Posterior Reversible Encephalopathy Syndrome, Part 1: Fundamental Imaging and Clinical Features

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SUMMARY: Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic state coupled with a unique CT or MR imaging appearance. Recognized in the setting of a number of complex conditions (preeclampsia/eclampsia, allogeneic bone marrow transplantation, organ transplantation, autoimmune disease and high dose chemotherapy) the imaging, clinical and laboratory features of this toxic state are becoming better elucidated. This review summarizes the basic and advanced imaging features of PRES, along with pertinent features of the clinical and laboratory presentation and available histopathology. Many common imaging/clinical/laboratory observations are present among these patients, despite the perception of widely different associated clinical conditions.

Initially recognized in association with eclampsia, cyclosporine after transplantation, and in the setting of severe hypertension, posterior reversible encephalopathy syndrome (PRES) has become synonymous with a unique pattern of brain vasogenic edema seen in the setting of neurotoxicity. On CT or MR imaging studies, the edema is often widespread but predominates in the parietal and occipital regions, leading Hinchey et al to suggest the “posterior” description. A substantial experience with PRES has evolved; and though more widely recognized, controversy still exists as to the mechanism responsible for the brain edema. Specifically, what is the role of hypertension and is the edema related to hyperperfusion or hypoperfusion? In this review, the fundamental clinical and imaging features of PRES will be emphasized with controversial issues to follow.

Imaging Patterns in PRES

At CT/MR imaging, the brain typically demonstrates focal regions of symmetric hemispheric edema (Fig 1A, B). The parietal and occipital lobes are most commonly affected, followed by the frontal lobes, the inferior temporal-occipital junction, and the cerebellum. Lesion confluence may develop as the extent of edema increases. MR diffusion-weighted imaging (DWI) was instrumental in establishing and consistently demonstrating that the areas of abnormality represent vasogenic edema. The edema usually completely reverses.

The basic PRES pattern resembles the brain watershed zones, with the cortex and subcortical and deep white matter involved to varying degrees. Three hemispheric pattern variants may be encountered with similar frequency (holohemispheric [Fig 1], superior frontal sulcus, and primary parietal-occipital). These demarcate lateral hemispheric blood supply (middle cerebral artery [MCA]) and medial hemispheric supply (anterior cerebral artery [ACA], posterior cerebral artery [PCA]) and further reflect the junctional/watershed nature of PRES.

Characteristic lesion locations such as the inferior tempo-occipital junction, superior frontal sulcus, and parietal-occipital region likely represent expression of PRES in the deep (intrahemispheric) watershed. A continuum is noted between diminutive and extensive expression of PRES; and partial, asymmetric, or mixed forms of these patterns may be encountered. Focal/patchy areas of PRES vasogenic edema may also be seen in the basal ganglia, brain stem, and deep white matter (external/internal capsule). When they accompany hemispheric or cerebellar PRES, it is easy to recognize these areas as companion lesions. Present in isolation or when the hemispheric pattern is incompletely expressed (partial/asymmetric), the diagnosis of PRES can be challenging. If cerebellar or brain stem involvement are extensive, hydrocephalus and brain stem compression may occur.

Focal areas of restricted diffusion (likely representing infarction or tissue injury with cytotoxic edema) are uncommon (11%–26%) and may be associated with an adverse outcome. Hemorrhage (focal hematoma, isolated sulcal/subarachnoid blood, or protein) is seen in approximately 15% of patients.

Basic Clinical Features of Neurotoxicity with PRES

Patients at risk for PRES are summarized in the Table. Neurotoxicity with characteristic watershed CT/MR imaging features was initially noted in eclampsia, allogeneic bone marrow transplantation (allo-BMT), solid organ transplantation (SOT), and in association with severe hypertension. With similar clinical/imaging presentation recognized the mid 1990s, additional associations were noted (autoimmune conditions, thrombotic thrombocytopenic purpura, and medical renal disease), and the term “PRES” was introduced. PRES is seen with unique or high-dose cancer chemotherapy (Table) and has recently been associated with infection, sepsis, and shock. Additional considerations have been suggested in numerous case reports as reviewed in the Table.

