Case Report

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Clinical features and outcomes of posterior reversible encephalopathy syndrome after heart transplantation: a case series

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Posterior reversible encephalopathy syndrome (PRES) is a rare neurological disease that may be associated with hypertension, autoregulatory failure, and the use of calcineurin inhibitors following heart transplantation (HT). In this article, we present a case series of PRES, discussing its potential causes and management strategies. Among the 126 HT recipients at our hospital, four were diagnosed with PRES. Three of these patients developed PRES within 7 days after HT. Prior to the onset of PRES, all patients experienced sustained hypertension, and strict blood pressure (BP) control was maintained. Three of the four patients recovered without PRES recurrence, while one patient died of sepsis after an episode of altered consciousness. Hypertension was observed in all patients prior to the onset of PRES, and the majority experienced symptom improvement with BP control. While most cases of PRES were reversible with conservative treatment, including the administration of antiepileptics, one irreversible case resulted in in-hospital mortality. Thus, PRES can have serious outcomes and is not invariably benign.

Keywords: Posterior reversible encephalopathy syndrome; Calcineurin inhibitors; Heart transplantation; Case report

INTRODUCTION

Heart transplantation (HT) is a definitive treatment for patients with advanced heart failure. Following HT, patients may experience a range of complications that can impact their survival and functionality. These complications, which can arise early or late, include graft rejection, infections, neoplasms, kidney disease, and neurological issues [1]. Perioperative neurological complications are reported in 10% to 20% of patients undergoing HT, including those with transient, mild symptoms [2]. Much less commonly, posterior reversible encephalopathy syndrome (PRES)—a rare neurological disorder—can develop after HT [3]. This condition typically presents as subacute or acute encephalopathy, often accompanied by symptoms such as headache, nausea, altered mental status, focal neurological deficits, visual impairment, and seizures [4]. While symptoms of PRES usually resolve over time [5], it can occasionally lead to outcomes such as parenchymal infarction or hemorrhage, permanent neurological dam-

HIGHLIGHTS

- Hypertension was observed in all patients prior to the onset of posterior reversible encephalopathy syndrome (PRES).
- Most patients exhibited symptom improvement with blood pressure control.
- The majority of PRES cases were reversible with conservative management, which included the administration of antiepileptics.
- One irreversible case resulted in in-hospital mortality, suggesting that PRES is not always benign.

age, or even death [6]. Factors associated with PRES include hypertension, the use of calcineurin inhibitors (CNIs), and metabolic derangements [4,7,8]. Although PRES has been repeatedly documented following solid organ transplantation, the specific clinical features and outcomes of perioperative PRES after HT have seldom been explored. This report aims to describe four cases of PRES that developed in recipients of HT at a single tertiary center.

CASE REPORT

This case report was reviewed and approved by the Institutional Review Board of Pusan National University Yangsan Hospital (IRB No. 05-2023-169). Written informed consent was obtained from all patients.

Between January 2014 and January 2023, four (3.2%) of the 126 HT recipients in a single tertiary center cohort were diagnosed with PRES following transplantation. Three of these patients received only a heart transplant, while the fourth underwent a simultaneous heart-kidney transplant. The first patient was a 62-year-old man

