Acta Neurochirurgica (2019) 161:217–224
https://doi.org/10.1007/s00701-019-03798-4

CASE REPORT - NEUROSURGERY AND PREGNANCY

Post-partum posterior reversible encephalopathy syndrome requiring decompressive craniectomy: case report and review of the literature

Gennadiy A. Katsevman[1] · Ryan C. Turner[1] · Cletus Cheyuo[1] · Charles L. Rosen[1] · Matthew S. Smith[2]

Received: 23 August 2018 / Accepted: 4 January 2019 / Published online: 18 January 2019

Abstract

Posterior reversible encephalopathy syndrome (PRES) is an uncommon but potentially devastating syndrome if not recognized and treated appropriately. As the name implies, recognition of the condition and proper management may reverse the clinical and radiological findings. However, diagnosis is not always straightforward. We present the case of a 24-year-old female who was 4 days post-partum and presented with headache, neck pain, and new-onset seizures. She had undergone epidural anesthesia during labor, and initial imaging was suggestive of intracranial hypotension versus pachymeningitis. Despite initial conservative therapy including anti-epileptic drugs, magnesium therapy, empiric antibiotics, and Trendelenburg positioning, the patient continued to deteriorate. Follow-up imaging was suggestive of PRES with signs of intracranial hypertension. The patient underwent a decompressive suboccipital craniectomy for refractory and severe PRES and later fully recovered. This case highlights the sometimes difficult diagnosis of PRES, possible association with pregnancy, eclampsia/preeclampsia and/or cerebrospinal fluid drainage, and the rare but life-saving need for decompression in severe cases.

Keywords Posterior reversible encephalopathy syndrome · Craniectomy · Eclampsia · Epidural

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic state in which subcortical vasogenic edema occurs within the brain resulting in neurologic sequelae. This syndrome is generally diagnosed via a constellation of clinical, neurological, and radiographic findings. Computed tomography (CT) and magnetic resonance imaging (MRI) typically demonstrate focal regions of symmetric hemispheric edema, most commonly in the parietal and occipital lobes resembling the brain watershed zones. PRES has been associated with seizures, encephalopathy, headache, visual impairment, and generally occurs in settings of acute renal failure, hypertension, cytotoxic medications, autoimmune disease, or pregnancy-related disorders (pre-eclampsia or eclampsia) [20]. At times, PRES has been reported in obstetric patients without signs of eclampsia or in the late post-partum period [59, 60, 71]. Although uncommon, PRES can also present with intracerebral hemorrhage, generally in the setting of coagulopathy or bleeding diathesis [3].

Studies demonstrate that full recovery following PRES occurs in 75–90% of patients [13, 20, 39, 42, 63]. Treatment for PRES is typically centered on the underlying cause (e.g., seizures or hypertension) and its associated symptoms [20]. Recently, multiple cases of refractory and severe PRES have been reported involving profound posterior fossa edema and brainstem compression, acute hydrocephalus, and/or diffuse cerebral edema with evidence of increased intracranial

This article is part of the Topical Collection on *Neurosurgery and pregnancy*

✉ Matthew S. Smith
mssmith@hsc.wvu.edu

Gennadiy A. Katsevman
gekatsevman@hsc.wvu.edu

Ryan C. Turner
rcturner@hsc.wvu.edu

Cletus Cheyuo
clcheyuo@hsc.wvu.edu

Charles L. Rosen
brnsrgn@gmail.com

[1] Department of Neurosurgery, West Virginia University, Morgantown, WV, USA

[2] Department of Neurology and Neurosurgery, Robert C. Byrd Health Sciences Center, West Virginia University, Morgantown, WV 26506-9180, USA

pressure [20, 24, 35]. Relatively few cases (< 10) have been reported that required neurosurgical intervention in the form of decompressive craniectomy, and most of those have been for hemorrhagic or pediatric-associated PRES [1, 2, 5, 24, 45].

We present a case of severe PRES associated with recent pregnancy and possible diagnosis of eclampsia that required surgical decompression. This case is remarkable for a multitude of reasons including the requirement for operative decompression, particularly in a non-hemorrhagic and non-pediatric case, possible association with eclampsia, and the overall unusual presentation with a differential diagnosis including both intracranial hypotension and pachymeningitis in the setting of recent epidural anesthesia. In addition to the illustrative case, the authors provide a review of the literature related to PRES to increase awareness of potentially complicated presentations and subsequent management options.

Case presentation

A 24-year-old gravida-3-para-3 Caucasian female 4 days postpartum after an uneventful spontaneous vaginal delivery presented to the emergency department (ED) with headaches, neck stiffness, seizure, and vomiting. The patient denied having preeclampsia/eclampsia during pregnancy and was normotensive upon arrival. She had epidural anesthesia during labor and experienced immediate headache and neck pain which persisted after delivery with the addition of nausea and vomiting, creating suspicion for a "wet tap" or dural puncture. The headaches were exacerbated on sitting up and relieved with recumbency. On suspicion for cerebrospinal fluid (CSF) leakage from the epidural, a blood patch was performed at the outside facility, and the patient was subsequently discharged home on post-partum day 2. However, the patient's headaches persisted and progressed, leading to presentation to our institution, where she developed a grand mal seizure that was aborted with lorazepam.

