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Case Report

A Rare Case of Cyclosporine-Induced Posterior Reversible Encephalopathy Syndrome in a Patient after Liver Transplantation

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Abstract:

Posterior reversible encephalopathy syndrome (PRES) is a neurological condition characterized by seizures, encephalopathy, visual disturbances, and headache, often occurring in the context of hypertension and immunosuppressive therapy after solid organ transplantation. Although classically presenting with vasogenic edema in the parieto-occipital regions, atypical patterns may also occur. Here we report our experience with a case of cyclosporine-related PRES after liver transplant and summarize PRES clinical features through a literature review.

The case was a 53-year-old man who received a deceased donor liver transplant. His initial immunosuppressive therapy comprised cyclosporine/mycophenolate mofetil/prednisolone. Five months after transplantation, he was admitted to our center with altered mental status. The patient was diagnosed with PRES based on neurological symptoms and neuroimaging findings and recovered after switching from cyclosporine to everolimus. In addition, the lowering of blood pressure with drugs reported in the literature for use in PRES proved to be effective but challenging, requiring the use of multiple agents and only slowly leading to adequate control of hypertensive peaks. Nonetheless, hypertension management and supportive therapy allowed for a complete neurological recovery of the patient.

In conclusion, cyclosporine-associated PRES has a generally favorable prognosis with early diagnosis and prompt treatment, including altering or discontinuing CNIs and controlling blood pressure. CNI-associated PRES should be considered in patients exhibiting acute neurological symptoms after transplantation. Early diagnosis and immediate treatment are critical for a favorable prognosis.

Keywords: PRES; Patient; Cyclosporine; Encephalopathy

Introduction

Posterior reversible encephalopathy syndrome (PRES) is an acute or subacute cerebral syndrome, characterized by varied neurological symptoms, such as headache, seizures, encephalopathy, visual disturbances in various combinations, or focal neurological deficits [1, 5]. Neuroimaging usually reveals bilateral subcortical vasogenic edema, predominantly involving the posterior cerebral hemispheres, which typically occurs within the first month after transplantation [11].

The frequency of PRES in solid organ transplant patients varies from 0.4% to 6% [6]; after liver transplantation (LT), it has been reported in about 1% of cases [4]. Demographically, it can occur in all age groups, particularly in middle-aged women, with no statistically significant difference in gender, race, or mortality [2,10].

Factors contributing to the development of PRES include hypertension, metabolic disturbances, neurotoxicity of chemotherapeutic and immunosuppressive agents, and it is also associated with renal dysfunction, severe infections, eclampsia, and autoimmune diseases [4,14,15]. In 1996, Hinchey et al. [1] first reported that calcineurin inhibitors (CNIs) could cause this syndrome in 1–4% of solid organ transplant recipients [16], most commonly associated with tacrolimus and more rarely with cyclosporine [3,4].

PRES is completely reversible in the majority of cases. Timely diagnosis after LT is essential to initiate suitable symptomatic therapy and adjust immunosuppressive treatment.

Here, we report a case of cyclosporine-associated PRES following deceased-donor liver transplantation. The distinctive features of this case include delayed onset several months after transplantation and atypical localization of vasogenic edema in the brainstem - a presentation not previously reported in the available literature. The patient achieved complete neurological recovery following timely modification of immunosuppressive therapy and antihypertensive management.

Case Presentation

A 53-year-old man with chronic viral hepatitis B, delta agent–related end-stage liver disease underwent deceased donor LT. Postoperatively, his hemodynamics were unstable, requiring vasopressors, and the course was complicated by severe blood loss and acute renal failure, which was corrected with renal replacement therapy. His initial immunosuppressive therapy comprised cyclosporine, mycophenolate mofetil, and prednisolone.

