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# Posterior Reversible Encephalopathy Syndrome in Kidney Disease

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## INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a clinico-radiographic syndrome of varying causes and is associated with characteristic neuroimaging findings. Many reports of PRES are associated with hypertension and the use of certain drugs. However, the relationship between kidney disease and PRES, given that most renal patients have the aforesaid risk factors, remains ill defined. We attempt here to share our experience with PRES in 4 different circumstances.

## CASE PRESENTATION

### Case 1

Case 1, a 35-year-old woman, was found to have solitary functioning kidney and mild renal insufficiency during routine evaluation. She was evaluated and continued on conservative management. She underwent a right open simple nephrectomy in view of recurrent pyelonephritis and was continued on hemodialysis. Renal biopsy showed chronic pyelonephritis with marked glomerulosclerosis. She underwent deceased donor renal transplantation. She was started on triple immunosuppression with tacrolimus, mycophenolate mofetil, and prednisolone. On postoperative day 4, she developed generalized tonic clonic seizures followed by respiratory failure. She was mechanically ventilated and was started on parenteral antiepileptic drugs. Magnetic resonance imaging (MRI) of the brain yielded findings suggestive of PRES with brain herniation. Her tacrolimus level at this point was 6.4 ng/ml, and the drug was withheld. There was no significant increase in antihypertensive requirement during the episode. She became symptomatically better and was discharged on a mechanistic target of rapamycin (mTOR) inhibitor–based triple immunosupression regimen (everolimus, mycophenolate mofetil, and prednisolone). She had 2 episodes of biopsy-proven acute rejection within the first 6 months after transplantation and was treated with i.v. methylprednisolone pulses, plasmapheresis, and rituximab. There was significant chronic allograft nephropathy on the biopsy sample. Graft function stabilized. The patient's antihypertensive requirement increased at this point. Six months after transplantation, she developed accelerated hypertension and seizures, which once again necessitated mechanical ventilation. There were no new lesions visible on brain MRI, and cerebrospinal fluid analysis results were normal. Graft function slowly worsened, and she was re-initiated on hemodialysis in view of graft failure within 1 year after transplantation. She has been on maintenance hemodialysis since.

### Case 2

Case 2, a 12-year-old girl, developed facial puffiness and pedal edema. Investigations revealed nephrotic syndrome with mild renal failure (serum creatinine, 1.6 mg/dl). Renal biopsy findings revealed focal segmental glomerulosclerosis. She was started on oral corticosteroids. She developed accelerated hypertension and seizures within 1 month of starting treatment. Brain MRI revealed PRES. Cerebrospinal fluid analysis results were normal. She was treated symptomatically with blood pressure (BP) control. Her condition improved, and she was extubated. However, she needed 5 antihypertensive agents for adequate BP control. She developed steroid resistance and was given 2 doses of rituximab and a cyclophosphamide trial. At present, she is on steroids (40 mg once daily) with the following basic parameters: serum creatinine, 1.85 mg/dl; serum albumin, 2.57 mg/dl; serum cholesterol, 529.3 mg%; and 3+ urine protein.

### Case 3

Case 3, a 12-year-old boy, was diagnosed as having steroid-dependent nephrotic syndrome at the age of 4 years. He developed severe renal failure and was initiated on hemodialysis. He had seizures secondary to accelerated hypertension, which resolved with BP control consisting of 2 antihypertensive agents. There was no evidence of PRES on MRI at this point. He underwent living related renal allograft transplantation with a mildly positive crossmatch. He was given plasmapheresis and started on triple immunosuppression with tacrolimus, mycophenolate mofetil, and prednisolone. In the immediate posttransplantation period, he had delayed graft function with biopsy-proven calcineurin toxicitiy and 2 acute rejections, for which he was treated with calcineurin inhibitor withdrawal, plasmapheresis, i.v. Ig, rituximab, antithymocyte globulin, and i.v. corticosteroids. He was then restarted on a tacrolimus-based regimen along with 3 antihypertensive agents for BP control. Four months after surgery, he had severe headache, convulsions, and altered sensorium with accelerated hypertension. Brain MRI showed evidence of posterior reversible encephalopathy. The tacrolimus level was 4.2 ng/ml. BP was controlled with 4 antihypertensive agents and the patient was switched to an mTOR inhibitor–based regimen (everolimus, mycophenolate mofetil, and prednisolone). His elevated BPs were controlled by optimizing his antihypertensive medications. He recovered fully with these measures. In the seventh postoperative month, he was diagnosed with gastrointestinal cytomegalovirus disease and started on treatment. Fourteen months after transplantation, he developed headache and accelerated hypertension. MRI showed evidence of PRES again. The patient improved with lowering of BP. Throughout the course of his illness, there was progressive worsening of graft function. At the time of writing, serum creatinine is 5.2 mg/dl.

