POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME:
MAGNETIC RESONANCE IMAGING AND
DIFFUSION-WEIGHTED IMAGING IN 12 CASES

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Posterior reversible encephalopathy syndrome (PRES) is a potentially devastating neurologic syndrome, but timely treatment may lead to complete reversal of the disease course. We reviewed 12 cases of PRES and describe the clinical history and imaging findings, including conventional magnetic resonance imaging (MRI), diffusion-weighted imaging (DWI), and calculated apparent diffusion coefficient (ADC) maps, used to establish the diagnosis of PRES. Three male and nine female patients aged between 11 and 70 years (mean, 37 years) with clinical and imaging findings consistent with PRES were enrolled in the study. All patients had undergone conventional MRI and 10 had undergone additional DWI studies. Ten patients had follow-up MRI studies. DWI was performed using a 1.5T system with a single-shot spin-echo echo-planar pulse sequence. Initial and follow-up neuroimaging and clinical history were reviewed. Lesions were almost always present over the posterior circulation, mainly the parieto-occipital region, affecting primarily the white matter. The anterior circulation region, brainstem, cerebellum, deep cerebral white matter, and thalamus were also involved in five cases. Conventional MRI revealed hyperintensity on T2-weighted and fluid-attenuated inversion recovery images. DWI showed isointensity and increased signal intensity on ADC values in all cases, indicating vasogenic edema. Clinical and MRI follow-up showed that the symptoms and radiologic abnormalities could be reversed after appropriate treatment of the causes of PRES in most patients (9 of 10). In one patient, the ADC value was lower on follow-up images, indicating cytotoxic edema with ischemic infarct. DWI was a useful complement to MRI in the diagnosis of PRES.

Key Words: posterior reversible encephalopathy syndrome, hypertensive encephalopathy, magnetic resonance imaging, diffusion-weighted imaging (Kaohsiung J Med Sci 2004;20:381–8)

Posterior reversible encephalopathy syndrome (PRES) is a potentially devastating neurologic syndrome characterized by rapidly progressive signs and symptoms, including headache, seizures, consciousness disturbance, and/or visual disturbances. The imaging abnormalities of PRES are predominantly in bilateral posterior circulation regions, mainly in parieto-occipital areas. When promptly recognized and treated, the symptoms and radiologic abnormalities can be reversed. When unrecognized, the patient’s condition can progress to ischemia, massive infarction, and death [1–3]. Alternative terms such as hypertensive encephalopathy, reversible posterior cerebral edema syndrome, and posterior reversible leukoencephalopathy are also used to describe this group of disorders [2]. A variety of causes such as preeclampsia/eclampsia, uremia, systemic lupus erythematosus (SLE), and immunosuppressant therapy (e.g. cyclosporine, cisplatin, or tacrolimus) have been associated with this syndrome [1–3]. Early diagnosis of PRES is im-
important for clinical management, so we reviewed 12 cases of PRES and describe the clinical history and imaging findings, including conventional magnetic resonance imaging (MRI), diffusion-weighted imaging (DWI), and calculated apparent diffusion coefficient (ADC) maps.

**Patients and Methods**

From January 1999 to January 2003, 12 consecutive patients (3 male and 9 female; mean age, 37 years; range, 11–70 years) with clinical symptoms and MRI findings consistent with PRES were included in the study. All patients underwent conventional MRI, and 10 patients also had DWI studies. Ten patients had follow-up MRI studies. Clinical history including symptoms, medical illness or medication, highest blood pressure, and patient outcome were reviewed. Although elevated blood pressure is a common clinical finding in PRES, some patients had normal or slightly elevated blood pressure (compared with the average blood pressure of the normal age-related population) (Cases 3 and 6). The underlying causes of PRES in the 12 patients included SLE (6), renal diseases (5), and eclampsia (1).

