EURASIAN JOURNAL OF EMERGENCY MEDICINE
Case Report
DOI: 10.4274/eajem.galenos. 2026.81904
Eurasian J Emerg Med. 2026;25: 171-4

Hypertensive Encephalopathy in the Emergency Department, a Case of Posterior Reversible Encephalopathy Syndrome (PRES)

Jeremy Zhenwen Pong, Aliviya Dutta, Jeevan Raaj Thangayah

Singapore General Hospital, Department of Emergency Medicine, Singapore

Abstract

Hypertension is commonly encountered in the emergency department. While emergency physicians are understandably concerned about hypertensive emergencies, such occurrences are rare. Here, we present a case report of hypertensive encephalopathy causing posterior reversible encephalopathy syndrome (PRES), which manifested in a patient as altered mental status and seizures. We also discuss risk factors for PRES, common presentations, investigations, management, and PRES as a crucial consideration in patients with hypertension and altered mental status, given differences in management compared with other differential diagnoses. With timely recognition and treatment, most patients with PRES recover completely within several days.

Keywords: Hypertension, hypertensive encephalopathy, hypertensive emergency, posterior reversible encephalopathy syndrome, emergency department

Introduction

Hypertension is commonly encountered in the emergency department (ED), either as a presenting complaint or as an incidental finding at triage. A hypertensive emergency is defined as a potentially life-threatening condition with end-organ damage secondary to markedly elevated blood pressure (1). This commonly occurs when blood pressure rises above 180/120 mmHg, causing end-organ injury in the ophthalmic, renovascular, cardiovascular, or cerebrovascular systems (2). Such patients may present with visual deficits, chest pain, shortness of breath, limb numbness or weakness, altered mental status, seizures, or even coma. Although emergency physicians are understandably concerned about hypertensive emergencies, such occurrences are rare and were found to account for less than 2% of all hypertensive cases in the ED (3,4). Hypertensive encephalopathy causing posterior reversible encephalopathy syndrome (PRES) is even rarer, poisted to result from hypertension-induced autoregulatory failure leading to cerebral edema that preferentially affects the parietal and occipital lobes (5). Distinguishing PRES from other diagnoses (e.g. ischemic stroke) is crucial because subsequent management differs and can significantly affect patient outcomes. Here, we present a case of PRES manifesting as altered mental status and seizures.

Case Report

Ms A was a 65-year-old woman with a history of diabetes mellitus, hypertension, and hyperlipidemia. While vacationing aboard a cruise ship, she failed to disembark when the ship docked. Ms A was subsequently found semiconscious in her cabin with fecal incontinence. Assessment by the onboard cruise doctor noted that she was drowsy, had a blood pressure of 227/105 mmHg, and a capillary blood glucose level of 27 mmol/L (486 mg/dL). She was transferred via ambulance to the hospital.

On arrival at the ED, Ms A was afebrile and hypertensive, with a blood pressure of 169/87 mmHg, a heart rate of 82, a respiratory rate of 18, and an oxygen saturation of 96% on room air. Though drowsy, she provided a brief history of feeling unwell the day before, although she was unable to specify exact symptoms. Notably, she admitted to omitting her chronic medications for diabetes and hypertension for one week (metformin 1000 mg twice daily and telmisartan 40 mg once daily in the morning).

Corresponding Author: Jeremy Zhenwen Pong MD, Singapore General Hospital, Department of Emergency Medicine, Singapore
E-mail: jeremypongzw@gmail.com **ORCID ID:** orcid.org/0000-0001-6011-0584

Cite this article as: Pong JZ, Dutta A, Thangayah JR. Hypertensive encephalopathy in the emergency department, a case of posterior reversible encephalopathy syndrome (PRES). Eurasian J Emerg Med. 2026;25: 171-4.

