"Posterior reversible encephalopathy syndrome", not always typical, not always reversible.

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Learning objectives

Posterior reversible encephalopathy syndrome (PRES) a neuro-radiological syndrome characterized by seizures, altered level of consciousness and visual disturbance. PRES is associated with hyperintense lesions on magnetic resonance imaging (MRI) most commonly seen in the posterior regions. In most cases symptoms and radiological lesions are reversible. The aims of this article are:

The posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity, which can vary widely its presentation, being atypical in some cases.

The clinical diagnosis of PRES includes the presence of headache, seizures, encephalopathy, and visual disturbances, as well as radiologic findings of focal reversible vasogenic edema, best seen on magnetic resonance imaging (MRI) of the brain.

Despite the syndrome's name, lesions in PRES are rarely isolated to the "posterior" parieto-occipital subcortical. White matter on imaging and instead often involve the cortex, frontal lobes, basal ganglia, and brainstem.

Reversible: The clinical presentation and the edema observed in imaging tests completely resolve with treatment.

The clinical spectrum and pathophysiology of PRES remains poorly defined. Complications, such as ischemic infarction or intracranial hemorrhage can cause substantial morbidity and mortality. This and the atypical imaging forms of presentation can make the diagnosis of PRES challenging and may delay the onset of treatment.

The underlying pathophysiology of PRES remains elusive. Several theories have been proposed, the most widely accepted states that rapidly developing hypertension leads to a breakdown in cerebral autoregulation, particularly in the posterior region of the head, where there is a relative lack of sympathetic innervation.
Background

CLINICAL FEATURES

The clinical presentation of PRES is very broad and includes headaches, confusion, nausea and vomiting, generalized seizures sometimes with status epilepticus, cerebellar syndrome, cortical blindness, hemianopsia, blurred vision, hemiparesis, coma and others. These symptoms may develop over several days or may be recognized only in the acute setting.

Certain clinical conditions have been established as risk factors for the development of PRES (Table 1):

- Hypertension was the first condition who was described as a factor that promotes the development of PRES (hypertensive encephalopathy). It is present in 70 to 80% of cases.

- Eclampsia or pre-eclampsia has always been linked to PRES, even in women with blood pressure within normal limits (20 to 30% of cases). The development of hypertension in preeclampsia is related to systemic vasoconstriction associated with intravascular volume depletion and hemoconcentration and is more frequent in the delayed form in postpartum.

- Also has been seen in chemotherapy regimens and organ transplants that require the use of immunosuppressive drugs especially in allogeneic bone marrow transplantation.

- Severe Sepsis and multiple organ failure.

- Chronic renal failure and dialysis.

- Autoimmune diseases such as lupus erythematosus, scleroderma, Wegener's granulomatosis, and others. The syndrome has developed even in periods when the patients were not being treated with immunosuppressive drugs.

IMAGING FEATURES
PRES CT and MR images are characterised by abnormalities of the white matter and the grey matter, predominantly affecting the posterior regions.

The parietal and occipital lobes are most commonly affected, followed by the frontal lobes, the inferior temporal-occipital junction, and the cerebellum. A bilateral and symmetrical appearance is highly typical although lesions can be asymmetrical in some cases.

On CT we can observe diffuse hypodense areas that indicate the affected regions. On MRI, patchy confluent cortical/subcortical lesions appear iso-intense or low signal intensity on T1-weighted images and hyperintense on T2-weighted images and in FLAIR sequence, distributed in superficial and deep border territories.

Enhancement is not usually seen after injection of a contrast agent.

There are three variants of hemispheric patterns:
1. Holohemispheric: Vasogenic edema affecting all lobes, separating the medial vascular regions of the anterior cerebral artery and posterior cerebral artery from the lateral vascular regions of the middle cerebral artery.
2. Superior frontal sulcal.
3. Primary parietal-occipital.
Partial, asymmetric, or mixed forms of these patterns may be encountered.

ATIPICAL IMAGING FEATURES

Some atypical findings on CT and MR have been described, including extension of lesions to the basal ganglia and the brain stem and the deep white matter, in particular the splenium of the corpus callosum.
In some cases the condition may be unilateral which requires a high level of suspicion.

Severe cases can cause progressive dysfunction of cerebrovascular regulatory mechanisms altering the permeability of the blood-brain barrier.