Routine imaging studies included axial T1-weighted spin-echo (500/30/2 repetition time/echo time/excitations), T2-weighted fast spin-echo (4,000/100/2) with echo train length 8, and fast fluid-attenuated inversion recovery (FLAIR; 9,000/2,200/133/1 repetition time/inversion time/echo time/number of excitations) sequences. Post-contrast gadopentetate (0.1 mmol/kg) T1-weighted images were also acquired. Sections (5 mm thick) with 2.5 mm interslice gaps, 24 cm field of view, and 256 × 192 matrix were used for all scans.

The imaging sequence for DWI was single-shot spin-echo echo-planar imaging (10,000/93 repetition time/echo time) with diffusion sensitivities $b = 0$ and $b = 1,000 \text{ s/mm}^2$. The diffusion gradients were applied sequentially in three orthogonal directions to generate three sets of axial DW images. Sections (5 mm thick) with 2.5 mm interslice gaps, 24 cm field of view, and 128 × 256 matrix were used for all scans. The scan time was 40 seconds. A composite isotropic trace image was made by multiplying the three DW images together and taking the cubic root of the result to remove the effects of diffusion anisotropy. Interpretations were made using three sets of DW images and the composite isotropic trace image.

Analysis of diffusion changes was performed by calculating the ADC, based on the Stejskal and Tanner equation [4], as the negative slope of the linear regression line best fitting the points for $b$ versus ln(SI), where SI is the signal intensity from a region of interest of the images acquired at the two $b$ values. ADC maps were generated by performing this calculation on a pixel-by-pixel basis. The results of the DWI accompanied by ADC maps were compared with those of conventional MRI.

**Results**

Clinical manifestations and outcome are summarized in the Table. Clinical symptoms included headache, seizure, consciousness disturbance, and/or visual disturbance. Most patients (11 of 12) presented with seizure and/or consciousness disturbance. In general, the goal of therapy for PRES was prompt reduction of blood pressure. An arterial line was placed as soon as possible to confirm the cuff readings and to guide therapy. Sodium nitroprusside was the drug of choice. Effective alternatives included labetalol, diazoxide, and nifedipine. Magnesium sulfate was used for eclampsia, and phenytoin or diazepam was sometimes required for seizure management. SLE was treated using corticosteroids, and renal failure was treated with hemodialysis. The neurologic abnormalities mostly resolved within a few weeks after appropriate treatment of the causes of PRES and antihypertensive therapy.

The Table also shows the imaging findings. PRES lesions were typically located in the territories of the posterior circulation, mainly in the parieto-occipital and posterior temporal regions, with white-matter predominance (Figures 1 and 2). Symmetrical or slightly asymmetrical distribution of lesions over the bilateral hemispheres was found in most cases, except in one patient who had a lesion mainly over the right cerebral hemisphere (Case 1). Frontal involvement of the anterior circulation region was also noted in four patients (Cases 4, 5, 10, and 11). Atypical lesions in the brainstem (Cases 2 and 11), cerebellum (Cases 10 and 11), and thalamus and central white matter (Case 2) were also identified (Figure 3). An initial diagnosis of a demyelinating disorder was made in Case 2 (hemolytic uremic syndrome). The patient received antihypertensive and anticonvulsant treatment, and hemodialysis. Two weeks later, blood pressure and serum creatinine returned to baseline values and consciousness cleared. Near-total resolution of the lesions was noted at follow-up MRI 4 months later.