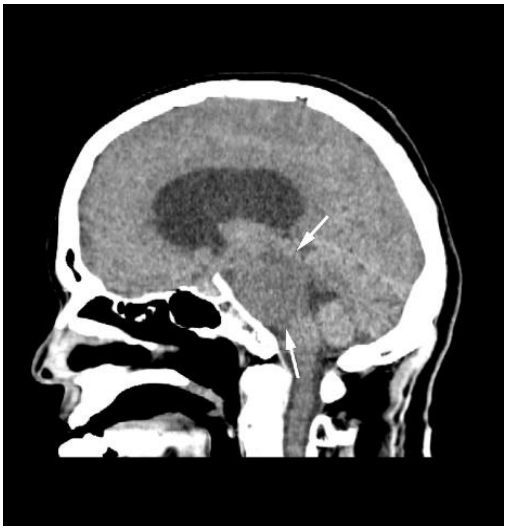
Five months after starting cyclosporine therapy, the patient experienced a fainting episode, likely due to high blood pressure. Thereafter, he began to complain of headache, which gradually worsened, and developed encephalopathy (confusion, disorientation), seizures, and visual abnormalities, accompanied by arterial hypertension. He was admitted to the hepatopancreatobiliary surgery and liver transplantation department for further evaluation.

On admission, the patient had altered mental status, fluctuating from somnolence and lethargy to agitation and confusion. Blood pressure was elevated at 220/140 mmHg and remained unstable throughout hospitalization. A femoral neck fracture aggravated his condition due to pain and limited mobility. The patient also had chronic kidney disease (CKD-EPI 42 mL/min/1.73 m²). His immunosuppression consisted of cyclosporine 400 mg BID, mycophenolate mofetil 360 mg BID, and prednisone 5 mg OD.

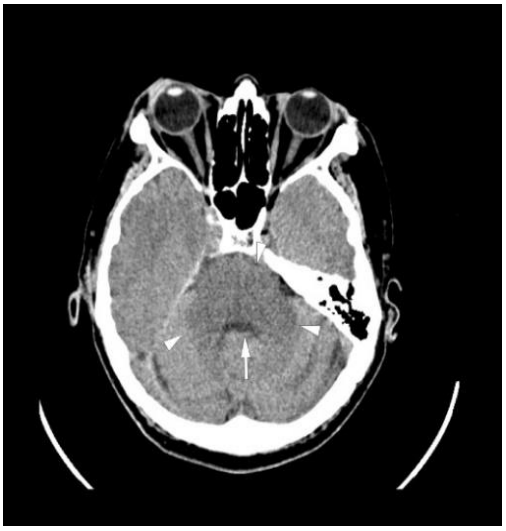
Initial investigations in the department identified elevated alkaline phosphatase (ALP) of 163 U/L (nr 40–129 U/L) and total bilirubin level of 27.49 U/L (nr 0–20.1 U/L). Other liver function tests were normal. The international normalized ratio (INR) was elongated to 1.32 (nr 0.8–1.2). Creatinine and urea were raised of 165 μmol/L (nr 44-133 μmol/L) and 17.46 mmol/L (nr 2.5-7.5 mmol/L), respectively. CRP - 42,65 mg/l (below 5 mg/l). His cyclosporine level was 141.2 ng/mL.

Computed tomography of his head showed focal hypodensities in the brainstem substance, with no hemorrhage (Figure 1). Magnetic resonance imaging (MRI) was not performed because the patient was hemodynamically unstable and experienced severe pain from a femoral neck fracture, making it unsafe and unfeasible for him to remain supine for the duration of the MRI study.

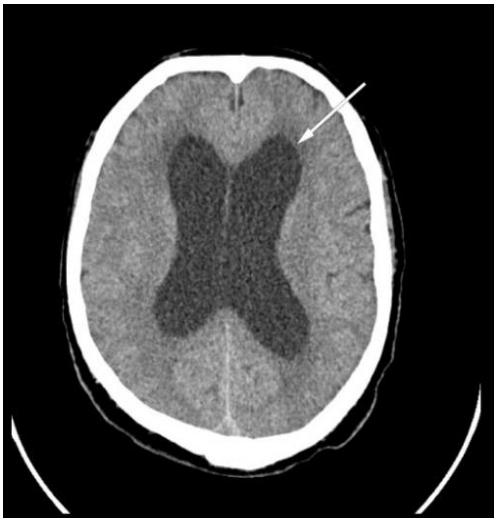
Figure 1. Computed tomography of the patient with PRES.