In these cases MRI with gadolinium injection shows enhancement on T1-weighted images.

We can also find diffusion restriction, infarction, cytotoxic edema and hemorrhage that may manifest as focal hematoma or subarachnoid hemorrhage.
<table>
<thead>
<tr>
<th>Clinical conditions associated with PRES</th>
<th>(Part 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Toxemia in pregnancy:</td>
<td>Preeclampsia</td>
</tr>
<tr>
<td></td>
<td>Eclampsia</td>
</tr>
<tr>
<td>2 Post-transplant:</td>
<td>Bone marrow</td>
</tr>
<tr>
<td></td>
<td>Solid organ (infection or rejection)</td>
</tr>
<tr>
<td>3 Immunosuppression:</td>
<td>Cyclosporine,</td>
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<tr>
<td></td>
<td>Tacrolimus</td>
</tr>
<tr>
<td>4 Infection / sepsis / shock:</td>
<td>Systemic inflammatory response syndrome</td>
</tr>
<tr>
<td></td>
<td>Multiple organ failure syndrome</td>
</tr>
<tr>
<td>5 Autoimmune diseases:</td>
<td>SLE (systemic lupus erythematosus syndrome)</td>
</tr>
<tr>
<td></td>
<td>Scleroderma</td>
</tr>
<tr>
<td></td>
<td>Wegener's granulomatosis</td>
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<tr>
<td></td>
<td>Polyarteritis nodosa</td>
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</table>

**Table 1:** Table 1: Part 1. Clinical condition associated with PRES

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<table>
<thead>
<tr>
<th>Clinical conditions associated with PRES</th>
<th>(Part 2)</th>
</tr>
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<tbody>
<tr>
<td>6 Cancer Chemotherapy:</td>
<td>high-dose combination chemotherapy</td>
</tr>
<tr>
<td>single substances: cytarabine, cisplatin, gemcitabine, tiazofurin, bevacizumab (Avastin), the kinase inhibitor BAY 34-9006</td>
<td></td>
</tr>
<tr>
<td>7 Miscellaneous</td>
<td>Hypomagnesemia, hypercalcemia, hypercholesterolemia</td>
</tr>
<tr>
<td></td>
<td>Thrombotic thrombocytopenic purpura</td>
</tr>
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<td></td>
<td>Blood transfusion</td>
</tr>
<tr>
<td></td>
<td>IV immunoglobulin</td>
</tr>
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<td></td>
<td>Guillain-Barré Syndrome</td>
</tr>
<tr>
<td></td>
<td>Ephedra Overdose</td>
</tr>
<tr>
<td></td>
<td>Dialysis / erythropoietin</td>
</tr>
<tr>
<td></td>
<td>Triple-H therapy</td>
</tr>
<tr>
<td></td>
<td>Tumor lysis syndrome</td>
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<td></td>
<td>Hydrogen peroxide</td>
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</tbody>
</table>

**Table 5:** Table 1: Part 2. Clinical condition associated with PRES

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Imaging findings OR Procedure details

• Patient 1

29 year old woman, without known risk factors. She was in her 30th week of pregnancy, transferred from surgery after an urgent cesarean for fetal distress, probably in the context of eclampsia, with arterial blood pressure levels of 145/96 and presenting with generalized seizures.

The first imaging test was a CT that showed a subcortical white matter hypodensity in the parasagittal left parietal lobe with petechial hemorrhage within the lesion. An MRI was performed one day later that showed vasogenic edema (hyperintensity on T2 and FLAIR with facilitated diffusion) except for a posterior region of restricted diffusion (low ADC values) that proved to be an infarct. Notice the hemosiderin deposits on the T2* sequence due to the petechial hemorrhage (red arrow).

Fig 2: Follow-up MRI performed 2 months later shows gliosis and malacia as infarct sequelae (yellow arrow).

As atypical imaging findings in this case we have: ischemic infarction (restricted diffusion), asymmetric almost unilateral distribution, and petechial hemorrhage (Images 1, 2).

• Patient 2

54 year old woman, smoker, with medical history of chronic hypertension and a non-small cell lung carcinoma, in her second cycle of chemotherapy, including cisplatin. The patient consulted for acute visual impairment, initially associated with headache and left homonymous hemianopia, later accompanied by dizziness of sudden onset and progressive visual impairment that progressed to complete cortical blindness in a period of hours. There were no other neurological, motor, sensory or language alterations.