Received: 24.12.2025
Accepted: 19.01.2026
Published: 06.02.2026

On physical examination, Ms A was drowsy but rousable, with a Glasgow coma scale (GCS) score of 13 (E3 V4 M6). Fecal incontinence was noted. Examination of the heart, lungs, and abdomen were unremarkable, and no overt neurological deficits or injuries were found.

Laboratory investigations showed an elevated serum glucose concentration of 25.1 mmol/L (452 mg/dL) (reference range: 3.9-11 mmol/L), with serum ketones marginally elevated at 0.8 mmol/L (reference range: 0-0.6 mmol/L), and venous blood gas pH and bicarbonate normal at 7.35 (reference range: 7.35-7.45) and 23 mmol/L (reference range: 19-29 mmol/L) respectively. There were mildly elevated transaminases (aspartate aminotransferase 76 U/L, reference range 12-42 U/L; alanine transaminase 194 U/L, reference range 6-66 U/L), markedly elevated alkaline phosphatase (1729 U/L, reference range 39-99 U/L), and normal bilirubin (21 µmol/L, reference range 7-32 µmol/L). The remainder of the full blood count and the renal panel with extended electrolytes were otherwise unremarkable. Bedside ultrasound examinations of the heart and abdomen were grossly normal. A computed tomography scan of the brain demonstrated small hypodensities in the right frontal lobe that were of indeterminate age which may have represented chronic lacunar infarcts. While still in the ED, Ms A became tremulous and developed asterixis. She subsequently experienced a witnessed generalised tonic-clonic seizure, which was swiftly aborted with 5 mg of intravenous diazepam. Systolic blood pressure for the patient was by now trending consistently above 220 mmHg.

Clinical impression was that of altered mental status possibly secondary to PRES, hyperlgycemia not in crisis, and deranged liver enzymes for investigation. An intravenous nicardipine infusion was started for blood pressure control, as was insulin therapy for the hyperglycemia, and levetiracetam for seizure control. Consequently, the patient appeared more alert as blood pressure began to decline, though she remained slightly confused with a GCS of 14 (E4, V4, M6).

In view of the patient's persistent drowsiness, seizure, and elevated blood pressure requiring intravenous antihypertensives, the Neurology department was informed with a view toward an early inpatient magnetic resonance imaging (MRI) of the brain. An MRI brain scan performed the following day showed findings suggestive of PRES: areas of cortical and subcortical white matter signal changes with associated swelling, predominantly in the posterior distribution, involving both occipital lobes (Figure 1a). Though Ms A's usual antihypertensive medication was initially held off on admission, she was restarted on them after the MRI diagnosis of PRES was confirmed. Ms A underwent a repeat brain MRI approximately two weeks after the initial MRI scan, with repeat MRI demonstrating near-complete resolution of the previously noted hyperintense areas (Figure 1b). She also made a full clinical recovery.

Discussion

PRES was named for the radiological and clinical features of white matter edema in the posterior cerebrum on brain MRI, and its potential for neurological symptoms to be reversed should prompt and appropriate treatment be initiated (6). The mechanism by which PRES and cerebral swelling result in