Conventional MRI revealed high signal intensity on T2-weighted and FLAIR images of lesion sites. No enhancement was seen after administration of contrast. DWI showed
### Table. Clinical information and imaging findings in 12 patients with posterior reversible encephalopathy syndrome

<table>
<thead>
<tr>
<th>Case/ Age (yr)/ Sex</th>
<th>Cause</th>
<th>Treatment</th>
<th>Symptoms</th>
<th>Highest BP (mmHg)</th>
<th>Initial MRI</th>
<th>Signal intensity on DWI</th>
<th>Signal intensity on ADC map</th>
<th>Follow-up interval</th>
<th>Follow-up MRI</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/23/M</td>
<td>Nephrotic syndrome</td>
<td>Antihypertensive, anticonvulsant, hemodialysis</td>
<td>Seizure, visual disturbance</td>
<td>194/120</td>
<td>Occipital (Rt), post. parietal (Rt), post. temporal (Bil), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>22 d</td>
<td>Total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>2/56/M</td>
<td>HUS</td>
<td>Antihypertensive, anticonvulsant, hemodialysis</td>
<td>Consciousness disturbance, seizure</td>
<td>203/112</td>
<td>Deep WM (Bil), thalamus (Bil), pons, WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>4 mo</td>
<td>Nearly total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>3/44/F</td>
<td>Eclampsia</td>
<td>Antihypertensive, anticonvulsant</td>
<td>Seizure</td>
<td>160/90</td>
<td>Occipital (Bil), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>10 d</td>
<td>Total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>4/35/F</td>
<td>SLE</td>
<td>Antihypertensive, corticosteroid</td>
<td>Headache</td>
<td>192/110</td>
<td>Occipital (Bil), post. parietal (Bil), frontal (Bil), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>20 d</td>
<td>Total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>5/11/M</td>
<td>IgM nephropathy</td>
<td>Antihypertensive, anticonvulsant, corticosteroid</td>
<td>Seizure</td>
<td>180/120</td>
<td>Occipital (Bil), parietal (Bil), frontal (Bil), WM &gt; GM</td>
<td>16 d</td>
<td>Total resolution</td>
<td>Recovery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6/26/F</td>
<td>SLE</td>
<td>Anticonvulsant, corticosteroid</td>
<td>Seizure</td>
<td>152/65</td>
<td>Occipital (Bil Rt &gt; Lt), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>2 mo</td>
<td>Total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>7/41/F</td>
<td>SLE</td>
<td>Antihypertensive, corticosteroid</td>
<td>Consciousness disturbance</td>
<td>200/110</td>
<td>Occipital (Bil Lt &gt; Rt), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>1.5 mo</td>
<td>Total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>8/57/F</td>
<td>Uremia</td>
<td>Antihypertensive, anticonvulsant, hemodialysis</td>
<td>Seizure, headache</td>
<td>200/105</td>
<td>Occipital (Bil), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>2 mo</td>
<td>Total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>9/18/F</td>
<td>SLE</td>
<td>Antihypertensive, anticonvulsant, corticosteroid</td>
<td>Consciousness disturbance, seizure</td>
<td>195/110</td>
<td>Occipital (Bil Rt &gt; Lt), post. parietal (Bil Lt &gt; Rt), WM &gt; GM</td>
<td>2 mo</td>
<td>Total resolution</td>
<td>Recovery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10/70/F</td>
<td>Uremia</td>
<td>Antihypertensive, anticonvulsant, hemodialysis</td>
<td>Consciousness disturbance, seizure</td>
<td>202/89</td>
<td>Occipital (Lt), parietal (Bil), frontal (Bil), cerebellum (Bil Lt &gt; Rt), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>2 wk</td>
<td>Small infarction over Lt occipital</td>
<td>Death</td>
</tr>
<tr>
<td>11/46/F</td>
<td>SLE</td>
<td>Antihypertensive, anticonvulsant, corticosteroid</td>
<td>Seizure, consciousness disturbance</td>
<td>380/113</td>
<td>Occipital (Bil), parietal (Bil), frontal (Bil), brainstem (Bil Lt &gt; Rt), cerebellum (Bil Lt &gt; Rt), genu of corpus callosum, WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td>9 d</td>
<td>Nearly total resolution</td>
<td>Recovery</td>
</tr>
<tr>
<td>12/22/F</td>
<td>SLE</td>
<td>Antihypertensive, anticonvulsant, corticosteroid</td>
<td>Seizure, headache</td>
<td>200/112</td>
<td>Occipital (Bil), parietal (Bil), temporal (Bil), WM &gt; GM</td>
<td>Iso</td>
<td>Hyper</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BP = blood pressure; MRI = magnetic resonance imaging; DWI = diffusion-weighted imaging; ADC = apparent diffusion coefficient; Rt = Right; post. = posterior; Bil = bilateral; WM = white matter; GM = gray matter; Iso = isointense; Hyper = hyperintense; HUS = hemolytic uremic syndrome; SLE = systemic lupus erythematosus; IgM = immunoglobulin M; Lt = Left.
isointense signal intensity and ADC maps revealed high signal intensity, suggesting the possibility of vasogenic edema. Follow-up images showed that most patients (9 of 10) had nearly total or total subsidence of the initial cortical/subcortical changes of the involved brain regions after appropriate treatment of the causes of PRES. Although PRES is considered a reversible encephalopathy, one patient developed high signal intensity on DWI and low