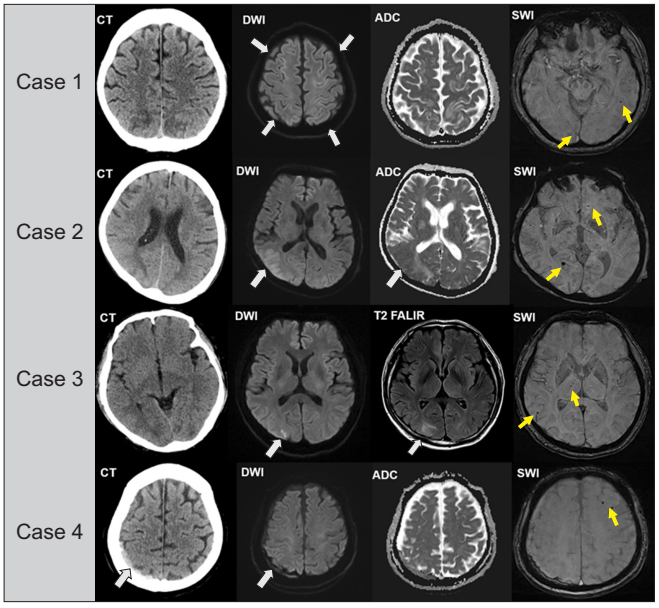

Fig. 1. Radiological examination results of four patients with PRES following heart transplantation. Case 1: CT reveals subtle areas of decreased density in both parietal lobes. MRI demonstrates diffuse high signal intensity along the sulci of the frontal, parietal, and occipital lobes on DWI (arrows). Additionally, multiple small microbleeds (yellow arrows) are scattered throughout both cerebral hemispheres. Case 2: CT scan does not reveal specific findings. However, MRI with DWI shows areas of restricted fluid (arrows) in the right parieto-occipital lobe, and multiple microbleeds (yellow arrows) are observed in the cerebrum and cerebellum on SWI. Case 3: CT indicates a subtle area of decreased density with a mass effect in both parieto-occipital lobes. MRI indicates a localized area of restricted diffusion (arrow) in the right parieto-occipital lobe on both FLAIR and T2-weighted sequences. Furthermore, multiple microbleeds (yellow arrows) are present in the cerebral and cerebellar hemispheres as well as the brainstem. Case 4: CT shows a gradually resolving small SDH (arrow) along the right parietotemporal cerebral convexity. MRI displays a small chronic SDH (arrow) in the right parietal cerebral convexity. Several microbleeds (yellow arrow) are observed in both cerebral hemispheres. CT, computed tomography; DWI, diffusion-weighted imaging; ADC, apparent diffusion coefficient; SWI, susceptibility-weighted imaging; FLAIR, fluid-attenuated inversion recovery; PRES, posterior reversible encephalopathy syndrome; MRI, magnetic resonance imaging; SDH, subdural hematoma.

diagnosed with dilated cardiomyopathy (DCMP) who underwent HT. Six days postoperatively, he experienced a decrease in consciousness and recurrent generalized tonic-clonic (GTC) seizures, despite having no history of epilepsy. Although his blood pressure was managed with medications such as nicardipine, his systolic pressure had been consistently elevated (>140 mmHg) before the onset of symptoms. After HT, the patient was administered tacrolimus, with a blood concentration of 7.2 ng/mL recorded before symptom onset. Brain magnetic resonance imaging (MRI) revealed high signal intensity along the cortex and subcortex of the frontal, parietal, and occipital lobes, along with focal diffusion restriction in the temporal lobe on diffusion-weighted imaging (DWI) and multifocal small microbleeds in both cerebral hemispheres on susceptibility-weighted imaging (SWI) (Fig. 1). The patient's blood pressure was successfully managed with medication, the CNI was not discontinued, and his condition improved without recurrence (Table 1, Fig. 1).

The second case involved a 62-year-old woman with DCMP that developed after a secondary valve operation. She had previously undergone kidney transplantation and surgical aortic valve replacement (SAVR) for severe aortic valve stenosis. Following transplantation, she experienced a recurrence of renal failure and underwent a second SAVR procedure due to infective endocarditis.

Table 1. Baseline characteristics of the four patients with posterior reversible encephalopathy syndrome

