

Posterior Reversible Encephalopathy Syndrome during Recovery from Hypovolemic Acute Kidney Injury after Trauma; Case Report and Literature Review

Richa Aggarwal¹*, Anudeep Saxena², Kapil Soni¹

¹Critical and Intensive Care Medicine, Jpn Apex Trauma Centre, All India Institute of Medical Sciences, New Delhi, India ²Sir Ganga Ram Hospital, New Delhi, India

*Corresponding author: Richa Aggarwal Address: Associate Professor of Critical and Intensive Care, Division of trauma surgery and critical care, JPNA Trauma center, AIIMS, New Delhi, 110029, India. Tel: +91-987-3731042; Fax: +91-986-8398612 e-mail: pathakricha@yahoo.co.in **Received:** December 28, 2016 **Revised:** May 8, 2017 **Accepted:** May 17, 2017

ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a rare clinicoradiological entity characterized by neurological symptoms. It is associated with various conditions like hypertension, renal diseases and use of cytotoxic agents. It occurs more often in adults than children. PRES has been described in pediatric patient with renal diseases like nephrotic syndrome, nephritis and in acute renal failure as in cases of Hemolytic-uremic syndrome but there are no reports of PRES in cases of recovery from acute kidney injury due to prerenal cause like hypovolemia. We herein present an interesting case of 6-year-old girl with traumatic amputation who developed PRES days after recovery of acute kidney injury. The patient was successfully managed medically. The presented clinical scenario demonstrates the fact that PRES can develop in a trauma patient in acute setting of recovering from hypovolemic acute kidney injury. Prompt recognition and treatment is important and can lead to complete recovery.

Keywords: Posterior reversible encephalopathy syndrome (PRES); Hypertension; Acute kidney injury (AKI); Trauma.

Please cite this paper as:

Aggarwal R, Saxena A, Soni K. Posterior Reversible Encephalopathy Syndrome during Recovery from Hypovolemic Acute Kidney Injury after Trauma; Case Report and Literature Review. *Bull Emerg Trauma*. 2017;5(3):215-218.

Introduction

P(PRES) is a rare clinicoradiological entity characterized by neurological symptoms like headache, seizures, and altered consciousness level and visual abnormalities [1]. It can occur in all age groups but more commonly seen in adults.

The estimated incidence in pediatric critical care unit is 0.4% [2]. In adults it is most commonly associated with conditions like hypertension, preeclampsia, renal diseases, autoimmune diseases and treatment with immunosuppressive drugs [3]. However, in children, PRES is mostly described with renal and hematological disorders [4] which include patients with nephrotic syndrome, acute nephritis and patients with malignancies treated with chemotherapeutic agents [5]. It has also been reported in patients with haemolytic uremic syndrome [HUS] and adrenocortical diseases [6]. The presentation of PRES in the settings of trauma is very rare. We herein present a 6-year-old girl with traumatic amputation who developed PRES, several days after recovery of acute kidney injury (AKI).

Case Report

A 6-year-old girl was brought to our emergency department following road traffic accident. On arrival, she had a patent airway but laboured breathing and was in shock with vitals of heart rate 154/min, and blood pressure 50/30 mmHg. She had undergone above knee traumatic amputation of right lower limb which was bleeding. She was immediately intubated and put on mechanical ventilation. Resuscitation was started with crystalloids, blood and blood products. The actively bleeding femoral vessels were ligated and primary wash and dressing was done. She responded well to initial resuscitation and became hemodynamically stable. There were no injuries in chest and abdomen. As per protocol, non-contrast computed tomography (NCCT) of brain and cervical spine was performed which were unremarkable. She was transferred to intensive care unit (ICU) intubated in view of massive blood loss and metabolic acidosis and resuscitation continued in the ICU. She received 5 units of packed RBCs, 3 units of fresh frozen plasma (FFP), 3 units of platelets and 3 units of cryoprecipitate in the first 24 hours. Next day her creatinine increased from 0.9 mg/dl to 1.7 mg/dl with decrease in urine output. On examination, there was significant oozing from the wound and she was given fluid boluses with the guide of central venous pressure monitoring following which creatinine started decreasing. Over a period of 3 days, serum creatinine values decreased to 0.6mg/