**Figure 1.** Posterior reversible encephalopathy syndrome (PRES) in a patient with systemic lupus erythematosus (Case 12): (A & B) axial fluid-attenuated inversion recovery (FLAIR) magnetic resonance images from different slice locations show symmetric hyperintensity over subcortical white matter of the bilateral occipital, bilateral posterior parietal, and bilateral posterior temporal lobes with some overlying cortical involvement; (C & D) diffusion-weighted imaging in the corresponding locations shows isointense signal to the gray matter; (E & F) apparent diffusion coefficient maps show mildly increased signal intensity in the corresponding areas of abnormal FLAIR, consistent with increased diffusion and vasogenic edema in PRES; (G & H) nearly complete interval resolution of prior abnormal signal at the same locations is noted in follow-up imaging study 9 days later, consistent with reversible vasogenic edema.
Figure 2. Posterior reversible encephalopathy syndrome in a patient with systemic lupus erythematosus (Case 11): (A) axial fluid-attenuated inversion recovery magnetic resonance image shows symmetric hyperintense signal over the bilateral occipital and right frontal subcortical white matter with some overlying gray matter involvement; (B) diffusion-weighted imaging (DWI) shows isointense signal to the gray matter; (C) apparent diffusion coefficient (ADC) map reveals mildly increased signal intensity in similar regions; (D) follow-up imaging 2 weeks later shows a small area of increased intensity on DWI; (E) with lower signal intensity on ADC map over the left occipital lobe (similar location to that in B), indicating irreversible brain injury and progress to ischemic infarction.

Figure 3. Atypical case of posterior reversible encephalopathy syndrome in a patient with hemolytic uremic syndrome (Case 2): (A, B & C) axial fluid-attenuated inversion recovery magnetic resonance images from different slice locations show involvement in bilateral periventricular deep white matter, the bilateral thalami, and the left pons.
signal intensity on ADC map on follow-up imaging study, indicating the development of cytotoxic edema and infarction (Case 11; Figure 2). This patient died due to poor medical condition and septic shock.

**DISCUSSION**

Patients with PRES often have nonlocalizing neurologic symptoms and signs, such as headache, seizure, consciousness disturbance, and/or visual dysfunction. Seizure may be the first neurologic symptom, either a focal attack or generalized event. Altered awareness such as lethargy, somnolence, restlessness, or agitation may also be noted. Headache and abnormalities of visual function such as blurred vision or transient blindness are also frequent complaints [5]. These neurologic abnormalities resolve within a few weeks if the causes of PRES are appropriately treated [1,2,5–7]. The clinical findings are often nonspecific, so the diagnosis may be difficult to establish, particularly in patients who have other illness. Various neurologic conditions such as stroke, intracranial hemorrhage, venous thrombosis, and encephalitis can mimic PRES [2,3,5].