Variable	Case 1	Case 2	Case 3	Case 4
Sex	Male	Female	Male	Male
Age (yr)	62	62	52	60
Height (cm)	165.8	155.6	173.0	163.4
Body weight (kg)	55.3	47.7	62.0	50.3
Body surface area (m^2)	1.60	1.44	1.73	1.51
Primary diagnoses before transplantation	Dilated cardiomyopathy	Dilated cardiomyopathy End-stage renal disease	Dilated cardiomyopathy	Dilated cardiomyopathy
Surgical history	None	Kidney transplantation (2015) SAVR due to aortic stenosis (2013) Redo SAVR due to infective endocarditis (2020)	None	SAVR and mitral valve replacement (1990)
Organ(s) transplanted	Heart	Heart, kidney	Heart	Heart
Donor/recipient predicted heart mass ratio	1.31	1.47	0.95	1.09
Hemoglobin (g/dL)	9.3	12	12.5	9.7
Platelets (μL)	108,000	164,000	384,000	70,000
White blood cells (μL)	8,480	11,120	26,720	17,080
Lactate dehydrogenase (U/L)	392	434	385	463
Sodium (mmol/L)	127	142	136	135
Potassium (mmol/L)	3.9	4.3	3.8	4.0
Calcium (mg/dL)	8.5	9.8	9.4	8.3
Blood urea nitrogen (mg/dL)	55.5	44.9	21.1	40.1
Ionized magnesium (mmol/L)	0.57	No data	0.65	0.76
Creatinine (mg/dL)	1.96	1.76	0.88	2.52
Calcineurin inhibitor	Tacrolimus	Tacrolimus	Tacrolimus	None
Diabetes mellitus	Yes	No	No	No
Pretransplantation blood pressure (mmHg)	83/54	92/60	99/60	83/45
Pretransplantation cardiac index ($L/min/m^2$)	2.7	3.8	2.7	2.7
Posttransplantation cardiac index ($L/min/m^2$)	4	4	3.2	4.1

SAVR, surgical aortic valve replacement.

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Subsequently, however, her cardiac function severely deteriorated. As a result, she was placed on continuous renal replacement therapy and venoarterial extracorporeal membrane oxygenation prior to simultaneous kidney-HT (sKHT). Her condition improved after sKHT, and she was managed with conservative treatment. However, 34 days post-sKHT, the patient experienced headaches, dizziness, and visual impairment, followed by a decrease in consciousness. Her kidney function had been well-maintained, and she was in the process of weaning from dialysis. However, her systolic blood pressure was elevated above 140 mmHg before symptom manifestation, prompting treatment with oral antihypertensives. The patient was also taking tacrolimus, with a blood concentration of 7.6 ng/mL before the symptoms appeared. Brain MRI with DWI revealed diffusion restriction in the right parieto-occipital lobe, and multiple microbleeds were observed in both cerebral and cerebellar hemispheres on SWI (Fig. 1). Additionally, brain computed tomography (CT) angiography was performed to exclude ischemic stroke, revealing no steno-occlusive lesions in the intracranial and neck vessels. Consequently, we diagnosed the patient with PRES. To manage this condition, we initiated antihypertensive therapy, discontinued tacrolimus, and resumed dialysis. Despite these interventions, epileptiform waves persisted on electroencephalography, and the patient's level of consciousness did not improve. Following admission to the intensive care unit and tracheostomy placement, the patient developed pneumonia and sepsis, which ultimately led to her death.

The third patient was a 52-year-old man with DCMP who presented with ventricular fibrillation and was resus-

Table 2. Clinical features, management strategies, and prognoses of PRES cases

Variable	Case 1	Case 2	Case 3	Case 4
Onset duration after HT (day)	6	34	6	3
Symptom	Altered level of consciousness Generalized tonic-clonic seizure	Altered level of consciousness Visual disturbance	Generalized tonic-clonic seizure Headache	Altered level of consciousness Generalized tonic-clonic seizure Stereotypical behavior Headache
Calcineurin inhibitor	Tacrolimus	Tacrolimus	Tacrolimus	None
Tacrolimus level (ng/mL)	7.2	7.6	3.3	
Radiologic feature	CT: Subtle low density in both parietal lobes MRI: Diffuse sulcal high signal intensity along both frontal, parietal, and occipital lobes on DWI; multifocal small microbleeds in both cerebral hemispheres	CT: Nonspecific findings MRI: Focal diffusion restriction at the right parieto-occipital lobe on DWI; multiple microbleeds in both cerebral and cerebellar hemispheres on SWI	CT: Subtle low density with mass effect at both parieto-occipital lobes MRI: Focal diffusion restriction at the right parieto-occipital lobe on FLAIR and T2; multifocal microbleeds in both cerebral/cerebellar hemispheres and the brainstem on SWI	CT: Gradually resolving, small SDH along the right parietotemporal cerebral convexity MRI: Small chronic subdural hematoma along the right parietal cerebral convexity; several microbleeds in both cerebral hemispheres on SWI
Blood pressure at PRES onset (systolic/diastolic, mmHg)	192/89	158/91	180/121	150/57
Management	Blood pressure control	Initiation of S-levetiracetam and valproate Blood pressure control Discontinuation of tacrolimus	Initiation of S-levetiracetam Blood pressure control	Initiation of valproate and clonazepam Blood pressure control
Antihypertensive treatment	Nicardipine	Nicardipine	Nicardipine	Nicardipine
Continuation of antiepileptic medication	None	S-levetiracetam Valproate	S-levetiracetam (maintained up to the present)	Valproate (maintained up to the present)
Prognosis	No recurrence in 6 months	No improvement in consciousness level; died of sepsis	No recurrence in 6 months	No recurrence in 6 months