A



B



C

A non-contrast CT scan of the brain demonstrated changes predominantly involving the brainstem. On the sagittal slice, areas of decreased density are identified within the brainstem tissue, indicating vasogenic edema (A). Additionally, the edema extends to the cerebellar peduncles, with narrowing of the cerebrospinal fluid spaces and the fourth ventricle (B, indicated by the white arrow). There is also narrowing of the subarachnoid spaces due to cerebral edema and moderate enlargement of the lateral ventricles, suggesting intracranial hypertension (C).

The observed findings are consistent with hypertensive encephalopathy, which is characteristic of posterior reversible encephalopathy syndrome (PRES).

Clinicians suspected that PRES might have been precipitated by cyclosporine. Cyclosporine was immediately discontinued and substituted with low-dose everolimus. Prednisolone and mycophenolate mofetil were continued. Hypertension was effectively controlled with multiple antihypertensive drugs (bisoprolol, fosinopril, amlodipine, urapidil). Supportive measures included anti-edema therapy, anticonvulsants, and antibiotics.

During treatment, the patient showed complete resolution of clinical manifestations and laboratory improvement, including reduction of cholestasis and recovery of renal function (CKD-EPI 95 mL/min/1.73 m^2). The patient was discharged on day 20 on everolimus and prednisone. Over one year of follow-up, he remained well with no recurrence of PRES (see Table 1 for significant investigation reports).

Table 1. Chronological overview of clinical events, investigations, and treatment during the patient's hospitalization for PRES following liver transplantation.

Post-LT Day	Event	Notes / Intervention
05.07.2023 (Day 0)	Orthotopic liver transplantation from deceased donor	Postoperative course complicated by severe blood loss (9 L), hemodynamic instability requiring vasopressors, acute renal failure
Postoperative Days 1–30	ICU stay	Renal replacement therapy with hemofiltration and daily hemodialysis; initial immunosuppression: methylprednisolone → taper, calcineurin inhibitor (cyclosporine) from day 2, MMF continued
Approx. 5 months post-LT	Fainting episode	Likely due to uncontrolled hypertension; initial headache noted
Following weeks	Gradual worsening neurological symptoms	Headache, confusion, disorientation, seizures, visual disturbances, persistent hypertension
December 2023	Admission to hepatopancreatobiliary surgery & LT department	BP 220/140 mmHg, altered mental status, femoral neck fracture, CKD-EPI 42 mL/min/1.73 m², on cyclosporine 400 mg BID, MMF 360 mg BID, prednisone 5 mg OD
Day 1–2 of hospitalization	Diagnostics	Head CT: focal brainstem hypodensities, no hemorrhage; MRI not performed due to condition; labs: ALP ↑, total bilirubin ↑, creatinine ↑, urea ↑, CRP ↑, cyclosporine 141 ng/mL
Day 2 of hospitalization	Diagnosis	PRES suspected, likely cyclosporine-induced
Day 2–3 of hospitalization	Treatment initiated	Cyclosporine discontinued, switched to everolimus; prednisolone + MMF continued; antihypertensive therapy started (bisoprolol, fosinopril, amlodipine, urapidil); supportive therapy: anti-edema, anticonvulsants, antibiotics
Hospitalization Days 4–20	Clinical course	Gradual improvement in neurological status, BP stabilization, reduction of cholestasis, recovery of renal function (CKD-EPI 95 mL/min/1.73 m²)
Day 20 / Discharge	Discharge from hospital	On everolimus + prednisone; fully recovered neurological and renal function
1-year follow-up	Outcome	No recurrence of PRES; patient clinically stable

Table 2. Follow-up examinations and treatment.

Hospitalization days	1	2-3	6	8	13	16	18
Encephalopathy	Consciousness confused, no contact,	Consciousness confused, intermitten	Consciousness clear, intermitt	Consciousness clear, makes	Consciousness clear, makes	Consciousness clear, makes	Consciousness clear, makes