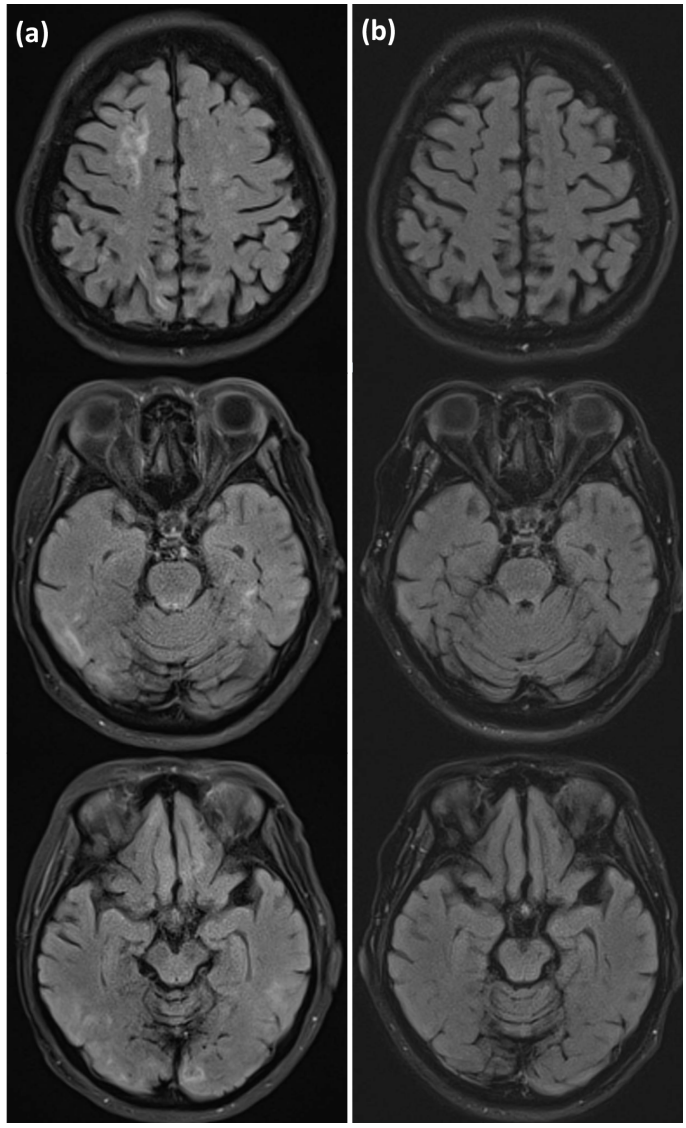

Figure 1. (a) Magnetic resonance imaging (MRI) of the brain on patient presentation, showing areas of T2W/FLAIR hyperintensity in the subcortical white matter and cortex predominantly in a posterior distribution involving both occipital lobes, which may suggest posterior reversible encephalopathy syndrome. Similar signal changes in the right temporal and bilateral frontal lobes. (b) Near resolution of T2W/FLAIR hyperintense areas on repeat MRI two weeks later.

neurological dysfunction is not well understood, but is postulated to occur when excessive hypertension damages the endothelial lining of the blood-brain barrier, leading to cerebral edema. This posterior predilection may reflect reduced autoregulatory capacity, supporting the "vasogenic" theory of PRES (7). Though PRES commonly occurs as a complication of hypertension, it has also been reported in normotensive patients who are immunosuppressed (e.g. transplant recipients), in patients with sepsis, in those with impaired renal function, in autoimmune conditions, and in association with blood transfusion and drugs of abuse. In these instances, cerebral swelling is postulated to result from cytotoxicity or autoimmune attack on cerebral blood vessels (6,8). This represents the "toxic theory" of PRES.

Recognition of PRES as the cause of neurological disturbance in the ED requires a high index of suspicion because the differential diagnoses for patients presenting with altered mental status are broad, and initial history and investigations may be unrevealing. This is important, as the management of PRES differs from that of ischemic stroke, another common cause of altered mental status in the ED. In the former, we aim to reduce blood pressure, whereas in the latter, permissive hypertension is allowed. This presents a conundrum, and the clinical features of each case are therefore paramount in determining the most likely etiology of the patient's altered mental status.

What then are the clinical predictors of PRES? In a retrospective cohort study of 220 patients presenting with acute neurological symptoms, statistically significant associations were found between PRES and symptoms such as epileptic seizures, encephalopathy, visual disturbances, as well as with risk factors such as hypertension, chemotherapy, and renal failure (9). A model using the above clinical parameters for prediction of PRES achieved an accuracy (area under the curve) of 0.793 (9). Patients with medical conditions such as systemic lupus erythematosus with renal involvement, glomerulonephritis, and patients receiving immunosuppressive treatment for transplantation or rheumatologic conditions were also at higher risk for developing PRES. A review article on the prevalence of symptoms and signs in patients with PRES noted that seizures (60-75%), encephalopathy (50-80%), headache (50%), visual disturbances (33%), focal neurological deficits (10-15%), and status epilepticus (5-15%) were the most common clinical manifestations (10). As the symptoms and signs of PRES are non-specific and can also be observed in a variety of other disorders, clinical judgment is critical for rapid identification and management. An expedited MRI of the brain should be strongly considered when the clinical suspicion of PRES is high.