PRES, posterior reversible encephalopathy syndrome; HT, heart transplantation; CT, computed tomography; MRI, magnetic resonance imaging; DWI, diffusion-weighted imaging; SWI, susceptibility-weighted imaging; FLAIR, fluid-attenuated inversion recovery; SDH, subdural hematoma.

citated in the emergency room. His cardiac function did not recover, and he underwent HT. Six days posttransplant, the patient experienced headache and GTC seizures. Prior to the onset of symptoms, he had exhibited an increase in systolic blood pressure (≥150 mmHg) that did not respond to antihypertensive treatment. Following HT, he was placed on tacrolimus, with a blood concentration of 3.3 ng/mL recorded before symptom onset. Brain MRI with DWI revealed focal diffusion restriction in the right parieto-occipital lobe. Symmetrical high signal intensities were observed in the subcortical regions of both occipital lobes and the left parietal lobe on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences. Additionally, SWI indicated multifocal microbleeds in both cerebral and cerebellar hemispheres as well as the brainstem (Fig. 1). Following continuous blood pressure management and the adjustment of the CNI toward the lower end of the therapeutic range, the patient recovered with no recurrence of symptoms.

The fourth case involved a 59-year-old man with advanced heart failure who had undergone aortic and mitral valve replacement. Three days after the transplant, he experienced headache and decreased consciousness and exhibited stereotypical behaviors (such as tapping his head, bending his arms, and repeating words), followed by GTC seizures. The patient had not received CNI treatment after the transplant and exhibited persistent hypertension despite receiving medication to lower blood pressure. Brain CT showed a small subdural hemorrhage along the right cerebral convexity. Brain MRI did