### Case 4

Case 4, a 34-year-old man, was found to have severe renal failure and was initiated on twice-weekly hemodialysis. He was hypertensive at the time (requiring 3 drugs). Fifteen months after initiation of hemodialysis, he developed headache, vomiting, seizures, and accelerated hypertension. He had skipped his antihypertensive medication prior to the episode. Brain MRI showed evidence of posterior reversible encephalopathy syndrome. Antihypertensive and antiepileptic medications were continued, and with this he showed symptomatic improvement. He is presently on maintenance hemodialysis with 5 antihypertensive drugs.

### Case 5

Mrs. E, a 25-year-old woman, had moderate renal failure, nephrotic-range proteinuria, and microhematuria, along with joint pains and palpable purpuras. A renal biopsy showed features of Henoch–Schönlein purpura nephritis with crescents and background interstitial fibrosis and tubular atrophy (30%). The patient was treated with i.v. methylprednisolone and was started on oral cyclophosphamide. However, 26 days after starting the drug, she developed altered behavior, drowsiness, staring spells, and seizures. Blood pressure was normal, and MRI brain showed evidence of PRES. She improved after withdrawal of cyclophosphamide. The patient has had no episodes of repeated PRES. At the time of writing, she has stage 5 chronic kidney disease and has not been initiated on renal replacement therapy.

## DISCUSSION

Posterior reversible encephalopathy syndrome is a clinico-radiographic syndrome of various etiologies, first described in a 1996 case series.[1] This described a clinical syndrome with varying presenting features such as headache, confusion, decreased consciousness, visual disturbances, and seizures. The clinical syndrome was associated with characteristic neuroimaging findings of posterior cerebral white matter edema. The true incidence is not known; however, it has been described in various case series. All age groups appear to be affected, with patients ranging from 2 to 90 years.[2,3] Various case series suggest that PRES is more common in women, even after excluding eclampsia.[1,4] In our series, 3 patients (60%) were female. The pathogenesis is unclear, but it appears to be related to disordered cerebral autoregulation and endothelial dysfunction.[1] A wide variety of medical conditions have been implicated as causes of PRES, including the following: thrombotic thrombocytopenic purpura (TTP), drugs such as cyclosporine, tacrolimus, sirolimus, cytarabine, gemcitabine, interferon-$\alpha$, i.v. Ig, ipilimumab, methotrexate, tyrosine kinase inhibitors (pazopanib), sorafenib, sunitinib, vincristine, porphyria, hypercalcemia, hypomagnesmia, blood transfusion, contrast media exposure (cerebral or coronary angiography), hypertensive encephalopathy, hemolytic uremic syndrome, eclampsia, systemic lupus erythematosus, polyarteritis nodosa, cryoglobulinemia, and Wegener granulomatosis.[5,6]

Not much is known about PRES in kidney disease despite various independent risk factors being present (sometimes simultaneously) in renal disease, such as hypertension, autoimmune disease, and

immunosuppression. PRES could be underrecognized in patients with end-stage renal disease (ESRD). Marked hypertension is a cardinal risk factor in this population, associated with extracellular fluid volume expansion. Neuroimaging findings can be diverse, involving both anterior and posterior circulation territories. There have been scattered reports of PRES in kidney disease, but systematic research is lacking. In Hinchey's original paper,[1] more than half of the patients had renal failure of varying degrees, and 80% had hypertension. The findings in our case series are summarized in Table 1.