Treatment of PRES revolves around the identification and removal of precipitating factors where possible (chemotherapy drugs, immunosuppressants), management of hypertension with titratable intravenous antihypertensives such as nicardipine or labetalol, aiming for a gradual reduction in blood pressure of approximately 25% from the presenting value over the first few hours, and antiepileptic medications for seizures if present (10). In studies examining the prognosis of patients with PRES, 75% to 90% of patients made a full recovery, with most recovering within two to eight days (10,11).

Conclusion

Hypertension and altered mental status are common presentations to the ED. The diagnosis of PRES or hypertensive encephalopathy requires a high index of suspicion and should be considered in patients with risk factors such as hypertension, chemotherapy administration, or renal failure, presenting with common symptoms and signs of PRES, such as seizures or status epilepticus, encephalopathy, headache, visual disturbances, or focal neurological deficits. In such patients, a brain MRI should be performed as it most accurately depicts disease-specific neuroradiological findings and helps to guide subsequent management. With timely recognition and treatment, most patients with PRES exhibit a complete recovery in several days.

Ethic

Informed Consent: This is a retrospective study.

Footnotes

Author Contributions

Surgical and Medical Practices: J.Z.P., A.D., J.R.T., Concept: J.Z.P., A.D., J.R.T., Design: J.Z.P., A.D., J.R.T., Data Collection or Processing: J.Z.P., A.D., J.R.T., Analysis or Interpretation: J.Z.P., A.D., J.R.T., Literature Search: J.Z.P., Writing: J.Z.P., A.D., J.R.T.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Potter T, Schaefer TJ. Hypertensive encephalopathy. StatPearls. Treasure Island FL: 2023, StatPearls Publishing LLC. 2023.
2. Miller J, McNaughton C, Joyce K, Binz S, Levy P. Hypertension management in emergency departments. Am J Hypertens. 2020;33:927-34.
3. Johnson W, Nguyen ML, Patel R. Hypertension crisis in the emergency department. Cardiol Clin. 2012;30:533-43.
4. Suneja M, Sanders ML. Hypertensive emergency. Med Clin North Am. 2017;101:465-78. Epub 2017 Mar 2.
5. Tetsuka S, Ogawa T. Posterior reversible encephalopathy syndrome: a review with emphasis on neuroimaging characteristics. J Neurol Sci. 2019;404:72-9. Epub 2019 Jul 17.

6. Zelaya JE, Al-Khoury L. Posterior reversible encephalopathy syndrome. 2024. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–.
7. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. Mayo Clin Proc. 2010;85:427-32.
8. Triplett JD, Kutlubaev MA, Kermode AG, Hardy T. Posterior reversible encephalopathy syndrome (PRES): diagnosis and management. Pract Neurol. 2022;22:183-9.
9. Faille LD, Fieuws S, Van Paesschen W. Clinical predictors and differential diagnosis of posterior reversible encephalopathy syndrome. Acta Neurol Belg. 2017;117:469-75.
10. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. Lancet Neurol. 2015;14:914-25. Epub 2015 Jul 13. Erratum in: Lancet Neurol. 2015;14:874.
11. Liman TG, Bohner G, Endres M, Siebert E. Discharge status and in-hospital mortality in posterior reversible encephalopathy syndrome. Acta Neurol Scand. 2014;130:34-9. Epub 2013 Dec 13.