On exam, the patient was drowsy and hyperreflexic but otherwise non-focal, without Kernig's and Brudzinki signs. Early diagnostic considerations included eclampsia, intracranial hypotension, PRES, and meningitis. Initial work-up showed mild leukocytosis (white blood cell count of 15,200/μL), sodium of 144 mmol/L, potassium 3.1 mmol/L, magnesium 1.7 mg/dL, and negative HELLP (Hemolysis, Elevated Liver Enzymes and Low Platelet count) syndrome panel. MRI of the brain demonstrated dural enhancement, concerning for intracranial hypotension versus pachymeningitis (Fig. 1). The patient was admitted to the intensive care unit. Treatments for intracranial hypotension and meningitis were initiated, including Trendelenburg position, intravenous fluids, and broad spectrum antibiotics per infectious disease recommendations including vancomycin, cefepime, ampicillin, and acyclovir. A lumbar puncture, performed under fluoroscopy prior to starting antibiotics, showed

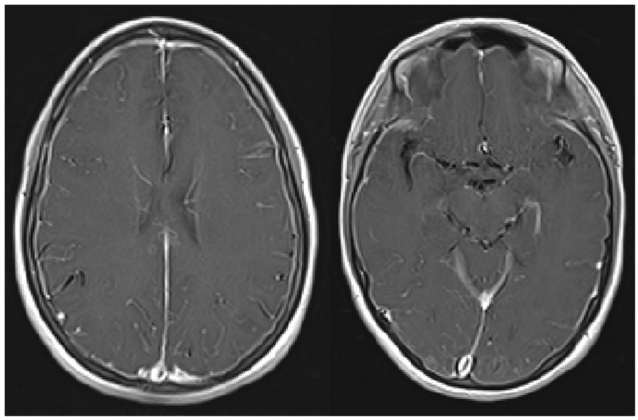

Fig. 1 MRI brain with contrast, showing diffuse meningeal enhancement concerning for intracranial hypotension versus pachymeningitis

no growth on cultures and normal opening pressure of 9 cm of water. Three milliliters of CSF were collected; glucose was normal at 53 mg/dL, protein elevated at 131 mg/dL, red blood cells elevated at 550 per microliter, and nucleated cells elevated at 9/μL. Anesthesiology declined performing a blood patch due to possible meningitis. Electroencephalography showed seizures, and patient was treated with levetiracetam 500 mg twice daily. Magnesium 1 g every 8 h with goal 3–4 mg/dL was started due to concern for preeclampsia/eclampsia until a definitive diagnosis was made. Magnesium counteracts calcium-dependent arterial vasoconstriction and is the mainstay of treatment in pregnant women to increase vasodilation, increase cerebral blood flow, and decrease ischemic insult [61]. The patient was also started on 3% hypertonic saline with sodium goal greater than 150 mmol/dL and dexamethasone 10 mg every 6 h in an effort to decrease cerebral edema. Multi-disciplinary teams were involved in patient's management, including neurology, neurosurgery, obstetrics and gynecology, anesthesiology, and infectious disease.

Despite optimum medical management, the patient's mental status deteriorated, and CT scan of the brain demonstrated diffuse effacement of the basilar cisterns, uncal herniation and sagging of the brainstem (Fig. 2). MRI/MR angiography revealed bilateral occipital edema suggestive of PRES without vessel occlusion or thrombosis (Fig. 3). The patient was taken to the operating room emergently for posterior fossa decompression via suboccipital craniectomy and upper cervical laminectomy (C1 and partial C2) with expansion duraplasty. A cortical biopsy performed during surgery was negative for vasculitis. Post-operatively, the patient was started on a 21-day dexamethasone taper, 14-day course of nimodipine (a cerebral selective calcium-channel blocker) 60 mg every 4 h for vasospasm prevention, and hypertonic saline treatment was weaned off. She made a complete recovery and was discharged home on postoperative day 5.