The incidence of PRES posttransplantation varies depending on the type of organ transplanted. A retrospective single-center study of 4222 solid organ transplant recipients showed an overall incidence of PRES of 0.49%,[7] with a 0.35% incidence in kidney transplants. This study showed that kidney transplant recipients with PRES had higher mean arterial pressure at presentation than liver transplant recipients but lower grades of vasogenic edema. The proposed causes were many, with calcineurin inhibitor toxicity being the chief culprit. However, toxicity has been difficult to prove, leading to the thought that it may not be a dose-related effect. In the article by Wong *et al.*,[8] most patients with PRES had serum tacrolimus levels in the therapeutic range. Other studies have shown that there is no correlation with neurotoxicity and the trough levels of tacrolimus.[9,10] A few case reports have shown high tacrolimus levels in cerebrospinal fluid, when corresponding serum levels have been within therapeutic limits.[11] Observations of the onset of PRES with low drug levels is seemingly paradoxical. Both transplant recipients in our series had PRES while on tacrolimus and had normal tacrolimus levels, lending weight to the previously mentioned studies. In addition, 1 patient while on tacrolimus did not have accelerated hypertension at the time of developing PRES, thus making it difficult to propose an exact mechanism for calcineurin inhibitor–induced PRES. The association of hypertension with PRES is well reported; however it may not always be a component in patients on immunosuppressive agents.[11] The interval between the starting of tacrolimus and the development of PRES is also variable; in our series, the intervals varied between 4 days and 16 weeks. The time course of developing PRES from the onset of beginning immunosuppressant therapy is particularly variable.[12]

Cyclophosphamide use has been reported in several cases of PRES, but mostly in combination with other cytotoxic agents for treatment of hematological malignancies and systemic lupus erythematosus.[13–15] PRES attributed to cyclophosphamide in these reports was confounded by other contributory factors such as fluid overload, hypertension, and/or renal failure. In contrast, our patient (20%) had only moderate renal failure with a controlled BP.

Interestingly, reported recurrence of PRES appears to be infrequent in a reported case series[7] despite the fact that the same precipitating factors may be present.

**Table 1.** Case patient characteristics

| Characteristic | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|---|---|---|---|---|---|
| Age, yr | 35 | 12 | 12 | 34 | 25 |
| Sex | F | F | M | M | F |
| Renal diagnosis at time of PRES | Renal allograft recipient POD 4 | FSGS with mild renal failure, nephrotic syndrome | Renal allograft recipient, 4 months posttransplant, NKD: FSGS | CKD 5D, presumed CGN | HSP nephritis, moderate renal failure |
| Recent initiation of HD (<4 wk) | No | No | No | No | No |
| Recent immunosuppression | Yes; tacrolmus | Yes; steroids | Yes- Tacrolimus, Rituximab, steroids | No | Yes; steroids, cyclophosphamide |
| Clinical features | Seizures | Seizures, altered sensorium | Headache, seizures, altered sensorium | Headache, seizures, vomiting | Altered behavior, drowsiness, staring spells, seizures |
| BP (MAP) | Acceptable | High (130/90) | High | High | Acceptable |
| MRI picture | Symmetrical bilateral parieto occipital and frontal white matter hyperintensities | Diffuse gyral swelling and hyperintensity seen affecting the supratentorial brain parenchyma. This is symmetrical bilaterally and affects all lobes with relative sparing of watershed areas. | Multiple and symmetric gyral T2 FLAIR hyperintensities noted in the bilateral frontal, parietal, and temporal cortices and bilateral cerebellar hemispheres | T2 and T2 FLAIR white matter hyperintensities seen in the bilateral high frontal, centrum semiovale parieto occipital region and also involving the left hemi midbrain with no asssociated diffusion restriction/blooming | T2 and FLAIR hyperintense signals noted in bilateral basal ganglia lateral and posterior temporal cortex (left > right) and posterior parietal cortex |
| Outcome | Resolution after prolonged ventilation | Resolution | Initial resolution, then another episode of symptomatic PRES, which also resolved completely | Resolution | Resolution prolonged |

