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Case Report

An unusual case of posterior reversible encephalopathy syndrome in post-renal transplant recipient

Anuj Thakral [a], *Rajeev Malhotra* [b], *Ankur Gupta* [c], *Manoj Khanal* [d], *Shashi Kumar* [e], *Hiralal* [f,*], *Narayan Prasad* [g]

[a] *Attending Consultant, Department of Radiology, Max Superspecialitiy Hospital, Shalimar Bagh, Delhi 110088, India*
[b] *Senior Consultant and Head, Department of Radiology, Max Superspecialitiy Hospital, Shalimar Bagh, Delhi 110088, India*
[c] *Consultant, Department of Nephrology, Max Superspecialitiy Hospital, Shalimar Bagh, Delhi 110088, India*
[d] *Consultant, Department of Neurology, Max Superspeciality Hospital, Shalimar Bagh, Delhi 110088, India*
[e] *Post Doctoral Fellow in Renal Transplantation, Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Rai Barielly Road, Lucknow, Uttar Pradesh 226014, India*
[f] *Associate Professor & Incharge of Uroradiology and Renal Transplantation, Department of Radiology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Rai Barielly Road, Lucknow, Uttar Pradesh 226014, India*
[g] *Additional Professor, Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Rai Barielly Road, Lucknow, Uttar Pradesh 226014, India*

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ABSTRACT

Patients with history of solid organ/bone marrow transplantation, chronic renal failure and hypertension are known to present with a sudden neurological deficit and a multifactorial clinicoradiological syndrome called posterior reversible encephalopathy syndrome (PRES). Occurrence of vasogenic edema is well demonstrated on magnetic resonance imaging (MRI) which tends to resolve with medical therapy. Here we report a case of post-renal transplant recipient with graft failure showing typical manifestation of PRES and its successful medical management.

1. Introduction

Posterior reversible encephalopathy syndrome (PRES), also known as reversible posterior leukoencephalopathy syndrome (RPLS), is a syndrome characterized by headache, confusion, seizures and visual loss. It may occur due to a number of causes, predominantly severe high blood pressure, eclampsia, renal failure, lupus and some medical treatments including immunosuppressive therapy with calcineurin

* *Corresponding author.*
E-mail address: hiralal2007@yahoo.co.in (Hiralal).
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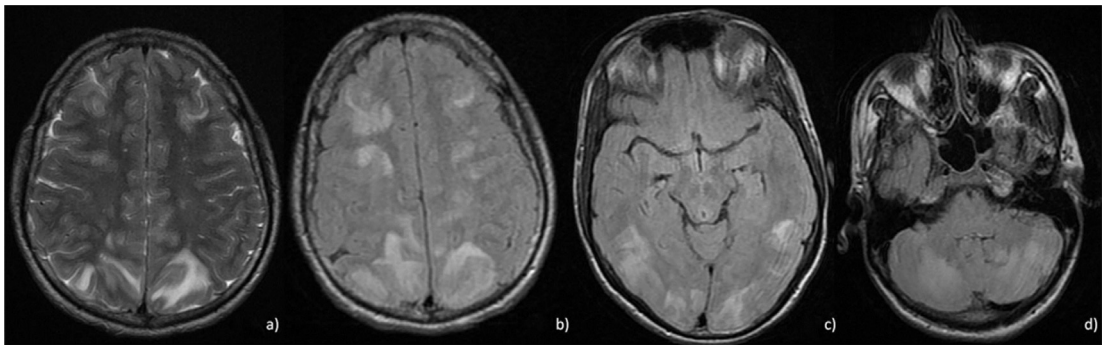

Fig. 1 – Abnormal T2 (a) & FLAIR (b–d) hyperintense signal involving the cortex and subcortical white matter in bilateral high frontal, parietal and occipital regions, bilateral cerebellar hemispheres.

inhibitors in renal transplant recipients. Here we report a case of PRES in a failed renal allograft recipient who were not on immunosuppression and developed PRES consequent upon severe hypertension and completely recovered with control of blood pressure after 4 weeks of therapy.

2. Case report

A 28 years old male renal allograft recipient, whose renal graft has failed consequent upon chronic allograft nephropathy, has been shifted on maintenance hemodialysis (MHD) for 12 months. After shifting to MHD, all his immune-suppressants, Cyclosporine and Mycophenolate mofetil were stopped, however 5 mg of prednisolone was continued. He presented in emergency with headache and generalized weakness for 2 days and an episode partial seizure followed by generalized tonic clonic seizure with accelerated hypertension. He had received intravenous Phenytoin and Levetiracetam before coming to emergency.

On general examination, he was afebrile, and had respiratory rate of 32/min, pulse rate 138 beats per min, and BP of 180/90 mm of Hg. On neurological examination, the patient was drowsy but moving all four limbs. The pupils showed sluggish reaction to light with non responsive plantar reflexes. There was no neck stiffness. His biochemical parameters revealed Blood urea 84 mg/dl, Serum Creatinine 9.71 mg/dl, Na^{+} 133 mg/dl, K^{+} 4.6 mg/dl, Ca^{++} 9.1 mg/dl and Mg^{++} 2.5 mg/dl.

To evaluate for the cause of seizures, magnetic resonance imaging (MRI) of the brain was performed. MRI Brain revealed abnormal T2 & FLAIR hyperintense signal involving the cortex and subcortical white matter in bilateral high frontal, parietal and occipital regions (Fig. 1a–c). Similar signal changes were also seen involving the right half of thalamus and both cerebellar hemispheres (Fig. 1d). The distribution of MRI findings was typically suggestive of posterior reversible encephalopathy syndrome (PRES). While none of these areas showed restriction of diffusion and were consistent with vasogenic edema, focal area of restricted diffusion was seen in the right parietal convexity cortex consistent with cytotoxic edema (Fig. 2).

Patient was given one session of hemodialysis. With Intravenous Labetalol and Benazepril, blood pressure was controlled and seizure also subsided. The patient improved neurologically by day 3 with normal level of consciousness and unremarkable neurological examination. Patient was discharged the next morning in stable condition. He was advised

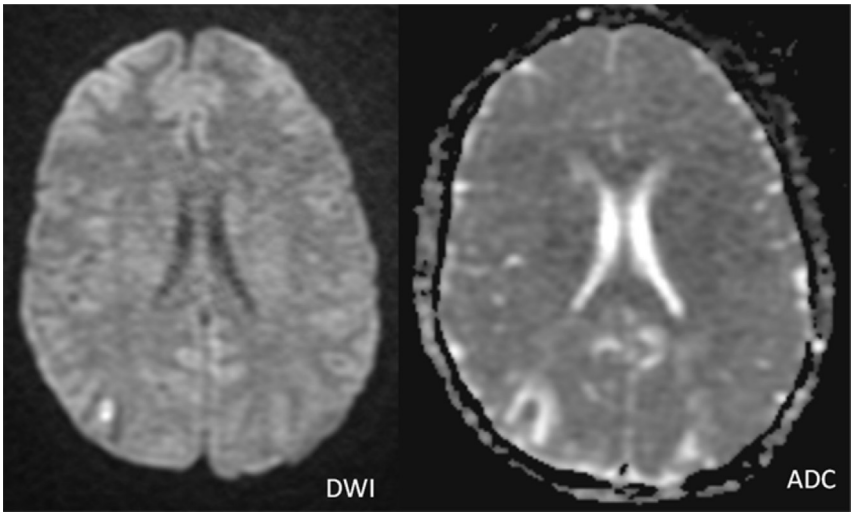

Fig. 2 – Focal area of restricted diffusion in right parietal convexity cortex suggestive of acute infarction.