The patient was re-admitted with a pseudomeningocele and CSF leak on postoperative day 18, requiring incision and

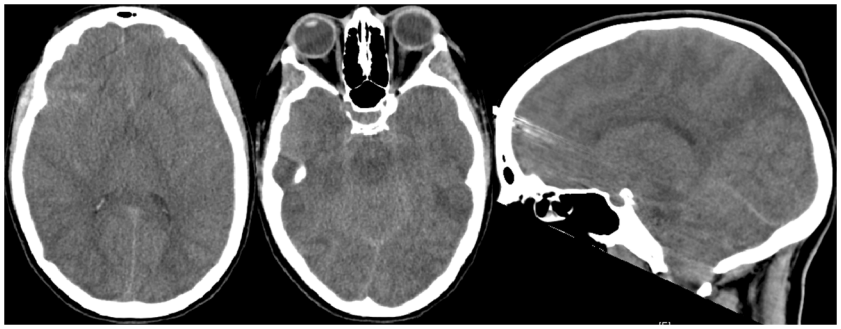

Fig. 2 CT brain without contrast, showing effacement of sulci and basal cisterns and sagging of brainstem

drainage, water-tight dural closure, and lumbar drain placement. CSF from surgery demonstrated low glucose (48 mg/dL), high protein (67 mg/dL), elevated red blood cells (380/μL), and elevated nucleated cells (585/μL). Intraoperative cultures grew coagulase negative Staphylococcus. She was treated with 6 weeks of intravenous vancomycin. Despite the interventions, the pseudomeningocele increased in size suggesting hydrocephalus, which may have resulted secondary to PRES or the recent meningitis. Due to the concern for hydrocephalus, a ventriculoperitoneal shunt was placed after the CSF was cleared of infection instead of opting to perform a re-revision of the pseudomeningocele. CSF 2 days before shunt placement demonstrated normal glucose (50 mg/dL), elevated protein (76 mg/dL), elevated red blood cells (2/μL), and elevated nucleated cells (17/μL), although much improved. On follow-up, the patient was asymptomatic, surgical wound was well-healed, and pseudomeningocele was resolved. Two years after surgery, she still has occasional headaches but has returned to all her previous activities as a full-time teacher and mother of three.

Systematic review of the literature

We performed a PubMed search in the English language using the search terms "Post-partum posterior reversible encephalopathy syndrome" and "Posterior reversible encephalopathy + Decompression" between January 2008 and October 2018. A total of 62 articles in the English language were found from the search. A total of 26 articles were excluded from analysis because they were reviews of the topic, articles not related to the topic, or articles not in the English language. A total of 36 articles were then available for further analysis. A flow diagram of the search is shown in Fig. 4. As shown in Table 1, these studies comprised 31 case reports and 5 case series, with a total of 55 patients involved. These case reports and case series provided only class IV evidence for the various management modalities. Out of the 55 patients, only 3 (5.5%) were treated by cranial decompression, while the rest were treated medically. Table 2 compares the clinical presentations, treatments, and outcomes of the 3 patients that underwent cranial decompression with our current patient who also required cranial decompression. Two of the 3 patients reported in the literature [1, 24] presented posterior fossa symptoms from edema involving mainly the cerebellum and brainstem. These patients were successfully treated with suboccipital craniectomy, with excellent modified Rankin score of 0 on follow-up. The third patient [27] presented with neurological deterioration and motor symptoms from edema involving the frontal lobes and corpus callosum. The patient was treated with decompressive hemicraniectomy and on follow-up had a modified Rankin score of 2. Our current patient, unlike the 3 patients described above, presented post-partum with edema involving both the posterior fossa and supratentorial compartments. Because of the more extensive brain involvement with edema, our patient's clinical course was more protracted, requiring suboccipital craniectomy and subsequent ventriculoperitoneal

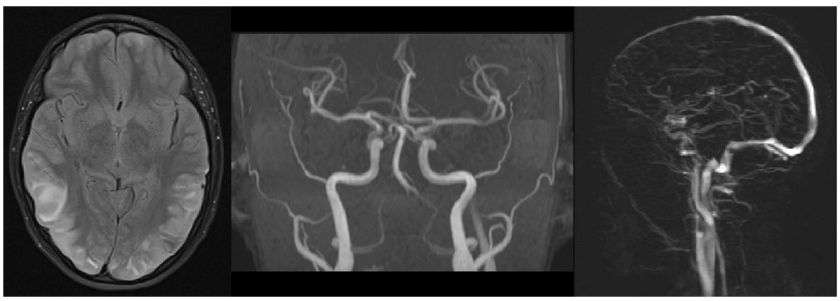

Fig. 3 MRI/MR angiography, showing bilateral occipital edema and absence of vessel occlusion or thrombosis, concerning for posterior reversible encephalopathy syndrome. Of note, edema in PRES is not always confined to the posterior regions of the brain any may extend beyond the occipital lobes into the posterior fossa

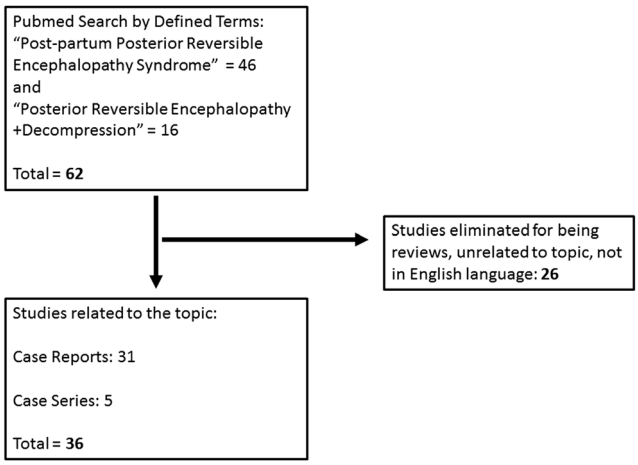

Fig. 4 Flow diagram for systematic literature review

shunt placement. Nonetheless, our patient made an excellent recovery, with a modified Rankin score of 0 on follow-up.