CT images showed hypodense cortico-subcortical lesions in parasagittal occipital regions and a similar lesion in superior right cerebellum, suggesting ischemic lesions in the vertebrobasilar system. CT angiography was completely normal.

MRI performed shortly after the CT scan, shows bilateral occipital cortico-subcortical lesions that appear hyperintense on T2- weighted images and FLAIR, hyperintense on diffusion and thin gyral enhancement on contrast- enhanced T1-weighted images. A focal lesion of the same characteristics was seen in the right superior cerebellum. These lesions were again highly suggestive of ischemic lesions in the vertebrobasilar system (Image 3).
MRI control was performed 9 months later, showing malacia and gliosis in parasagittal occipital regions and focal malacia in right superior cerebellum, that confirm the ischemic infarcts. (Image 4).

Given that a thrombus was not found in vascular studies and that the patient was on cisplatin at the onset of symptoms, cisplatin toxicity was assumed and the drug was removed. This case illustrates a doubtful severe case of PRES with the atypical finding of extense infarction. The real cause was never known.

- **Patient 3**

49 year old woman, smoker, with a history of hypertension and long-standing diabetes mellitus (DM).
Consults for a 72 hours clinical picture consisting of pulsatile headache, phonophobia and photophobia, starting after epidural catheter placement for anesthesia. The patient underwent a lumbar puncture at the emergency department, after which rapidly developed bilateral blindness.

CT performed at the emergency department showed bilateral parieto-occipital subcortical hypodensities, highly suggestive of PRES.

MRI one day later a typical distribution of the lesions (holo-hemispheric pattern), consisting in cortico-subcortical bilateral hyperintense lesions on long TR sequences, suggestive of vasogenic edema, predominantly in the posterior vascular territory (superior cerebellar hemispheres, occipital and parietal lobes and right thalamus), as well as in superficial water-shed territory (in the left temporal and bilateral frontal lobes) and deep water-shed territory.

Two of the lesions (left parieto-occipital and left occipital) show hyperintensity on diffusion weighted image (blue arrow), (Image 5).

The hypertension is corrected and the patient clinically improves, being asymptomatic at discharge.

Three weeks later. Control MRI (Image 6), radiological improvement with mild residual subcortical white matter vasogenic edema in occipital lobes. Notice scarce focal lesions of gliosis in deep border territories, and a small focus of peripheric malacia in the left occipital lobe, suggesting sequelae of small infarctions (blue arrow).

- **Patient 4**
46 year-old woman, heavy smoker, hospitalized for 48 hours for dyspnea of one month evolution a lymphoproliferative disorder. The preset presents with cardio respiratory arrest following generalized seizures. During admission presents new convulsive episodes, dysplopia and low level of consciousness.

CT showed characteristic digitiform hypodensities of bilateral fronto-parietal subcortical white matter, mainly on the left and mild cortical involvement without mass effect or enhancement after intravenous contrast administration. MRI showed a typical pattern of symmetrical holohemispheric vasogenic edema. Notice that right thalamus and left internal capsule are also involved. (Image 7).

Final diagnosis was oat cell carcinoma with severe bone marrow infiltration. Severe anemia and thrombopenia associated, that precluded chemotherapy. The patient presented multiorgan failure and died 16 days after admission. The possible causes of PRES are a combination of leucoeritroblastic syndrome and severe thrombopenia, with hypertension.

• Patient 5

51 year-old woman, smoker, who presents medical history of chronic hypertension and stage IV lung cancer diagnosed 1 month ago, currently treated with the first cycle of cisplatin, started 2 weeks ago. Who attends to the emergency department with clinical symptoms that include left hemiparesis, left central facial paresis and dysarthria.

MRI imaging shows an acute infarct in the right medial cerebral artery territory (MCA) (yellow arrow) and left hemispheric subcortical vasogenic edema in frontier territory of ACA-PCA/MCA (blue arrow) (Holohemispheric left asymmetric pattern).

This is an atypical case of left holohemispheric pattern of PRES with a right MCA ischemic infarct. (Images 8 and 9). The patient died 15 days after clinical manifestations onset.

• Patient 6

32 year-old woman, 24 weeks pregnancy. Ex-smoker with a history of nephrotic syndrome, renal function currently within normal limits. Presents with severe headache and blurred vision, which evolves within hours to bilateral blindness.
Urgent cesarean is performed because of severe preeclampsia. After treatment, the patient shows complete recovery of vision, although three medications were needed for arterial pressure control.