BP, blood pressure; CGN, chronic glomerulonephritis; CKD, chronic kidney disease; F, female; FLAIR, fluid-attenuated inversion recovery; FSGS, focal segmental glomerulosclerosis; HD, hemodialysis; HSP, Henoch–Schönlein purpura; M, male; MRI, magnetic resonance imaging; NKD, native kidney disease; POD, postoperative day; PRES, posterior reversible encephalopathy syndrome.

In our case series, case patient 3 had a recurrence of PRES, having accelerated hypertension at both times despite multiple antihypertensive agents, reflecting the role of possible fluctuating or intermittent hypertension as described earlier. In addition, the first episode of PRES was associated with tacrolimus, which was then withdrawn. This patient also had graft dysfunction, and therefore multiple factors may have contributed to the second episode. Recurrent episodes of PRES have also been described in patients requiring dialysis, and this group of patients needs close monitoring.[16,17]

Neuroimaging is essential to the diagnosis of PRES. Typical findings are symmetrical white matter edema in the posterior cerebral hemispheres, but variations do occur.[18] Lesions of the anterior cortex have been reported but tend to occur in more severe cases, usually with edema being present in the posterior circulation territories.[19] Only 1 patient (20%, case patient 5) in our case series had lesions restricted to the posterior cerebral areas, with the others having diffuse cerebral involvement (Table 1).

Case series suggest that, in general, PRES has a benign prognosis, usually being reversible within days to weeks.[20–22] Radiologic improvement lags behind clinical recovery. All our patients recovered completely with no neurological deficits, including case patient 4, who had 2 documented episodes. Potentially grave consequences of this disorder include progressive cerebral edema or intracerebral hemorrhage.

The hallmark of PRES is vasogenic edema. The role of hypertension, often cited as the main cause of the vasogenic edema, is still controversial. Two theories have been proposed.[23] In the most popular theory, severe hypertension overwhelms the autoregulation of the cerebral vasculature, leading to hyperperfusion, arteriolar dilatation, and vasogenic edema. A predilection of PRES for the posterior regions of the brain has been explained by a relative lack of sympathetic innervation (as the sympathetic system raises the threshold of autoregulation). However, BP is not always elevated in PRES, especially in cases associated with immunosuppression and organ transplantation. In addition, the degree of vasogenic edema does not always correlate with the severity of hypertension.[7]

In the second theory, the principal problem is cerebral vasoconstriction resulting in downstream hypoperfusion, ischemia, and vasogenic edema due to capillary leak. Thus, hypertension may not be the sole driver of injury in all cases. Endothelial cell dysfunction, seen in organ transplant recipients and autoimmune diseases, can alter vascular tone and cause vasospasm and hypoperfusion. Cerebral vessels respond to the ensuing hypoxia by secreting vascular endothelial growth factor (VEGF) and increasing their permeability, thereby resulting in edema.[10]

These theories may not be mutually exclusive. Although there is some evidence to support either theory, retrospective study design, varying terminology, and different imaging modalities all make it difficult to choose 1 theory over another. In addition, they may not be distinct from one another. For instance, in our cohort, although hypertension was a common feature among all 5 patients, the peak mean arterial pressure (MAP) was below the recognized upper limit of autoregulation in 4 cases. Various BP cut-offs have been proposed by different authors, a peak SBP usually being between 170 and 190 mm Hg. Other authors have proposed an upper MAP threshold of 116 mm Hg to define clinically severe hypertension in the context of PRES.[2,12] Using this cut-off, all 5 of our case patients would have been classified as having severe hypertension. In this context, it is important to recognize that PRES can occur with acute severe hypertension or rapidly developing, fluctuating, and intermittent hypertension, as well as in patients with normal BP. Therefore it is important to assess the percentage rise of BP from the patient's baseline. Renal failure appears to be prominent among patients with hypertension who develop PRES, indicating that uremia, fluid overload, and electrolyte imbalance may be contributing factors.[22] Two of our patients (case patients 1 and 4) had a coexistent systemic inflammatory disorder with recent exposure to potent immunosuppressive agents. It is plausible that another process could reduce the upper autoregulatory threshold by altering vascular dynamics, thus increasing the potential for hypertension-mediated injury. PRES is not uncommon in the pediatric population,[24] as shown in 2 of our patients (case patients 2 and 3). It is important to consider this diagnosis in children presenting with seizure, visual disturbances, headache, and altered mentation in an appropriate clinical setting.