Discussion

PRES is a generally reversible process that improves when the precipitating cause is removed or treated [20]. Severe forms of the disease, however, can result in death, with studies citing 3–6% mortality in general, 26–29% mortality for hemorrhagic PRES and a 16% fatality rate, and 37% rate of significant functional impairments in severe PRES involving neurological deficits requiring intensive care management[1] [3, 26, 37]. Profound neurological injury or death may occur due to intracranial hemorrhage, posterior fossa edema with brainstem compression, elevated intracranial pressure, or acute hydrocephalus.

Pregnant and post-partum women are a unique population described to present with PRES [14, 16, 40, 55, 56, 60, 71]. Most commonly, PRES is associated with toxemia of pregnancy—pre-eclamspia and eclampsia—in which the placenta is thought to be the primary cause and placental removal and fetal delivery considered curative. [7] Hypertension is seen in about 75% of patients with PRES, and most eclamptic/pre-eclamptic patients are hypertensive at toxicity, although 23% have normal or only minimally elevated blood pressure [7]. Severe hypertension with failed autoregulation, injury to the capillary bed, and hyperperfusion remains the most popular etiology explaining the cerebral edema in PRES [8]. Retained placental fragments have also been described as an etiology for toxemia and PRES [15]. A placental-maternal immune reaction has also been postulated as an etiology, with possible immune system activation, endothelial cell activation and injury, and an inflammatory cytokine response resulting in systemic vasoconstriction and labile blood pressure [8]. Presentation of PRES may occur weeks after post-partum. [11, 59] Differential diagnosis may be challenging, especially in the absence of expected MRI findings as seen with our patient's initial imaging. Our patient was initially suspected to have intracranial hypotension and/or meningitis, secondary to dural enhancement seen on her initial MRI. As such, our initial treatment consisted of empiric antibiotics and anti-epileptic medications for her seizure.

PRES has been described in association with dural breaches, CSF leaks, post-lumbar puncture syndrome, or even caffeine intake used to treat this syndrome [51]. Pregnant women often undergo epidurals and spinal anesthesia, so PRES may be associated with pre-eclampsia/eclampsia of pregnancy, or with the epidural that is often performed during delivery. Case reports describe patients who developed PRES after spinal anesthesia complicated by a post-dural puncture headache [28, 41, 67]. Headaches have been described in up to 39% of patients, with > 16% of cases being attributed to dural puncture [18]. A headache in PRES may be difficult to distinguish from post-dural puncture headache, which may delay appropriate diagnosis and treatment [18].

Multiple publications suggest an association between PRES and a drop in intracranial pressure that may come with CSF drainage [19, 23, 25, 48, 62, 64]. PRES has been reported after a lumboperitoneal shunt for idiopathic intracranial hypertension, ventriculo-peritoneal shunt for obstructive hydrocephalus, overdrainage of CSF via an external ventricular drain, large volume lumbar puncture for hydrocephalus, incidental durotomies during surgeries and/or lumbar drains, or even posterior fossa tumor resection [19, 22, 23, 25, 48, 62, 64]. The same mechanism may have resulted in the development of PRES in our patient given there was concern for dural puncture during her epidural.

Two papers describe cases in which post-partum females with prior epidurals developed imaging consistent

Table 1 Reported studies on posterior reversible encephalopathy syndrome

Type of study (level of evidence)	Treatment	References	Number of patients
Case report (IV)	Medical	[4, 6, 10, 12, 21, 31–33, 35, 36, 38, 43, 44, 46, 47, 49, 50, 52–54, 58, 65, 66, 68–70, 72, 73]	28
	Decompression	[1, 24, 27]	3
Case series (IV)	Medical	[9, 17, 29, 30, 34]	24
Total			55

Table 2 Cases of posterior reversible encephalopathy treated by cranial decompression

Ref.	Clinical presentation	Location of edema	Decompression		Modified Rankin score (duration of follow-up)
			Indication	Type	
[1]	Hypertension, headaches, vertigo, blurred vision, progressing somnolence and left unresponsive pupil, tongue deviation to the right, left hemipalsy, and ataxia	Cerebellum, brainstem	Brainstem compression	Suboccipital craniectomy	mRS 0 (2 weeks)
[24]	Hypertension, unresponsive, non-reactive right pupil, weak corneal reflexes, absent gag reflex, triple flexion response, hyperreflexia, positive Hoffman reflexes	Cerebellum	Brainstem compression	Suboccipital craniectomy	mRS 0 (6 months)
[27]	Hypertension, sudden onset hemiplegia, Rapid neurological deterioration	Frontal lobes, corpus callosum	Persistent elevation of intracranial pressure	Hemicraniectomy	mRS 2 (6 months)
Current Case	Post-partum, headaches, grand-mal seizure, altered mental status, hyper--reflexia	Frontal, parietal, temporal, and occipital lobes and cerebellum	Uncal herniation and sagging of brainstem	Suboccipital craniectomy	mRS 0 (2 years)