Clinical/Laboratory Characteristics in PRES

Clinical symptoms at toxicity are broad but include headache, vision change, paresis, hemianopsia, nausea, and altered mentation. Symptoms may develop over several days or may be recognized only in the acute setting. Generalized seizures...
are common and coma may develop. In approximately 70%–80% of patients, moderate-to-severe hypertension is observed. Toxicity blood pressure is normal or only minimally elevated in 20%–30% of patients in eclampsia, allo-BMT, and most large reported PRES series.11,15,17,22,27,39-42

Best studied in preeclampsia/eclampsia, laboratory evidence of endothelial injury is often present with platelet consumption (thrombocytopenia) and evidence of red cell fragmen-
tation (schistocyte formation, increase in lactate dehydrogenase [LDH]).43,44 Developing hypertension in pre-
eclampsia is related to systemic vasoconstriction with accompanying reduced intravascular volume and hemoco-
centration. Renal dysfunction with proteinuria and hypomagnesemia occur; systemic edema develops due to a combination of altered endothelial function and reduced oncotic pressure. Hepatic ischemia may lead to liver dysfunction and, when severe, hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome.

In infection/sepsis/shock-associated PRES, a clinical pattern consistent with systemic inflammatory response syn-
drome develops with evidence of multiple organ dysfunction syndrome (MODS), including alteration of coagulation (thrombocytopenia), liver function (increased bilirubin), renal function (increased creatinine), pulmonary function, and cardiovascular instability.46

Similar features may develop in patients after allo-BMT. The effects of graft-versus-host disease (GVHD) are managed by immune suppression with cyclosporine or tacrolimus (FK-506). Cyclosporine can injure the endothelium.45,46 At toxicity, diffuse endothelial dysfunction is often present, termed “bone marrow transplant thrombotic microangiopathy,” with development of significant schistocyte counts (exceeding 10% when severe) and marked elevation of LDH.16,17,47 A MODS pattern can develop with systemic or pulmonary edema and ischemic hepatic dysfunction, similar to preeclampsia.48

Major PRES-Associated Clinical “Conditions”

Preeclampsia/Eclampsia. The association of PRES with toxemia of pregnancy is well established.1-3,5,21,24,26,49,50 Preeclampsia develops in approximately 5% of pregnancies and eclampsia, in approximately 1 in 3000 births with current management.43,44 Eclampsia develops before gestation in 50% of patients, interpartum in 25%, and within 48 hours of delivery in 25%. Although most women are hypertensive at toxicity, blood pressure is reported as normal or only minimally elevated in 23% of patients.39 The placenta is thought to be the primary cause of toxemia, with placenta removal and fetal delivery considered curative.13,44

“Delayed Eclampsia” (PRES within several weeks after de-
livery) can occur, and the clinical presentation is often confus-
Conditions at risk for PRES

Conditions
Toxemia of pregnancy (preeclampsia/eclampsia)
Posttransplantation:
  allo-BMT
  SOT
Immune suppression:
  Cyclosporine
  Tacrolimus (FK-506)
Infection/sepsis/shock:
  Systemic inflammatory response syndrome
  Multiorgan dysfunction syndrome
Autoimmune diseases:
  Systemic lupus erythematosus
  Systemic sclerosis (scleroderma)
  Wegener’s
  Polyrteritis nodosa
Status-post cancer chemotherapy:
  Combination high-dose chemotherapy
  Reported miscellaneous drugs
  Cytarabine
  Cisplatin
  Gemcitabine
  Tiazofurin
  Bevacizumab (Avastin)
  Kinase inhibitor DAV 34–9006
Miscellaneous reported associations
  Hypomagnesemia
  Hypercalcemia
  Hypocholesterolemia
  Intravenous immunoglobulin
  Guillain-Barré syndrome
  Ephedra overdose
  Dislysis/erythropoietin
  Triple-H therapy
  Tumor lysis syndrome
  Hydrogen peroxide
  Dimethyl sulfoxide stem cells

Blood pressure may be normal or mildly elevated, severe headache is common, and conventional angiography is often performed to exclude intracranial aneurysm. In a recently reported case, delayed eclampsia appeared to have been associated with retained placental fragments. PRES has also been reported 3 weeks following resection and chemotherapy for hydatidiform mole.

Infection/Sepsis/Shock. In infection, sepsis, and shock-associated PRES, gram-positive organisms predominate, and in 40% of patients, toxicity blood pressure is normal or only minimally increased. At imaging, vasogenic edema is greater in normotensive patients and lower in severely hypertensive patients; and at MR angiography (MRA), reversible “vasculopathy” (diffuse/focal vasoconstriction) or vessel pruning is noted. Recent reports note PRES in the setting of post-streptococcal glomerulonephritis, Henoch-Schonlein purpura, and infection-induced hypercoagulable state.

Autoimmune Disease. PRES has been identified in patients with systemic lupus erythematosus, Wegener’s granulomatosis, systemic sclerosis (scleroderma, Fig 2), and polyarteritis nodosa. Detailed accounts of the clinical circumstances surrounding PRES in association with autoimmune disease are infrequent. Patients are commonly managed with intermittent doses of immunosuppression (cyclosporine, cyclophosphamide) for disease control.