Although markedly elevated blood pressure is noted in most patients at initial presentation, it has been observed that some patients have only mildly elevated or even normal blood pressure (compared with the average blood pressure of the normal population), and such findings were also noted in our study. The well-recognized underlying causes associated with PRES include uremia, hemolytic-uremic syndrome, SLE, and pre-eclampsia/eclampsia. Immunosuppressive medications such as cyclosporine and tacrolimus, and chemotherapeutic agents such as cisplatin, interferon alpha, and intrathecal methotrexate have also been reported in association with PRES [1–3,5–7].

The most common neuroimaging abnormality of PRES is brain edema, mainly in the white matter in the posterior portions of the cerebral hemispheres and especially in the parieto-occipital regions (Figure 1). The edema may extend to the adjoining gray matter [6,7]. The predominantly white-matter distribution of PRES may be explained by the possibility that the progressive edema mainly accumulates in the subcortical white matter, which is relatively loosely organized, and the cortex, where more compaction and tightly organized cells allow less fluid accumulation [7]. Most often, the lesions are symmetrically distributed over bilateral cerebral hemispheres, but asymmetric appearance has also been noted. Conventional MRI shows high signal intensity on T2-weighted and FLAIR images. The lesions usually do not enhance on post-contrast T1-weighted MR images. Few patients with subtle focal enhancement and petechial hemorrhage have been reported, probably due to breakdown of the blood-brain barrier [6]. Involvement of the anterior circulation region, brainstem, cerebellum, deep white-matter region, basal ganglia, and the thalamus have been reported in several studies [2,7–9], and were also identified in our study (Figure 3).

Two theories have been proposed for the pathophysiology of the radiologic abnormalities associated with PRES. The earlier theory postulated that spasm of the cerebral vasculature, in response to sudden elevations of systemic blood pressure, results in ischemic and cytotoxic edema of the brain parenchyma mainly over the borderzone arterial regions [10]. Recently, a more widely accepted hypothesis, based on several DWI and quantitative ADC analysis studies, suggests that increased systemic blood pressure breaks through the upper limit of the autoregulation of the cerebral blood flow, causing brain hyperperfusion and passive distension of the cerebral arterioles, with resultant interstitial extravasation of proteins and fluid, and focal parenchymal hydrostatic edema. This is consistent with the imaging findings of increased ADC values and lack of high signal intensity on DWI, representing increased interstitial fluid as vasogenic edema [6,8,11]. Lesions are isointense on DWI, reflecting the net balance of the combined decreased signal from the vasogenic edema and increased signal from the T2 “shine through” effect. ADC maps show increased signal intensity as a result of increased water diffusibility, distinguishing the changes from infarction, which gives a high signal on DWI and low signal on ADC maps [6,8,11]. The radiologic abnormalities can be reversed when PRES is promptly recognized and treated. However, prolonged vasogenic edema with increased interstitial pressure may be followed by focal tissue ischemia and cytotoxic edema, which presents with low signal on ADC maps due to restricted diffusion. Complicated ischemic infarction may have low signal on ADC maps [2,6,7], as in one of our patients who had a small infarction of the lesion in the follow-up image (Figure 2).

It has been postulated that the predilection for posterior circulation involvement in PRES is due to the uneven sympathetic innervation of cerebral vasculature. The sympathetic nerve from the superior cervical sympathetic ganglion innervates the blood vessels of the pia, with maximal density of innervations in the internal carotid and anterior cerebral territories [3]. The vertebrobasilar circulation has relatively sparse sympathetic innervation. Healthy cerebral autoregulatory mechanisms depend on
both myogenic and neurogenic components to maintain constant blood perfusion of the brain. When the myogenic component is blunted by passive over-distension of vessels due to elevated blood pressure, as in hypertensive encephalopathy, or blunted by direct toxic effects on the endothelium, probably due to uremic encephalopathy or immunosuppressant, the neurogenic response plays a more important role in maintaining constant cerebral flow [3,5–7]. The effectiveness of the neurogenic component is directly proportional to the degree of sympathetic innervation. The posterior circulation is poorly innervated, increasing the risk of vasogenic edema in regions supplied by the vertebrobasilar system [6]. Increased perfusion to abnormal regions has been noted on single photon emission computed tomography imaging [9].