with PRES, but also had radiographic or clinical findings concerning for intracranial hypotension; these patients improved with Trendelenburg positioning and volume repletion in one case, and support therapy with a blood patch in the other [50, 57]. This is similar to our case, in which initial imaging was concerning for intracranial hypotension without findings suggestive of PRES with an initial plan for Trendelenburg positioning and blood patch.

Treatment for malignant PRES includes aggressive management of mass effect and edema using steroids, hyperosmolar therapy, blood pressure control, and treatment of coagulopathy and seizures [61]. Magnesium sulfate (competitive antagonist to calcium) has been described in PRES to improve seizures and hypertension, in addition to acting as a possible barrier against cerebral edema formation and vasoconstriction [14, 16]. In a minority of cases, craniotomy/craniectomy with possible hematoma evacuation is required [1, 2, 24, 27, 45]. We employed the aforementioned conservative therapy, but the patient continued to deteriorate, requiring posterior fossa decompression.

During surgery, brain biopsy may be performed. At times, intracranial pressure monitoring and external ventricular drain placement are necessary. Atkins et al. suggested that more aggressive treatment with craniectomy and evacuation of hematoma should be pursued in patients with malignant PRES, defined as Glasgow Coma Score less than 8 and clinical decline despite medical management for elevated intracranial pressure, along with radiographic criteria such as edema or hemorrhage with mass effect, effacement of cisterns, and/or herniation [2]. Their data with aggressive surgical management demonstrated a 100% survival rate at 90 days without severe functional disability. Our patient underwent successful posterior fossa decompression and later returned to her baseline functional status.

Conclusions

A diagnosis of PRES is not always easily made due to confounding clinical or imaging data. Conservative management may be inadequate in the treatment of severe and malignant PRES, and a craniectomy for decompression may be required even in the absence of intracranial hemorrhage. It may be associated with pregnancy, eclampsia/preeclampsia, and/or CSF drainage.

Compliance with ethical standards

Conflict of interest The authors declare no conflict of interest.

Patient consent The patient has consented to the submission of the case report for submission to the journal.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

1. Abusabha Y, Petridis AK, Kraus B, Kamp MA, Steiger HJ, Beseoglu K (2017) Life-threatening posterior reversible encephalopathy syndrome in the cerebellum treated by posterior fossa decompression. Acta Neurochir 159:1325–1328. https://doi.org/10.1007/s00701-017-3228-7
2. Akins PT, Axelrod Y, Silverthorn JW, Guppy K, Banerjee A, Hawk MW (2014) Management and outcomes of malignant posterior reversible encephalopathy syndrome. Clin Neurol Neurosurg 125:52–57. https://doi.org/10.1016/j.clineuro.2014.06.034
3. Aranas RM, Prabhakaran S, Lee VH (2009) Posterior reversible encephalopathy syndrome associated with hemorrhage. Neurocrit Care 10:306–312. https://doi.org/10.1007/s12028-009-9200-5