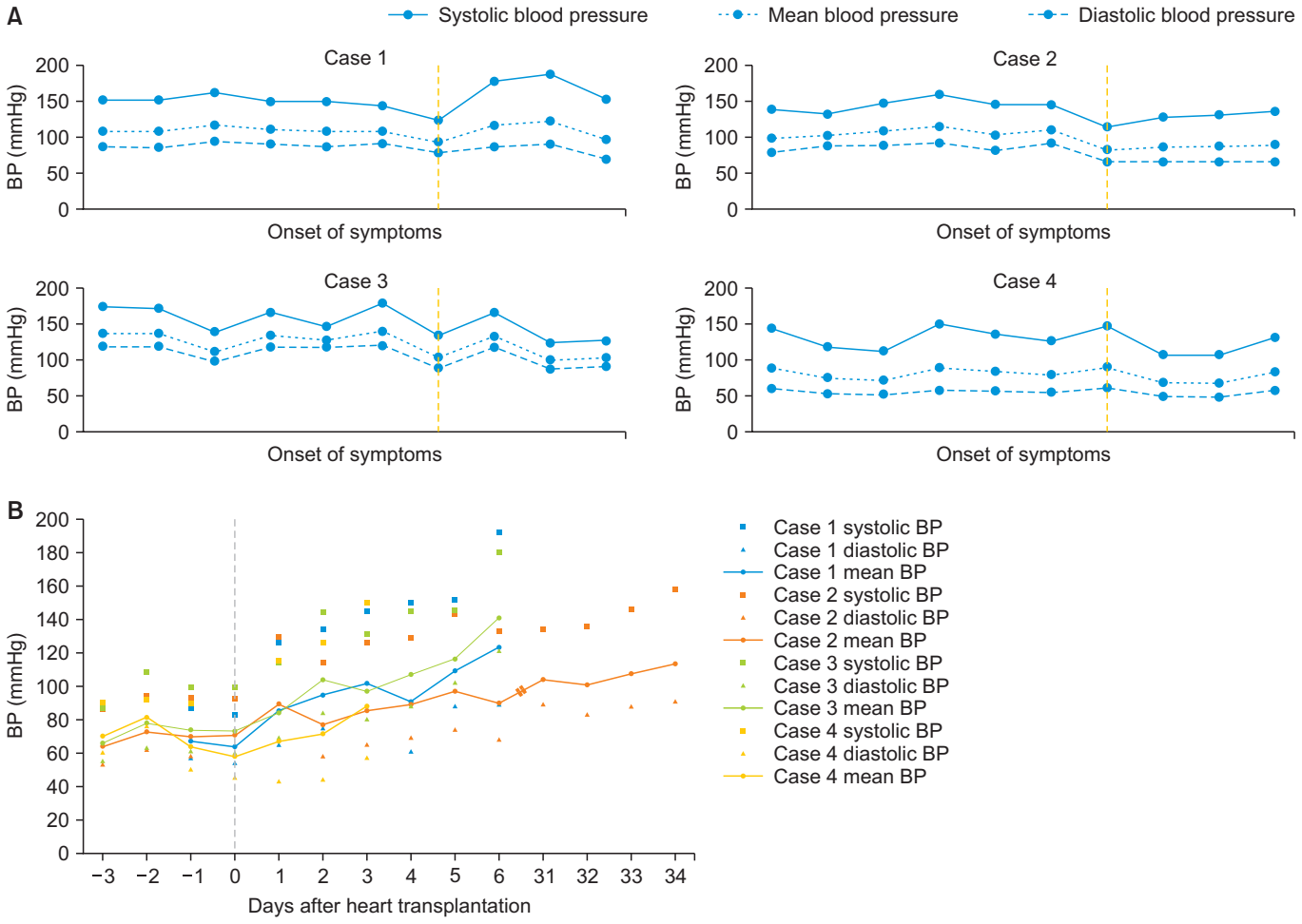

Fig. 2. (A) Blood pressure (BP) trends before and after posterior reversible encephalopathy syndrome (PRES) symptom onset. Measurements were taken at 3-hour intervals. Persistently elevated BP was observed before the onset of symptoms. (B) BP trends before and after transplantation for the four patients with PRES.

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not display diffusion restriction on DWI, but it did reveal several microbleeds in both cerebral hemispheres on SWI (Fig. 1). Following the administration of antiepileptic drugs (clonazepam and valproate) and the management of his blood pressure, the patient's consciousness gradually improved. He experienced no recurrence of seizures or other symptoms.

Unfortunately, only one of the four patients underwent T2 and FLAIR brain MRI examinations at symptom onset. The remaining three patients were only scanned using a diffusion protocol, which included DWI and SWI brain MRI examinations. Serum magnesium levels remained stable in all but one patient, in whom magnesium levels were not assessed. The basic characteristics of the patients are detailed in Table 1, while Table 2 summarizes the clinical features, management, and prognosis of the PRES cases.