dl and she was extubated. After 3 days in ICU, her blood pressure started keeping up on hypertensive side, ranging from 150/90 to 160/100mmHg. Despite adequate pain control, she remained hypertensive for which oral amlodipine was started (5mg twice a day). While she was being evaluated for hypertension, she developed 2 episodes of seizures in a day. The first one was a left side focal seizure and second was generalized tonic-clonic convulsions and she was re-intubated. NCCT brain was done which revealed bilateral multiple hypo-dense foci in white matter in all frontal, parietal, temporal and occipital lobes, with parieto-occipital predominance (Figures 1). These radiographic changes along with the clinical presentation and co-existing hypertension suggested the diagnosis of posterior reversible encephalopathy syndrome. She was started on phenytoin for seizures and labetolol and enalapril were added to control the BP to the target value of 90th percentile for her age, i.e 105/68. Over a period of 3-4 days her consciousness level improved to normal. She was extubated and transferred to the ward. All the investigations of hypertension were unremarkable. Contrast enhanced CT of abdomen revealed normal kidney size and renal arterial dimensions. Renal Doppler was also normal. Urine routine microscopy was unremarkable. Volume overload was ruled out clinically and by input output charts. A table correlating the date, BP, creatinine values and previous day volume balance with seizures is provided (Table 1). She recovered fully neurologically in 3-4 days. At the time of discharge, she had regained her preinjury status. Further during the follow up visit, she was absolutely normal with no neurological deficit.

Discussion

PRES is a rare entity. Various triggers for PRES in children are hypertension, renal disease and immunosuppressive agents. The underlying

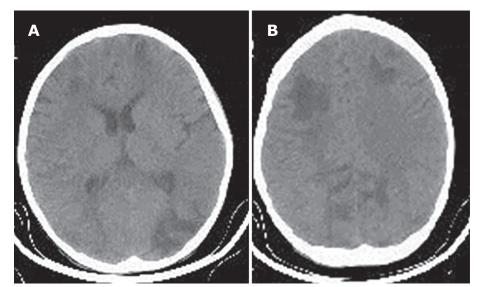


Fig. 1. The axial non-contrast brain CT-Scan demonstrating hypodensity involving white and grey matter of bilateral occipital lobes (A) and bilateral frontal lobes (B).

Date	Blood Pressure	Serum Urea	Serum Creatinine	Volume Balance
1 st day	140/106	10	0.5	-40ml
2 nd day	150/100	8	0.3	-75ml
3 rd day (Seizures)	138/102	11	0.4	+75 ml

pathophysiology of PRES is breakdown in cerebral autoregulation which leads to hyperperfusion and vasogenic edema of cerebral white matter [7]. Endothelial dysfunction has also been implicated as pathogenic mechanism in cases of preeclampsia or cytotoxic drugs [8].

There are various case reports and case series published about PRES in children [3, 9, 10,]. All series show boys are more frequently affected than girls. The majority of reported cases were either patients with renal diseases like nephrotic syndrome, glomerulonephritis, AKI due to renal cause or patients with tumors and transplant recipient on drugs. Gera et al [3] described 11 paediatric patients with PRES out of which 6 patients had acute renal failure because of renal disease per se [4 had HUS, 1 case of glomerulonephritis and 1 had obstructed solitary kidney]. None had AKI due to prerenal cause. Similarly, Ishikura et al., [9] studied 20 paediatric patients who developed PRES out of which 10 patients were kidney transplant recipients, 7 had nephrotic syndrome and 3 had glomerulonephritis. There are no reported cases of PRES because of acute renal failure attributed to prerenal cause like hypovolemic shock. Our patient was a 6 year old girl child who had no history or symptoms of any renal disease but developed acute kidney injury because of hypovolemic shock which was recovering. She was not a known hypertensive but developed acute rise in blood pressure in ICU over 3 days and developed PRES. We investigated her for hypertension but did not find any conclusive findings and posttraumatic stress was kept as a possibility. We are uncertain why PRES developed in the renal function recovery phase. As in other case series, 9 of 10 cases from PICU [2], our patient also presented with seizures and altered consciousness level. There have been few case reports of PRES in trauma patients. Sigurta et al., [11] reported PRES in a15 year old case of traumatic pancreatitis. In this case, both hypertension and endothelial dysfunction caused by sepsis were triggers for PRES. Another case of young patient with pelvic trauma has been described who developed PRES because of immunoglobulin infusion [12].