4. Aygün BK, Baykuş Y, Berilgen S, Kavak B, Celik H, Gürateş B (2010) Posterior reversible encephalopathy syndrome in severe preeclampsia: case report and literature review. J Turk Ger Gynecol Assoc 11:216–219. https://doi.org/10.5152/jtgga.2010.41
5. Azriel A, Melamed I, Shelef I, Sufaro Y, Gidon M, Merkin V, Shtreizent A, Cohen A (2015) Decompressive craniectomy for posterior reversible encephalopathy syndrome (PRES): case report. Case Rep Clin Med 4:169–174. https://doi.org/10.4236/crcm.2015.45034
6. Babahabib MA, Abdillahi I, Kassidi F, Kouach J, Moussaoui D, Dehayni M (2015) Posterior reversible encephalopathy syndrome in patient of severe preeclampsia with Hellp syndrome immediate postpartum. Pan Afr Med J 21:60. https://doi.org/10.11604/pamj.2015.21.60.5546
7. Bartynski WS (2008) Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol 29:1036–1042. https://doi.org/10.3174/ajnr.A0928
8. Bartynski WS (2008) Posterior reversible encephalopathy syndrome, part 2: controversies surrounding pathophysiology of vasogenic edema. AJNR Am J Neuroradiol 29:1043–1049. https://doi.org/10.3174/ajnr.A0929
9. Bembalgi S, Kamate V, Shruthi KR (2015) A study of eclampsia cases associated with posterior reversible encephalopathy syndrome. J Clin Diagn Res 9:QC05–QC07. https://doi.org/10.7860/JCDR/2015/14039.6276
10. Block HS, Biller J (2014) Neurology of pregnancy. Handb Clin Neurol 121:1595–1622. https://doi.org/10.1016/B978-0-7020-4088-7.00105-X
11. Bo QY, Zhao XH, Yang X, Wang SJ (2016) Reversible posterior encephalopathy syndrome associated with late onset postpartum eclampsia: a case report. Exp Ther Med 12:1885–1888. https://doi.org/10.3892/etm.2016.3533
12. Borovac JA, Božić J, Žaja N, Kolić K, Hrboka V (2016) A global amnesia associated with the specific variant of posterior reversible encephalopathy syndrome (PRES) that developed due to severe preeclampsia and malignant hypertension. Oxf Med Case Rep 2016:76–80. https://doi.org/10.1093/omcr/omw016
13. Burnett MM, Hess CP, Roberts JP, Bass NM, Douglas VC, Josephson SA (2010) Presentation of reversible posterior leukoencephalopathy syndrome in patients on calcineurin inhibitors. Clin Neurol Neurosurg 112:886–891. https://doi.org/10.1016/j.clineuro.2010.07.023
14. Cozzolino M, Bianchi C, Mariani G, Marchi L, Fambrini M, Mecacci F (2015) Therapy and differential diagnosis of posterior reversible encephalopathy syndrome (PRES) during pregnancy and postpartum. Arch Gynecol Obstet 292:1217–1223. https://doi.org/10.1007/s00404-015-3800-4
15. Delefosse D, Samain E, Helias A, Regimbeau JM, Deval B, Farah E, Marty J (2003) Late onset of cortical blindness in a patient with severe preeclampsia related to retained placental fragments. Anesthesiology 98:261–263
16. Demirel I, Ozer AB, Bayar MK, Kavak SB (2012) Anesthesia and intensive care management in a pregnant woman with PRES: a case report. Case Rep Anesthesiol 2012:745939. https://doi.org/10.1155/2012/745939
17. Demirel İ, Kavak BS, Özer AB, Bayar MK, Erhan Ö (2014) An intensive care approach to posterior reversible encephalopathy syndrome (PRES): an analysis of 7 cases. J Turk Ger Gynecol Assoc 15:217–221. https://doi.org/10.5152/jtgga.2014.14072
18. Doherty H, Hameed S, Ahmed I, Russell IF (2014) Post-dural puncture headache and posterior reversible encephalopathy syndrome: a misdiagnosis or co-presentation? Int J Obstet Anesth 23:279–282. https://doi.org/10.1016/j.ijoa.2014.02.003