Cancer Chemotherapy. PRES is usually encountered after high-dose multidrug cancer therapy, typically in hematopoietic malignancies. A variety of cancer chemotherapeutic drugs have also been noted in association with PRES (Table).
thickening, segmental vessel narrowing, and dissection noted in PRES on MRA.51,109 These CA/MRA features reflect previously described vessel histologic observations. At catheter angiography (CA), diffuse vasoconstriction, focal vasoconstriction, vasodilation, and even a string-of-beads appearance have been noted in PRES, consistent with what is typically described as vasospasm or arteritis (Fig 1C, -D). 2,4,14,51,103,104 Reported blood pressure in most of these patients demonstrated moderate but not severe hypertension (mean arterial pressure <130 mm Hg). CA in PRES is typically performed in eclampsia, delayed eclampsia, and after cancer chemotherapy, usually in the setting of a clinical presentation in which aneurysm is suspected.

At MRA using a 3D time of flight (TOF) technique, patterns resembling vasculopathy have been noted, with vessel irregularity consistent with focal vasoconstriction/vasodilation and diffuse vasoconstriction (Fig 1E). 17,36,60,95,103,105-108 When performed, repeat MRA often demonstrates reversal of the vasculopathy. In normotensive patients, vessels may appear normal or demonstrate pruning of distal intracranial branches, in particular the PCAs. 56

Abnormal internal carotid or vertebral arteries have, on occasion, been noted in PRES, with intimal irregularity resembling fibromuscular disease demonstrated by CA in delayed eclampsia and dissection noted in PRES on MRA.31,103 These CA/MRA features reflect previously described vessel histologic observations. MR venography has tended to be normal in PRES.107

Cerebral Blood Flow in PRES

The state of brain blood flow in PRES remains controversial (Review, Part 2). Vasoconstriction seen by CA in early studies prompted the authors to postulate that reduced brain perfusion led to the imaging features. In contrast, animal studies suggested that experimentally induced hypertension above the autoregulatory limit (mean arterial pressure >150–160 mm Hg) led to hyperperfusion, breakdown of the blood-brain barrier, and hemispheric edema. Hyperperfusion was suggested in a single patient with PRES and Wegener’s in an early study using technetium Tc99m-hexamethylpropyleneamine oxime (Tc99m-HMPAO) single-photon emission CT (SPECT), with a second patient (eclamptic) demonstrating variable radiotracer distribution. In a later study, increased Tc99m-HMPAO SPECT activity suggesting hyperperfusion was reported in a single patient with a molar pregnancy and eclampsia. In patients

Recurrence of PRES

Recurrence of PRES has been anecdotally reported in severe hypertension and after allo-BMT. 66,80 In a recent reported series, recurrent PRES was noted in 3 (3.8%) of 78 patients and was associated with sickle-cell disease with infection, allo-BMT with infection, or atypical autoimmune disease and possible viral infection.57 Recurrent eclampsia is well recognized with a reported incidence of ~2% of live births.96

Histopathology in PRES

Histologic evaluation of PRES is uncommon and often obtained late in the course of complex systemic disease. Biopsy/autopsy obtained during acute toxicity demonstrates vasogenic edema, paralleling observations on DWI. 17,76,97-99 Activated/reactive astrocytes, scattered macrophages, and lymphocytes have been often noted without inflammation, ischemia, or neuronal damage. Late autopsy studies have generally demonstrated evidence of demyelination and myelin pallor along with evidence of ischemia, neuronal anoxic damage, laminar necrosis, or older hemorrhage in the white matter and cortex. 11,13,17,100 Evidence of acute and chronic vessel injury has been described in late autopsy studies with identification of intimal thickening, segmental vessel narrowing, intimal dissection, and organized thrombi.11,101 Acute vasculopathy has also been described in a patient with a liver transplant with vessel inflammation and adjacent deep basal ganglia and periventricular infarction. 102

Advanced Imaging in PRES

Fig 2. The patient is a 38-year-old woman with scleroderma, severe hypertension (190/110 mm Hg), and acute renal failure, with altered mental status that progressed to seizure. A, Axial brain CT image obtained at toxicity demonstrates vasogenic edema in the parietal region bilaterally (curved arrows), consistent with PRES. B, Axial technetiumTc-99m-HMPAO SPECT study performed the following day demonstrates reduced radiopharmaceutical uptake bilaterally in the parietal region (curved arrows), consistent with hyperperfusion.
with aneurysmal subarachnoid hemorrhage and vasospasm, Tc99m-HMPAO studies demonstrated patterns of variable perfusion.\textsuperscript{111} Increased radiopharmaceutical activity in Tc99m-HMPAO brain studies has been noted in many circumstances associated with stroke but remains controversial.\textsuperscript{112}