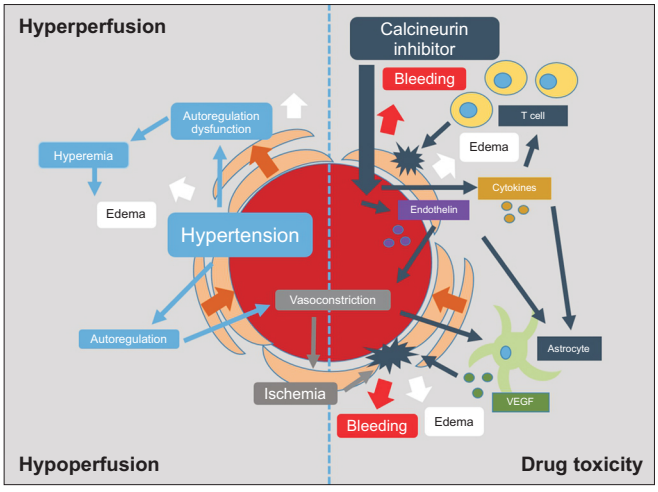

Fig. 3. The two primary hypotheses of posterior reversible encephalopathy syndrome (PRES) are illustrated in the box. The left side presents the hyperperfusion-hypoperfusion hypothesis. According to this concept, elevated blood pressure that surpasses the autoregulatory capacity of blood vessels causes them to dilate. This dilation leads to sustained hyperemia and, consequently, perivascular edema. In turn, the hypoperfusion theory posits that an overactive autoregulatory response to high blood pressure results in excessive vasoconstriction. This can cause ischemia, swelling, and hemorrhage due to the overly constricted blood vessels. The right side of the box illustrates the hypothesis that PRES is induced by toxicity from calcineurin inhibitors (CNIs). This hypothesis proposes that CNIs stimulate the vascular endothelium to release cytokines, which in turn activate cytotoxic T lymphocytes. The activated T cells inflict damage on the endothelium, causing hemorrhage and edema. Furthermore, this hypothesis includes the increased secretion of endothelin, which induces vasoconstriction and subsequent ischemia. It also involves the activation of astrocyte vascular endothelial growth factor (VEGF) secretion, which damages endothelial cell junctions, resulting in hemorrhage and edema.

Fig. 2 illustrates the blood pressure trends prior to the onset of PRES symptoms in the four patients.

DISCUSSION

PRES is observed in patients with hypertensive encephalopathy and in those who undergo organ transplantation, particularly following cyclosporine therapy. The most common and prominent feature of PRES is a nonspecific encephalopathy, present in up to 94% of patients [8,9]. About half of these patients gradually develop a dull and diffuse headache, although its onset can occasionally be sudden and severe [9]. Approximately three-quarters of patients experience either focal or GTC seizures. While seizures are usually not the initial symptom, they may progress to continuous seizure activity, known as status epilepticus, in up to 18% of patients [9]. Visual abnormalities are also prominent, occurring in 20%–39% of individuals with PRES [8,9]. Less frequently observed symptoms include focal weakness, ataxia, hyperreflexia, and spinal symptoms [8]. Among the four patients discussed, three experienced GTC seizures. Three patients exhibited altered levels of consciousness, and two individuals reported diffuse mild headaches. The clinical presentations of these patients were consistent with previous reports of PRES. All but one patient showed improvement and the absence of recurrence over a 6-month period. Of the three patients without symptom recurrence, one managed their symptoms without antiepileptic medications, while the other two remained on antiepileptic therapy.

In patients with PRES, brain imaging often reveals reversible lesions in the bilateral parieto-occipital regions. The term "reversible posterior leukoencephalopathy syndrome" was initially used, when lesions were thought to be confined to the posterior cerebral white matter. However, the condition was later renamed to PRES upon the discovery of cortical lesions [5,8]. Both vasogenic and cytotoxic edema may be present, and some cases progress to cerebral infarction on subsequent imaging, indicating that not all PRES cases are fully reversible [10]. Lesions associated with PRES are not restricted to the posterior brain but can also involve the frontal lobes, brainstem, cerebellum, basal ganglia, or thalamus [11]. The characteristic radiological findings of PRES can be categorized into three types based on cerebral hemisphere involvement, as analyzed by Bartynski et al. [10] using MRI data.

The first and generally most common type is confined to the parietal and occipital lobes, which was observed by Bartynski et al. [10] in only 22% of cases. The second type, impacting the superior frontal sulcus, accounted for 27% of cases and involves the middle and posterior parts of the superior frontal gyrus, cortex, and subcortical white matter, sparing the anterior frontal region. The third type, characterized by a holohemispheric watershed pattern, was seen in 23% of cases, with lesions occurring in the watershed regions of the frontal, parietal, and occipital lobes. Lesions can present partially or asymmetrically within these categories, and in some cases, two types may coexist. Brain hemorrhage has been reported in 5%–27% of PRES cases [11]. In a systematic study by Hefzy et al. [12], hemorrhage occurred in 15% of cases and included subarachnoid hemorrhage less than 5 mm, subdural hemorrhage, and intraparenchymal hematoma. In the present study, among the four patients studied, two exhibited swelling in the posterior cortex, subcortical white matter, or deep white matter, which are typical radiologic characteristics of PRES. No swelling was observed in the other two patients. This discrepancy may stem from cortical damage occurring in the early stages of PRES, with subsequent involvement of the subcortical white matter over time. Microbleeds were observed on MRI in all four patients, and one patient exhibited an asymmetric subarachnoid hemorrhage in the posterior brain.