19. Fok A, Chandra RV, Gutman M, Ligtermoet M, Seneviratne U, Kempster P (2016) Posterior reversible encephalopathy syndrome and subarachnoid hemorrhage after lumboperitoneal shunt for fulminant idiopathic intracranial hypertension. J Neuroophthalmol 36:164–166. https://doi.org/10.1097/WNO.0000000000000355
20. Fugate JE, Rabinstein AA (2015) Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. Lancet Neurol 14:914–925. https://doi.org/10.1016/S1474-4422(15)00111-8
21. Garg RK, Malhotra HS, Patil TB, Agrawal A (2013) Cerebral-autoregulatory dysfunction syndrome. BMJ Case Rep 2013. https://doi.org/10.1136/bcr-2013-201592
22. Gonzalez Quarante LH, Mena-Bernal JH, Martin BP, Ramirez Carrasco M, Munoz Casado MJ, Martinez de Aragon A, de las Heras RS (2016) Posterior reversible encephalopathy syndrome (PRES): a rare condition after resection of posterior fossa tumors: two new cases and review of the literature. Childs Nerv Syst 32:857–863. https://doi.org/10.1007/s00381-015-2954-5
23. Grelat M, Debaux JB, Sautreaux JL (2014) Posterior reversible encephalopathy syndrome after depletive lumbar puncture: a case report. J Med Case Rep 8:261. https://doi.org/10.1186/1752-1947-8-261
24. Grossbach AJ, Abel TJ, Hodis B, Wassef SN, Greenlee JD (2014) Hypertensive posterior reversible encephalopathy syndrome causing posterior fossa edema and hydrocephalus. J Clin Neurosci 21:207–211. https://doi.org/10.1016/j.jocn.2013.07.005
25. Hammad T, DeDent A, Algahtani R, Alastal Y, Elmer L, Medhkour A, Safi F, Assaly R (2015) Posterior reversible encephalopathy syndrome secondary to CSF leak and intracranial hypotension: a case report and literature review. Case Rep Neurol Med 2015:538523. https://doi.org/10.1155/2015/538523
26. Hefzy HM, Bartynski WS, Boardman JF, Lacomis D (2009) Hemorrhage in posterior reversible encephalopathy syndrome: imaging and clinical features. AJNR Am J Neuroradiol 30:1371–1379. https://doi.org/10.3174/ajnr.A1588
27. Hernandez-Duran S, Barrantes-Freer A, Rohde V, von der Brelie C (2017) Posterior reversible encephalopathy syndrome presenting in the anterior circulation with malignant intracranial hypertension requiring surgical decompression: a case report and literature review. Acta Neurochir 159:1321–1324. https://doi.org/10.1007/s00701-017-3197-x
28. Ho CM, Chan KH (2007) Posterior reversible encephalopathy syndrome with vasospasm in a postpartum woman after postdural puncture headache following spinal anesthesia. Anesth Analg 105:770–772. https://doi.org/10.1213/01.ane.0000278128.26896.b2
29. Jamieson DG, Cheng NT, Skliut M (2014) Headache and acute stroke. Curr Pain Headache Rep 18:444. https://doi.org/10.1007/s11916-014-0444-1
30. Jessani LG, Moin A, Karnati S, Keshavamurthy (2016) Atypical posterior reversible encephalopathy syndrome (PRES) in de novo late post-partum eclampsia. J Assoc Physicians India 64:77–79
31. Joshi V, Mort DJ (2008) Unrecognised late post-partum eclampsia presenting as posterior reversible encephalopathy syndrome. Acute Med 7:87–88
32. Karuppannasamy D, Vikrant K, Raghuram A, Kumaar TM (2014) Cortical visual loss in posterior reversible encephalopathy syndrome in late postpartum eclampsia: case series. Indian J Ophthalmol 62:635–638. https://doi.org/10.4103/0301-4738.133525
33. Kauntia R, Valsalan R, Seshadri S, Pandit VR, Prabhu MM (2009) Late postpartum preeclampsia with posterior reversible encephalopathy syndrome. Indian J Med Sci 63:508–511. https://doi.org/10.4103/0019-5359.58880
34. Krishnamoorthy U, Sarkar PK, Nakhuda Y, Mullins PD (2009) Posterior reversible encephalopathy syndrome (PRES) in pregnancy: a diagnostic challenge to obstetricians. J Obstet Gynaecol 29:192–194. https://doi.org/10.1080/01443610802716026