In contrast, watershed hypoperfusion has been demonstrated by using Tc99m-HMPAO SPECT in a large series of women with eclampsia, with focal hypoperfusion also noted after chemotherapy and in autoimmune disease (Fig 2).\textsuperscript{63,113} Reduced perfusion has also been demonstrated using MR perfusion (MRP) in PRES (Fig 3).\textsuperscript{103,114,115} Cortex and white matter relative cerebral blood volume (rCBV) has been shown to be reduced moderately in areas of PRES (average, 65%), when compared with normal uninvolved regions in 2 studies.\textsuperscript{103,115} Comparing anterior-to-posterior hemispheric flow, Brubaker et al\textsuperscript{114} found that MRP has also demonstrated significant posterior brain hypoperfusion with increased mean transit time, reduced CBV, and reduced cerebral blood flow.\textsuperscript{114} Critical cortex hypoperfusion (12.2 mL/100 g brain per minute) has been demonstrated by stable xenon CT after blood pressure reduction in a child with hypertensive encephalopathy and PRES, which partially reversed with re-established moderate hypertension (mean arterial pressure, 114 mm Hg).\textsuperscript{116}

**Proton MR Spectroscopy in PRES**

Reduced N-acetylaspartate:choline and N-acetylaspartate:creatine ratios have been described by MR spectroscopy in regions of PRES vasogenic edema as well as in unaffected regions.\textsuperscript{107,117,118} Quantitative metabolite assessment in 2 patients demonstrated an absolute reduction of metabolite concentration (considered a dilution effect from vasogenic edema), which corrected in 1 patient on follow-up MR spectroscopy.\textsuperscript{117} Abnormal metabolite ratios may persist.\textsuperscript{107} Lactate has been reported in PRES, and when accompanied by vasoconstriction, a contribution from ischemia has been suggested.\textsuperscript{42,107}

**The Controversy over the Mechanism of PRES**

The cause of PRES is not yet understood. Hypertension with failed autoregulation and hyperperfusion remains a popular consideration for the developing brain edema.\textsuperscript{18,92,119,120} Alternatively, endothelial dysfunction/injury, hypoperfusion, and vasoconstriction may lead to altered integrity of the blood-brain barrier.\textsuperscript{4,121,122}

Although commonly cited, several problems exist with the hypertension/hyperperfusion theory. PRES is seen in the absence of hypertension in 20%–40% of patients.\textsuperscript{17,27,36,39} In the remainder, though some degree of hypertension is present, reported blood pressure does not typically reach the limit of autoregulation (mean arterial pressure >150–160 mm Hg).\textsuperscript{123} Also, several recent studies have noted less vasogenic edema in severely hypertensive patients when compared with normotensive patients, contrary to the expected result if severe hypertension with failed autoregulation was the mechanism behind PRES.\textsuperscript{36,103} The biologic observations (strengths/weaknesses) related to both theories will be reviewed in detail in Part 2.

**Conclusion**

PRES develops in patients with complex systemic conditions such as eclampsia, after transplantation, in infection/sepsis/shock and autoimmune disease, and after cancer chemotherapy. Hypertension is absent in ~25% of patients and, when present, does not typically reach the level of failed autoregulation. The imaging appearance typically demonstrates symmetric vasogenic edema with several characteristic patterns, generally representing a distribution between lateral and medial cerebral arterial branches (ie, a watershed distribution). Vasculopathy is commonly identified by CA or MRA, and most studies have demonstrated reduced brain perfusion in regions of PRES. The mechanism responsible for the imaging appearance remains unclear and controversial.

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**Fig 3.** The patient is a 56-year-old woman with a thigh abscess (Klebsiella and Enterococcus species), baseline blood pressure of 156/88 mm Hg, and multiple organ failure (coagulopathy, acute respiratory distress syndrome, hepatic dysfunction with shock liver, and renal failure). She developed altered mentation and a seizure with toxicity blood pressure 164/75 mm Hg. A. Axial MR image (fluid-attenuated inversion recovery sequence) demonstrates extensive PRES vasogenic edema in the parietal region bilaterally (curved arrows). B. rCBV color map demonstrates severe flow reduction in the parietal region bilaterally (curved arrows), consistent with the regions of PRES imaging abnormality. PRES cortex rCBV relative to the reference cortex is 31% in the right parietal and 33% in the left parietal region.
Do not hallucinate.
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