The diagnosis of PRES is usually made clinically

with underlying conditions and with supportive finding on imaging. MRI is more sensitive than CT scan. The characteristic imaging pattern is presence of edema involving white matter of posterior portions of both cerebral hemispheres especially the parietooccipital regions in a symmetric manner [1]. However other structures such as brainstem, cerebellum, frontal and temporal lobes may also get involved [13]. In our case, both parietooccipital regions and frontal lobes were involved as depicted in CT scan. The differential diagnosis of such findings include progressive multifocal leukoencephalopathy, posterior circulation stroke and hypoxic -ischemic encephalopathy. These were excluded by history and clinical findings. Hypoxic event was ruled out seeing the patient's records which are recorded every hour in ICU. Posterior circulation stroke is usually unilateral but our patient had characteristic bilateral involvement. Progressive multifocal leukoencephalopathy (PML) is a demyelinating disease which results from the reactivation of John Cunningham virus (JC virus) infecting oligodendrocytes in patients with compromised immune systems. There was no acute bleeding event as CT scan did not show any hemorrhage. Our patient recovered fully neurologically in 3-4 days further strengthening our diagnosis of PRES. We did not get a repeat CT scan after her improvement because that would have required deep sedation or general anesthesia for the child. At the time of discharge, she had regained her preinjury status. To the best of our knowledge, this is a rare case of PRES during recovery from AKI due to prerenal cause and points to the importance of managing hypertension in posttrauma state. Other causes of PRES were also ruled out. Blood transfusion was done initially only at the time of admission to ICU but seizures occurred after 8 days of injury.

In conclusion, the presented clinical scenario demonstrates the fact that PRES can develop in a trauma patient in acute setting of recovering from hypovolemic AKI. Prompt recognition and treatment is important and can lead to complete recovery.

Conflicts of Interest: None declared.

References

- Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334(8):494-500.
- 2. Raj S, Overby P, Erdfarb A, Ushay HM. Posterior reversible encephalopathy syndrome: incidence and associated factors in a pediatric critical care population. *Pediatr*

Neurol. 2013;49(5):335-9.

3. Gera DN, Patil SB, Iyer A, Kute VB, Gandhi S, Kumar D, et al. Posterior reversible encephalopathy syndrome in children with kidney disease. *Indian J Nephrol.* 2014;**24**(1):28-34.

- Yamamoto H, Natsume J, Kidokoro H, Ishihara N, Suzuki M, Tsuji T, et al. Clinical and neuroimaging findings in children with posterior reversible encephalopathy syndrome. *Eur J Paediatr Neurol.* 2015;19(6):672-8.
- 5. Incecik F, Herguner MO, Altunbasak S, Erbey F, Leblebisatan G. Evaluation of nine children with reversible posterior encephalopathy syndrome. *Neurol India*. 2009;**57**(4):475-8.
- Lodish M, Patronas NJ, Stratakis CA. Reversible posterior encephalopathy syndrome associated with micronodular adrenocortical disease and Cushing syndrome. *Eur J Pediatr.* 2010;169(1):125-6.
- 7. Granata G, Greco A, Iannella G,

Granata M, Manno A, Savastano E, et al. Posterior reversible encephalopathy syndrome--Insight into pathogenesis, clinical variants and treatment approaches. *Autoimmun Rev.* 2015;**14**(9):830-6.

- 8. Al-Jameil N, Aziz Khan F, Fareed Khan M, Tabassum H. A brief overview of preeclampsia. *J Clin Med Res.* 2014;6(1):1-7.
- 9. Ishikura K, Ikeda M, Hamasaki Y, Hataya H, Shishido S, Asanuma H, et al. Posterior reversible encephalopathy syndrome in children: its high prevalence and more extensive imaging findings. *Am J Kidney Dis.* 2006;**48**(2):231-8.
- **10.** Kwon S, Koo J, Lee S. Clinical spectrum of reversible posterior

leukoencephalopathy syndrome. *Pediatr Neurol.* 2001;**24**(5):361-4.

- Sigurta A, Terzi V, Regna-Gladin C, Fumagalli R. Posterior Reversible Encephalopathy Syndrome Complicating Traumatic Pancreatitis: A Pediatric Case Report. *Medicine* (*Baltimore*). 2016;95(22):e3758.
- Ortega-Carnicer J, Ambros A, Diarte JI. Reversible posterior leukoencephalopathy syndrome in a young trauma patient. *Resuscitation*. 2005;64(1):119-20.
- Lamy C, Oppenheim C, Meder JF, Mas JL. Neuroimaging in posterior reversible encephalopathy syndrome. *J Neuroimaging*. 2004;14(2):89-96.