The role of steroids in the treatment of PRES is debatable, with a possible role in reducing the vasogenic edema. Case patients 2 and 3 were given short courses of i.v. steroids in their management. It was not possible to be dogmatic about steroid efficacy in these cases because whether the response was due to steroid effect or withdrawal of the inciting factor (accelerated hypertension in both cases) was difficult to determine. Literature evidence has shown multiple theories, with a possible role in systemic lupus erythematosus (SLE)–related PRES,[25] but no controlled trials have been conducted. Other authors have suggested no role for steroid therapy in PRES.[26] It remains a controversial subject.

PRES may occur after solid organ transplantation, but the exact incidence is unknown. In a study published by Bartynski *et al.*,[27] PRES developed in around 0.5% (27) of 4222 patients who underwent solid organ transplantation within the study period, thereby showing a low incidence in the transplantation group, similar to the general population. Further categorization showed that PRES typically developed in the first 2 months in patients who had liver transplants (9 of 10 patients) and was associated with cytomegalovirus, mild rejection, or systemic bacterial infection. PRES also typically developed after 1 year in patients who had a kidney transplant (8 of 9 patients) and was associated with moderate rejection or bacterial infection.

The composite picture of PRES in the renal transplantation scenario is complex. As mentioned previously in this article, the use of calcineurin inhibitors, particularly tacrolimus, predisposes a transplant recipient to PRES even in the presence of apparently normal drug levels. In this situation, conversion to an alternative regimen such as cyclosporine or mTOR inhibitor—based therapy may help, but each is fraught with its own risks. Sirolimus itself has been known to cause PRES, so the decision to substitute tacrolimus with either cyclosporine or sirolimus should be done taking into consideration the other side effects associated with each drug. Reduction in drug dosage or prompt removal of the cytotoxic drug is usually recommended in cases of PRES associated with cytotoxic agents. However, cases are reported in which symptoms resolve while the medication is maintained.[28] Another possible strategy is to withdraw the presumed offending drug at the time of PRES and then re-introduce it at a lower dose once PRES has resolved.[29] Opinions vary on the subject, and the lack of a definitive strategy reflects a paucity of studies on the condition.

The relationship between PRES and acute rejections is difficult to define. In the often-cited Bartynski *et al.* study,[27] PRES developed after 1 year in patients who had SOT of the kidney and was associated with moderate rejection or bacterial infection. This association is not definitively causal and may be multifactorial, with elevated BP due to steroid therapy, worsening renal function, and increased dosage of calcineurin inhibitors being possible contributory factors. The type of solid organ transplant seems to have little role in determining the incidence of PRES.

In general, kidney transplant recipients developed PRES many years after transplantation compared to early in the course in liver transplant recipients. Although the average BP at the time of PRES development was higher in kidney transplant recipients (average mean arterial pressure, 143 ± 20 mm Hg), brain edema was greater in liver transplant patients. PRES was also seen in association with infections and narcotic or cocaine use.

In conclusion, posterior reversible encephalopathy syndrome can be seen in association with kidney disease in various scenarios, common ones being accelerated hypertension and calcineurin inhibitor use. Patients on immunosuppressive regimens need not have toxic levels of the drug or hypertension at the time of development of PRES. It is a condition that requires early detection and prompt treatment in view of a generally benign prognosis.

## DISCLOSURE

All the authors declared no conflict of interest.

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