review. *Semin Neurol*, 2011; 31(2): 202–15
24. Sadry P, Sheikh I, Rashid M: Metamorphopsia and other visual distortions associated with the recovery from posterior reversible encephalopathy syndrome (PRES). *Neurology*, 2018; 90(15 Suppl.): P4.342
25. Fernandez A, Lichtshein G, Vieweg W: The Charles Bonnet syndrome: A review. *J Nerv Ment Dis*, 1997; 185(3): 195–200
26. Satoh M, Suzuki K, Miyamura M et al: Metamorphopsia and transient increase in the cerebral blood flow of the left occipital pole on 123I-IMP SPECT: A case report. *Clin Neurol*, 1997; 37: 631–35
27. Schwartz RB, Jones KM, Kalina P et al: Hypertensive encephalopathy: Findings on CT, MR imaging, and SPECT imaging in 14 cases. *Am J Roentgenol*, 1992; 159(2): 379–83
28. Kanata A, Yonenaga E, Tsukamoto H, Katsuta T: A case of hypertensive encephalopathy with extensive lesions throughout the whole brain on MRI. *Nosotchu*, 2013; 35(1): 36–41
29. Singer S, Grommes C, Reiner AS et al: Posterior reversible encephalopathy syndrome in patients with cancer. *Oncologist*, 2015; 20(7): 806–11
30. Chardain A, Mesnage V, Alamowitch S et al: Posterior reversible encephalopathy syndrome (PRES) and hypomagnesemia: A frequent association? *Rev Neurol*, 2016; 172(6–7): 384–88
31. Gao B, Yu BX, Li RS et al: Cytotoxic edema in posterior reversible encephalopathy syndrome: Correlation of MRI features with serum albumin levels. *Am J Neuroradiol*, 2015; 36(10): 1884–89
32. Pirker A, Kramer L, Voller B et al: Type of edema in posterior reversible encephalopathy syndrome depends on serum albumin levels: An MR imaging study in 28 patients. *Am J Neuroradiol*, 2011; 32(3): 527–31
33. de Havenon A, Joos Z, Longenecker L et al: Posterior reversible encephalopathy syndrome with spinal cord involvement. *Neurology*, 2014; 83(22): 2002–6
34. Datar S, Singh TD, Fugate JE et al: Albuminocytologic dissociation in posterior reversible encephalopathy syndrome. *Mayo Clin Proc*, 2015; 90(10): 1366–71
35. Wu Q, Marescaux C, Wolff V et al: Tacrolimus-associated posterior reversible encephalopathy syndrome after solid organ transplantation. *Eur Neurol*, 2010; 64(3): 169–77
36. Mathew J Jr., Koo M, Crook IR, Jahanmir J: An unusual cause of altered mental status: The importance of monitoring a patient’s blood pressure. *BMJ Case Rep*, 2014; 2014: pii: bcr2013203181
37. Kastrop O, Gerwig M, Frings M, Diener HC: Posterior reversible encephalopathy syndrome (PRES): Electroencephalographic findings and seizure patterns. *J Neurol*, 2012; 259(7): 1383–89