MRI shows bilateral symmetric holohemispheric pattern of vasogenic edema, with scattered areas of facilitated diffusion. Notice the involvement of other structures less frequently described in PRES, such as splenium of corpus callosum, basal ganglia and bilateral corona radiata (Image 10).

The patient shows complete recovery both clinically and on image studies after treatment, (Image 11).

This case is typical both in clinical and imaging presentation and evolution. Even in this case, some atypical anatomical structures were involved along with the typical lesions.

• Patient 7

53-year-old woman, smoker, with long standing hypertension, who presents at the emergency department with a hypertensive emergency with headache and seizures.

The MRI showed characteristic diffuse, bilateral, posterior lesions (primary parieto-occipital pattern) with bilateral deep water-shed zones and posterior limb of internal capsule involvement, both after treatment, the patient showed complete recovery of her clinical picture (Image 12).

• Patient 8

Pregnant 30 year-old woman with preclampsia, who had undergone a cesarean with epidural anesthesia 2 weeks before admission, presents at the emergency department with orbital and hemicranial headache, sensory and motor deficit with progressive impairment and tonic-clonic seizures.

CT showed bifrontal subarachnoid hemorrhage and left frontal hypodensity. Heparin was started because of venous thrombosis suspicion.

The initial MRI showed supratentorially, numerous cortico-subcortical bilateral vasogenic edema lesions in occipital, parietal and frontal lobes. Cerebellum was also involved (not shown). In T1 and FLAIR small hyperintensities are observed in the left frontal convexity.
suggesting petechial bleeding or SAH (blue arrow). T2* sequence showed hemosiderin subarachnoid deposits in the same localization.
The neurological signs and MR alterations quickly disappeared after anti-hypertensive treatment (3 weeks later).
DSA and venous MR angiography were normal, (Image 13).

No sequelae in imaging control two months later (Image 14).

• Patient 9

51 year-old female smoker, who presented a 5 day clinical picture of dizziness, unsteadiness and asthenia associated with elevated blood pressure values (SBP 170 mmHg). The patient consulted the emergency department for appearance of dysarthria and central facial paralysis.

Initial MR shows acute ischemic infarct in pons (blue arrow), and bilateral occipital and posterior temporal subcortical vasogenic edema that reverses in follow-up imaging, suggesting PRES.
Notice the focal hyperintensities in posterior periventricular white matter suggesting ischemic lesions from small vessel disease, (Image 15).

Control MRI shows disappearance of the temporo-occipital lesions a chronic lacunar infarction in pons and periventricular white matter lesions of small vessel disease without change, (image 16).

• Patient 10

47 year-old woman, with newly diagnosed hypertension, who comes to the emergency room presenting a clinical picture of muscle and joint pain, paresthesia and asthenia in all extremities with abnormal gait and increased support base.

Laboratory analysis performed at the emergency department shows important leukocytosis with eosinophilia, hematuria and proteinuria.

In initial MRI shows cortico-subcortical, bilateral occipital lesions, bilateral internal watershed lesions and left cerebellar lesion, all with restricted diffusion and patchy enhancement with intravenous contrast agent, (Image 17) compatible with acute/subacute ischemic lesions in border territory, predominantly in posterior territory.
MR angiography was also performed, in which there were no pathological findings.

The patient was diagnosed of Churg-Strauss vasculitis. She improved of her systemic clinical picture after steroid and anticoagulant therapy and was discharged without an imaging control.

This case represents either a case of atypical vasculitis (in border zones), cardiac embolus or atypical PRES. The confirmation was not possible.

**Patient 11**

22 year-old male without known pathological antecedents, who is brought to the emergency room for sudden loss of consciousness. On arrival, the patient presented with bilateral unresponsive miosis, visual deviation to the left, slight vertical nystagmus and unresponsive to painful stimuli. Tracheal intubation was performed.

CT is performed as the initial image study showing no pathological findings (not shown). Another CT scan is done 24 hours later (not shown), that showed multiple patchy cortico-subcortical hypodensities predominantly in occipital, parietal / temporal lobes and also in thalamus and cerebellar hemispheres. No enhancement is seen after administration of CIV.

CT angiography shows no abnormalities.