Prompt diagnosis of PRES is of the utmost importance so that initiation of antihypertensive therapy can reverse the MRI lesions. With a delay in diagnosis and treatment, permanent neurologic damage or even death can result from cerebral infarction or hemorrhage [1–3,5]. The diagnosis of PRES might be difficult or be unsuspected in that the blood pressure might not be acutely elevated and the clinical findings are nonspecific and are mimicked by a range of neurologic disorders, including cerebrovascular accident, dural venous sinus thrombosis, demyelination, encephalitis, and “top of the basilar artery” embolism. Dural venous sinus thrombosis is characterized by venous sinus thrombosis associated with venous ischemia and/or hemorrhage, which can be shown by MRI combined with MR angiography. PRES can be differentiated from demyelination and encephalitis by its predominantly symmetric posterior cerebral lesions. DWI is a good tool for differentiation between PRES and acute infarction (especially “top of the basilar artery” infarction) because acute infarction has high signal intensity on DWI and low signal intensity on ADC maps [2,6,7].

A limitation of this study is that no MR angiography (venography) was done to rule out superior sagittal sinus thrombosis. Both PRES and superior sagittal sinus thrombosis associated with venous congestive edema may have similar findings on conventional MRI and DWI.

**Conclusion**

The term PRES describes a syndrome of headaches, seizures, consciousness and/or visual disturbances associated with transient, predominantly posterior cerebral lesions. DWI is a useful complement to MRI in the diagnosis of PRES. Neurologists and neuroradiologists should be aware of the characteristics of PRES because the clinical and neuroradiologic findings can be reversed by timely initiation of appropriate treatment of the underlying causes.

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**References**

後側腦部可逆性腦病變徵候群：
12 位病患之磁振造影及擴散加權影像分析

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為了研究有關後側可逆性腦病變徵候群，我們回顧 12 位有關這類症候群的病患，研究其臨床資料及放射影像，包括傳統磁振造影檢查、擴散加權影像檢查及擴散係數之腦部圖譜，並進而研究擴散加權影像對於後側可逆性腦病變徵候群之診斷所扮演之角色。12 位病患（男性 3 位；女性 9 位），年齡介於 11 至 70 歲（平均年齡為 37 歲），經臨床資料及磁振造影影像診斷為後側可逆性腦病變徵候群，均收集在本次研究中。所有病患均接受傳統磁振造影檢查，其中有 10 位病患同時接受擴散加權磁振造影。10 位病患於後續之磁振造影影像追蹤。此擴散加權磁振造影在 1.5T 之磁振造影儀器下進行，使用 single-shot spin-echo echo-planar 之脈衝序列。有關病人之臨床資料以及初次及追蹤之神經學影像檢查發現，均在此研究中詳加記錄，以作為比較。磁振造影對於後側可逆性腦病變徵候群之發現，通常病灶位於腦部皮質下白質為主之深部循環區域及少數病患之前側深部區、腦幹、小腦、深部白質及視丘皆亦受到侵犯。傳統 T2 加權影像及 FLAIR 加權影像為高診斷表現。擴散加權影像顯示等信號，並且有較高之擴散係數，此代表血管通透性引起之腦水腫。在追蹤過程中，大部份病患經及時適度針刺後側可逆性腦病變徵候群之原因治癒後，臨床症狀及原本病灶的不正常信號會消失。本研究中只有一位病患在追蹤影像中，原本病灶形成細胞毒性水腫之腦梗塞。擴散加權影像可幫助提升傳統磁振造影來診斷後側可逆性腦病變徵候群，是一可行及有價值的輔助診斷檢查工具。

關鍵詞：後側可逆性腦病變徵候群，高血壓腦病變，磁振造影，擴散加權影像

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