The exact mechanism underlying PRES is not fully understood; however, major contributing factors include uncontrolled hypertension and immunosuppressive therapy following organ transplantation [10,13]. Infections, autoimmune diseases, and renal disorders are also known to precipitate PRES. Central to the pathogenesis of PRES are the theories of hyperperfusion-hypoperfusion and endothelial damage (Fig. 3) [10,14]. Previous studies indicate that approximately 75% of patients with PRES experience hypertension before the onset of symptoms [5,8,10], and the theory of hyperperfusion-hypoperfusion and impaired autoregulation is the most plausible [4,7]. Specifically, when blood pressure exceeds the autoregulatory capacity of cerebral vessels, arterioles dilate. This dysfunction in hypertension-induced cerebrovascular autoregulation can lead to vasoconstriction and subsequent brain tissue hypoperfusion, resulting in blood–brain barrier disruption, edema, and microhemorrhage. PRES typically first manifests with cortical edema, especially in the parieto-occipital regions; this is due to the relative lack of sympathetic innervation in these areas, which contributes to vascular dysregulation. In patients undergoing HT, hypertension is common—affecting about 50% to 80% of patients—and is likely a meaningful factor in the development of PRES after HT [9,15]. Hypertension following HT can be induced by various mechanisms, including medications used posttransplantation, which can contribute to elevated blood pressure and, consequently, PRES. Another credible hypothesis involves endothelial damage. Immunosuppressive drugs, such as CNIs, can activate endothelial cells, leading to cytokine release [4,16]. This cascade of events results in vasoconstriction, tissue ischemia, and increased blood–brain barrier permeability, all of which are implicated in the development of PRES. Additionally, these mechanisms act on astrocytes, prompting them to produce more vascular endothelial growth factor (VEGF). This disrupts the integrity of tight junctions in cerebral blood vessels, leading to edema [4,10,11]. The resulting endothelial damage stimulates an increase in VEGF secretion, further exacerbating the edema and leading to red blood cell damage and microhemorrhages.

The four patients experienced ongoing hypertension prior to the onset of symptoms and were receiving medication for its management. Of these patients, three were on continuous CNIs; after symptom onset, two of these individuals showed improvement through blood pressure management alone, without discontinuing CNIs. Instead, a lower blood concentration of CNI was maintained by adjusting the dosage, guided by therapeutic drug monitoring, and no aggravation or recurrence of symptoms occurred. Although CNI-induced endothelial damage is a key factor in the development of PRES in patients after HT, as supported by reports of several cases [6,13], the theory of hyperperfusion-hypoperfusion due to elevated blood pressure may also play an important role. Treatments targeting this mechanism appear to be effective in improving symptoms. Our cases contribute valuable insights into the relationship between CNI use and PRES treatment, suggesting that if blood pressure can be controlled without discontinuing CNIs, symptom improvement may be achieved through the use of antiepileptic medications alone. This approach could also provide a reference for managing patients after HT, as it might reduce the risk of acute graft rejection associated with CNI discontinuation. However, these cases may not be generalizable to all patients, as individual characteristics and drug responses must be carefully considered.

Among the various neurological complications that

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can arise following HT, PRES is relatively uncommon and is associated with factors that occur after HT. In our study, of the 126 patients who underwent HT, PRES developed in only four individuals. In most of these cases, symptoms improved through blood pressure control and the administration of antiepileptic medications. Notably, PRES arose even in the absence of CNIs, and symptoms were alleviated without discontinuing CNIs. This indicates that the proposed mechanisms—immunosuppressant-induced epithelial cell damage and the hyperperfusion-hypoperfusion theory of blood pressure fluctuations exceeding autoregulatory capacity—may substantially influence the development of PRES after HT. Of the four patients, one did not respond to treatment, suggesting that not all instances of PRES follow a reversible, benign course.

ARTICLE INFORMATION

Conflict of Interest
No potential conflict of interest relevant to this article was reported.

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