	disoriented, periodically agitated	t contact, disoriented, periodically agitated	ent contact - slowed, disoriented, periodically agitated	contact, oriented, mental status stable	contact, oriented, mental status stable	contact, oriented, mental status stable	contact, oriented, mental status stable
Total bilirubin, μmol/L	27.49		10.56	7.8	8.12		8.42
AST/ALT, U/L	12.8/23		19/29.75	24/35.8	16/43		10.63/25.42
ALP/GGT, U/L	162/49		141/46	143/82.8	164/104.76		131.94/66
CRP, mg/l	42.65		30.44	6.36	1.03		
Creatinine, mg/dL	165		126	100	82.17		84.7
Cyclosporine, ng/mL		141.2					
Everolimus, ng/mL				1.02	7.84	7.73	5.25
Steroid dose, mg	P 5	P 5	P 5	P 10	P 10	P 10	P 10
Immunosuppression	Cys 200 mg BD MMF 360 mg BD	Everolimus 0.25 mg BD MMF 360 mg BD	Everolimus 0.25 mg BD MMF 360 mg BD	Everolimus 1 mg BD MMF 180 mg BD	Everolimus 1 mg BD MMF 180 mg BD	Everolimus 0.75 mg BD	Everolimus 0.75 mg BD

ALT, alanine aminotransferase; AST, aspartate aminotransferase; FLP, Alkaline phosphatase; GGT, gamma-glutamyl transferase; CRP, C-reactive protein; BD, twice daily; OD, once daily; Cys, cyclosporine; MMF, mycophenolate mofetil; P, prednisolone;

Discussion

In a systematic review, posterior reversible encephalopathy syndrome (PRES) is a rare disease, with varied neurological symptoms of acute onset, vasogenic edema on neuroimaging and the recovery of clinical and radiological manifestations in post-transplant patients [2]. Bartynski W.S. et al. [3] demonstrated that the incidence of PRES among those who have received solid-organ transplantation was 0.5%, CNI-associated PRES was 0.13% [17]. PRES can present in all age groups and vary widely, however, it is more prevalent among middle-aged people, as well as according to the National Inpatient Sample database the average age of transplant recipients with PRES was 57 years, and most common in females [10].

Most reported PRES cases occur in the immediate or early post-transplant period [3,4]. Only 16.4% develop between 3 and 12 months post-transplant, as in our patient, and just 7.3% occur beyond the first year [17]. The delayed onset, five months after LT, represents one of the key distinguishing aspects of our case, underscoring that PRES may arise even when the transplantation course appears clinically stable. To date, no clear correlation has been established between the duration of CNI therapy and the timing of PRES onset.

Calcineurin inhibitors (CAIs) have become the standard of immunosuppression immediately after organ transplantation. Nevertheless, many authors reported that CAIs are associated with the development of PRES, particularly tacrolimus [9, 17-18], but this is because cyclosporine (CsA) is used less frequently after liver transplantation. Moreover, it is thought to have been caused by use of immunosuppressive and cytotoxic medications, blood pressure fluctuations,

renal failure, eclampsia or autoimmune disorders [1,5]. It has also been observed that, the highest risk of developing PRES is seen in patients with multiple risk factors. In our patient, several risk factors were present simultaneously: cyclosporine exposure, severe hypertension, and chronic kidney disease. Notably, the cyclosporine level remained within the therapeutic range. These findings are consistent with previous studies indicating that PRES can occur even when CNI levels are not elevated, as only about half of reported CNI-associated PRES cases exceed therapeutic drug ranges [17,23].

Although the pathophysiology of PRES remains uncertain, various theories have been suggested. Firstly, CNI has toxic impact on vascular endothelium, resulting in increased vascular permeability and cerebral edema. In addition, cyclosporine, due to vasoconstrictor property, can lead to impaired blood supply to the brain, especially in the posterior regions [3,5]. Dysfunction of cerebral autoregulation caused by hypertension has been suggested as a potential cause of PRES. However, since 25% of cases present with normal or low blood pressure, it is likely that sudden fluctuations in blood pressure, rather than sustained elevation, contribute to the pathogenesis of PRES [22].

PRES is characterized by neurological presentations including headache (50%), seizures (81%), encephalopathy (28%), visual disturbances (39%), focal neurological deficits (10-15%) and rarely vomiting [5,8,13], and based on a literature review, CNI-associated PRES accompanied by hypertension in many cases [10]. Presenting symptoms of our patient was consistent with those reported in previous studies. The main symptoms included encephalopathy, headache and seizures, going along with elevated arterial blood pressure.