MRI (image 18) shows multiple acute/early subacute ischemic lesions (restricted diffusion), in occipital and temporal lobes, superior cerebelar hemispheres and thalami (posterior circulation) Notice some milimetric lesions in basal ganglia

Control CT One week later (image 18) shows hemorrhagic transformation of the infarcts localized in parietal and occipital lobes.

So we do not have control images control as the patient is transferred to another hospital. The last news we have is that the patient clinically improved.

This atypical case couldn’t be confirmed. Differential diagnosis was between top of basilar thrombosis (not evidenced on CT angiography 24 hours later to admission) or severe case of PRES.

**RESULTS**
In our 11 cases, 10 were female patients with a mean age of 45 years.

In 9 of the 11 cases, the diagnosis of PRES was confirmed either with clinical evolution imaging controls.

No clarification of the cause for the lesions seen in imaging studies was possible in the case of Churg-Strauss syndrome.

Arterial hypertension was the prevalent risk factor in our series, in accordance to the literature. Other triggering factors were eclampsia, cisplatin toxicity or renal failure, (Table 2).

We had infrequent risk factors like multiple organ failure and a rare cause (lumbar puncture).

All 11 cases had initial neuroimaging studies, with either MRI or computed tomography (CT) of the head.

All patients showed parieto-occipital involvement on imaging studies, in accordance to the literature, (Table 3).

Lesions were symmetric in half of the cases, unilateral in 1 case and asymmetric in the rest of the cases.

All except one case had atypical imaging findings, including basal ganglia or pons involvement, or the presence of one or more severity signs such as enhancement after intravenous contrast administration, diffusion restriction and hemorrhage, (Table 4).

Half of the patients had severe forms of the disease, either because the presence of hemorrhage or sequelae, (Table 4).

The most common clinical presentation was headache, visual disturbances and seizures, (Table 2).
Fig. 1: Image 1: September 7th (patient 1)

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**Fig. 2:** Image 2: November 10th, Patient 1. Control

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**Fig. 3:** Image 3: Patient 2

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**Fig. 4:** Image 4: Patient 2. Control

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**Fig. 5:** Image 5: Patient 3

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**Fig. 6:** Image 6: Patient 3. Control

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Fig. 7: Image 7: patient 4

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Fig. 8: Image 8: Patient 5. CBF= cerebral blood flow

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Fig. 9: Image 9: Patient 5 CBF= cerebral blood flow.
**Fig. 10:** Image 10: Patient 6

**Fig. 11:** Image 11: Patient 6. Control
Fig. 12: Image 12: Patient 7

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**Fig. 13:** Image 13: Patient 8 DSA= digital subtraction angiography.

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**Fig. 14:** Image 14: Patient 8 control

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**Fig. 15:** Image 15: Patient 9

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**Fig. 16:** Image 16: patient 9. Control

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**Fig. 17:** Image 17: Patient 10

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Fig. 18: Image 18. Patient 11  

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<table>
<thead>
<tr>
<th></th>
<th>AT</th>
<th>Clinical findings</th>
<th>Trigger</th>
<th>Test</th>
<th>Evolution after treatment</th>
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<tbody>
<tr>
<td>1</td>
<td>145/96</td>
<td>Seizures</td>
<td>Eclampsia</td>
<td>CT, MR</td>
<td>Small parietal infarction, with complete recovery</td>
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<td>2</td>
<td>*</td>
<td>Headache, blindness</td>
<td>Treatment with cisplatin</td>
<td>CT, MR</td>
<td>Complete recovery</td>
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<tr>
<td>3</td>
<td>200/100</td>
<td>Headache, blindness</td>
<td>Epidural anesthesia</td>
<td>CT, MR</td>
<td>Small infarction, with complete recovery</td>
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<td>180/100</td>
<td>Status epilepticus, diplopia, low level of consciousness</td>
<td>Multiorgan failure</td>
<td>CT, MR</td>
<td>Death after 16 days</td>
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<tr>
<td>5</td>
<td>160/100</td>
<td>Dysarthria, facial palsy, right hemiparesis</td>
<td>Treatment with cisplatin</td>
<td>MR</td>
<td>Death after 15 days</td>
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<td>6</td>
<td>175/88</td>
<td>Headache, blindness, low level of consciousness</td>
<td>Severe preeclampsia</td>
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<td>Complete recovery</td>
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<td>7</td>
<td>172/104</td>
<td>Headache, seizures</td>
<td>Hypertension-renal failure</td>
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<td>Complete recovery</td>
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<td>8</td>
<td>*</td>
<td>Status epilepticus, low level of consciousness</td>
<td>Puerperal eclampsia</td>
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<td>9</td>
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<td>Chronic headache, dizziness, unsteadiness, neurological focality</td>
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<td>CT, MR</td>
<td>Resolution of vasogenic edema, Pons infarction</td>
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<td>sudden loss of consciousness, unresponsive miosis</td>
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<td>Clinical improvement</td>
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**Table 2:** (Table 2) Clinical Findings *= not available in medical records