Neuroimaging is crucial for diagnosing PRES. Computed tomography (CT) of the brain typically shows vasogenic edema in the bilateral cerebral regions supplied by the posterior circulation. However, magnetic resonance imaging (MRI) is the preferred modality, as it is more sensitive to detecting vasogenic edema as a hyperintense signal on T2-weighted and fluid-attenuated inversion recovery sequences [8,18]. MRI/CT scans changes are most commonly seen in the bilateral white matter, particularly in the parieto-occipital lobe (65-99%), although other regions less frequently may be affected, including the frontal region (54-88%), temporal region (68%), brainstem (18-27%), cerebellum and basal ganglia [12,14]. There may also be imaging abnormalities in different areas simultaneously [25]. In our case, vasogenic edema was restricted to the brainstem, an uncommon and atypical imaging pattern. Brainstem-only involvement has been reported in a minority of PRES cases, including those associated with calcineurin inhibitors such as cyclosporine; however, the exact frequency in cyclosporine-related PRES is not well established [12,14]. This atypical distribution underscores the clinical relevance of our report, highlighting that PRES can present with isolated brainstem lesions, which may be underrecognized in routine practice. Although MRI would have provided superior diagnostic detail, it could not be obtained due to the patient's hemodynamic instability and severe pain.

The differential diagnosis of PRES involves acute violation of cerebral circulation, progressive multifocal leukoencephalopathy, infections of CNS, toxic encephalopathies, and especially reversible cerebral vasoconstriction syndrome, which may present similarly to PRES or even overlap it [6]. All possible common causes were ruled out in our patient immediately after admission to our center. Therefore, based on the evaluation of the medical history, the presence of several risk factors such as immunosuppressant intake, hypertension and renal failure, typical cerebral imaging, helps in differentiating among potential diagnoses.

The primary approach to treating PRES is the discontinuation or dose reduction of the causative agent, controlling hypertension, and managing brain edema and encephalopathy [5]. The literature review indicated that switching CNIs with another CNIs or alternative immunosuppressive drugs (43.7%) was preferred strategy [4,17]. However, Heidenhain et al [19] described a case of PRES where neurological symptoms progressed after replacing to another CNIs, and the patient achieved full recovery after CNIs were completely discontinued. Based on this, they proposed stopping all CNIs in PRES cases. In addition, Lunardi et al. revealed that immunosuppression could be safely managed with everolimus and MMF in patients with PRES [7]. In this case, a significant improvement in the patient's symptoms was observed following a substitution cyclosporine to everolimus, supporting the hypothesis that this syndrome was induced by the immunosuppressant. Besides that, controlling hypertensive episodes and maintaining normal blood pressure are also key elements in the treatment of PRES [2,5]. The selection of antihypertensive medications typically follows general guidelines for managing hypertensive crises or emergencies. As in other conditions, sudden fluctuations should be prevented, and continuous antihypertensive therapy with hemodynamic monitoring is advised [27]. Also, patients often require anticonvulsant therapy. There are no standardized recommendations regarding the

choice of specific antiepileptic medications. Furthermore, appropriate treatment duration remains uncertain. In most cases, anticonvulsant drugs may be gradually discontinued once the patient becomes asymptomatic and imaging abnormalities have completely resolved [2].

The prognosis in PRES cases is usually favorable with timely diagnosis and discontinuation of the underlying cause. Nevertheless, it is important to remember that, the presence of severe complications in PRES may result in lasting neurological sequelae. Follow-up neuroimaging revealed resolution of MRI or CT changes in 81% of reported case series [2]. As demonstrated in our case, the patient achieved full recovery without long-term neurological deficits, supporting the reversibility of the syndrome with appropriate treatment.

Conclusion

Early recognition of neurological symptoms in post-transplant patients receiving calcineurin inhibitors is essential, as these clinical changes should prompt consideration of PRES even months after liver transplantation. Timely diagnosis, appropriate supportive management, and careful adjustment of immunosuppressive therapy can lead to full reversal of the syndrome while maintaining graft function. Prompt identification and control of hypertensive episodes are also critical. This case emphasizes the importance of considering PRES in the differential diagnosis of liver transplant recipients presenting with altered mental status, especially in non-specialized transplant settings.

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Statement of Ethics: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. This case report was conducted in compliance with the principles of the Declaration of Helsinki. Ethical committee approval was not required for this case report because no identifiable personal patient information (such as name, date of birth, address, photos, or any other identifying details) was included in the manuscript.

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