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<thead>
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<th>Pattern</th>
<th>PO</th>
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<th>Cerebellum</th>
<th>BG</th>
<th>Po ns</th>
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<tr>
<td>1</td>
<td>PO</td>
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<td>No</td>
<td>No</td>
<td>No</td>
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<td>2</td>
<td>PO</td>
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<tr>
<td>3</td>
<td>HH</td>
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<td>Yes</td>
<td>Yes (bilateral)</td>
<td>No</td>
<td>No</td>
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<tr>
<td>4</td>
<td>HH</td>
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<tr>
<td>11</td>
<td>PO</td>
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<td>No</td>
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**Table 3:** Table 3: hemispheric patterns -PO=primary parieto-occipital, HH=holohemispheric, symmetric asymmetric

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**Table 4:** (Table 4) RESULTS: According to imaging findings. -?= information not available at clinical history or sequence not performed -SAH= subarachnoid hemorrhage

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<table>
<thead>
<tr>
<th>P</th>
<th>Atypical signs</th>
<th>Hyper-intense on diffusion</th>
<th>Low ADC</th>
<th>Enhancement</th>
<th>Hemorrhage</th>
<th>Sequelae</th>
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<tbody>
<tr>
<td>1</td>
<td>Infarction</td>
<td>Yes</td>
<td>Yes</td>
<td>?</td>
<td>Petechial</td>
<td>Malacia and hemosiderin deposits for small parietal infarct</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>Cerebellum</td>
<td>Yes</td>
<td>?</td>
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<td>Bilateral occipital infarcts</td>
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Conclusion

-Apart from the known risk factors, such as hypertension, immunosuppressants, and renal failure of various etiologies may be the precipitants of PRES.

- Early recognition of PRES is important for early therapy start. The patients must have clinical history of acute neurologic symptoms, such as headache, visual disturbance, seizures or neurological deficit; brain imaging findings such as focal vasogenic edema and clinical or radiologic proof of reversibility.

-A large proportion of our patients with PRES present with atypical neuroimaging findings. In accordance to recent articles published in the literature. Thus it is important for radiologists to be familiar with both the typical and atypical imaging features of PRES.

-The appearance of severe cases of PRES is not uncommon in clinical practice, being difficult to confirm the diagnosis in these situations, because these patients have complex medical histories, with more than one possible cause of the imaging findings.

-The diagnosis is not always straightforward and typical imaging features require correlation with clinical and laboratory data for accurate assessment. We found that it may not be easy to find a clear clinical-radiological association even in typical reversible cases.

-Thalamus, midbrain, and pons affection was significantly less frequent in preeclampsia-eclampsia associated PRES \( (P=0.01) \). Preeclampsia-eclampsia patients had significantly less severe edema, less cytotoxic edema, hemorrhage and contrast enhancement, while more frequent complete resolution of edema and less frequent residual structural lesions were seen on follow-up imaging.

-We found an involvement of the basal ganglia, brainstem, or cerebellum in about one-third of cases studied. But, such an occurrence never appeared isolated (Fig 1). The main MRI lesions are located particularly in the parieto-occipital regions (90-95%).

-All our patients showed a bilateral affection, frequently asymmetrically. This suggests that PRES shows a typical MRI pattern on fluid-attenuated inversion recovery (FLAIR)-weighted imaging in PRES patients that makes the following diagnoses more likely:

  - The predominant affected region is parieto-occipital.
  - Both hemispheres are affected, sometimes asymmetrically.
  - The subcortical white matter is always affected, but an involvement of cortex is also very common.
- Differential diagnosis considered in the most challenging cases and couldn't be confirmed, were ischemic lesions either in vasculitis, or emboli in vertebrobasilar system or emboli in border zones.
References