35. Lee SY, Dinesh SK, Thomas J (2008) Hypertension-induced reversible posterior leukoencephalopathy syndrome causing obstructive hydrocephalus. J Clin Neurosci 15:457–459. https://doi.org/10.1016/j.jocn.2006.12.019
36. Lee SH, Yun SJ, Choi YH (2017) Reversible cerebral vasoconstriction syndrome presenting as subarachnoid hemorrhage: a rare cause of postpartum seizure. Am J Emerg Med 35:807.e801–807.e803. https://doi.org/10.1016/j.ajem.2016.12.021
37. Legriel S, Schraub O, Azoulay E, Hantson P, Magalhaes E, Coquet I, Bretonniere C, Gilhodes O, Anguel N, Megarbane B, Benayoun L, Schnell D, Plantefeve G, Charpentier J, Argaud L, Mourvillier B, Galbois A, Chalumeau-Lemoine L, Rivoal M, Durand F, Geffroy A, Simon M, Stoclin A, Pallot JL, Arbelot C, Nyunga M, Lesieur O, Troche G, Bruneel F, Cordoliani YS, Bedos JP, Pico F, Critically IIIPRESSG (2012) Determinants of recovery from severe posterior reversible encephalopathy syndrome. PLoS One 7:e44534. https://doi.org/10.1371/journal.pone.0044534
38. Lemmens R, Smet S, Wilms G, Demaerel P, Thijs V (2012) Postpartum RCVS and PRES with normal initial imaging findings. Acta Neurol Belg 112:189–192. https://doi.org/10.1007/s13760-012-0051-z
39. Liman TG, Bohner G, Endres M, Siebert E (2014) Discharge status and in-hospital mortality in posterior reversible encephalopathy syndrome. Acta Neurol Scand 130:34–39. https://doi.org/10.1111/ane.12213
40. Mayama M, Uno K, Tano S, Yoshihara M, Ukai M, Kishigami Y, Ito Y, Oguchi H (2016) Incidence of posterior reversible encephalopathy syndrome in eclamptic and patients with preeclampsia with neurologic symptoms. Am J Obstet Gynecol 215(239):e231–e235. https://doi.org/10.1016/j.ajog.2016.02.039
41. Minai FN, Hasan SF, Sheerani M (2011) Post-dural puncture posterior reversible encephalopathy syndrome. J Coll Physicians Surg Pak 21:37–39. 01.2011/JCPSP.3739
42. Moon SN, Jeon SJ, Choi SS, Song CJ, Chung GH, Yu IK, Kim DH (2013) Can clinical and MRI findings predict the prognosis of variant and classical type of posterior reversible encephalopathy syndrome (PRES)? Acta Radiol 54:1182–1190. https://doi.org/10.1177/0284185113491252
43. More A, Garg RK, Malhotra HS, Kumar N, Uniyal R (2016) Acute vision loss in post-partum period as presenting symptom of HIV-associated cryptococcal meningitis-an unusual case report. BMC Infect Dis 16:582. https://doi.org/10.1186/s12879-016-1925-0
44. Morelli N, Gori S, Michelassi MC, Falorni M, Cafforio G, Bianchi MC, Cosottini M, Orlandi G, Murri L, Tartaglione A (2008) Atypical posterior reversible encephalopathy syndrome in puerperium. Eur Neurol 59:195–197. https://doi.org/10.1159/000114044
45. Nagaoka Y, Ishikura K, Hamada R, Miyagawa T, Kono T, Sakai T, Hamasaki Y, Hataya H, Honda M (2013) Severe posterior reversible encephalopathy syndrome resolved with craniectomy. Pediatr Int 55:644–646. https://doi.org/10.1111/ped.12084
46. Ng MD, Manikappa S (2012) Postpartum seizure and ischaemic stroke following dural puncture and epidural blood patch. Anaesth Intensive Care 40:347–351
47. Nishimura M, Hiraoka E, Kanazawa K, Akita H (2015) Postpartum vertebral artery dissection with posterior reversible encephalopathy syndrome. BMJ Case Rep 2015. https://doi.org/10.1136/bcr-2014-207332
48. Niwa R, Oya S, Nakamura T, Hana T, Matsui T (2017) Rapid intracranial pressure drop as a cause for posterior reversible encephalopathy syndrome: two case reports. Surg Neurol Int 8:103. https://doi.org/10.4103/sni.sni_55_17
49. O'Kane M, Elhalwagy H, Kumar S, Badawi C (2014) Unusual presentation of PRES in the postnatal period. BMJ Case Rep 2014. https://doi.org/10.1136/bcr-2013-203406
50. Orehek EK, Burns JD, Koyfman F, Azocar RJ, Holsapple JW, Green DM (2012) Postpartum trifecta: simultaneous eclamptic intracerebral hemorrhage, PRES, and herniation due to intracranial hypotension. Neurocrit Care 17:434–438. https://doi.org/10.1007/s12028-012-9742-9
51. Ortiz GA, Bianchi NA, Tiede MP, Bhatia RG (2009) Posterior reversible encephalopathy syndrome after intravenous caffeine for post-lumbar puncture headaches. AJNR Am J Neuroradiol 30:586–587. https://doi.org/10.3174/ajnr.A1321
52. Oyinloye OI, Adesiyun OA, Atobatele MO, Fawole AA (2014) Posterior reversible encephalopathy syndrome in a adult female. Ann Afr Med 13:138–141. https://doi.org/10.4103/1596-3519.134422
53. Pala HG, Baris MM, Artunc Ulkumen B, Gurkan MA, Pala EE (2015) Posterior reversible encephalopathy syndrome: an atypical complication of postpartum wound infection. J Obstet Gynaecol 35:306–308. https://doi.org/10.3109/01443615.2014.948819
54. Peng WX, Nakaii M, Matsushima T, Asakura H (2008) Atypical case of reversible posterior leucoencephalopathy syndrome associated with puerperal HELLP syndrome. Arch Gynecol Obstet 278:269–271. https://doi.org/10.1007/s00404-008-0578-7
55. Poma S, Delmonte MP, Gigliuto C, Imberti R, Delmonte M, Arossa A, Iotti GA (2014) Management of posterior reversible syndrome in preeclamptic women. Case Rep Obstet Gynecol 2014:928079. https://doi.org/10.1155/2014/928079
56. Postma IR, Slager S, Kremer HP, de Groot JC, Zeeman GG (2014) Long-term consequences of the posterior reversible encephalopathy syndrome in eclampsia and preeclampsia: a review of the obstetric and nonobstetric literature. Obstet Gynecol Surv 69:287–300. https://doi.org/10.1097/OGX.0000000000000069
57. Pugliese S, Finocchi V, Borgia ML, Nania C, Della Vella B, Pierallini A, Bozzao A (2010) Intracranial hypotension and PRES: case report. J Headache Pain 11:437–440. https://doi.org/10.1007/s10194-010-0226-z
58. Reynaud Q, Killian M, Tardy B (2015) An uncommon case of post partum seizures. Eur J Intern Med 26:e41–e42. https://doi.org/10.1016/j.ejim.2015.02.004
59. Rijal JP, Giri S, Dawadi S, Dahal KV (2014) Posterior reversible encephalopathy syndrome (PRES) in a patient with late postpartum eclampsia. BMJ Case Rep 2014. https://doi.org/10.1136/bcr-2013-203162
60. Servillo G, Striano P, Striano S, Tortora F, Boccella P, De Robertis E, Rossano F, Briganti F, Tufano R (2003) Posterior reversible encephalopathy syndrome (PRES) in critically ill obstetric patients. Intensive Care Med 29:2323–2326. https://doi.org/10.1007/s00134-003-1901-1
61. Servillo G, Bifulco F, De Robertis E, Piazza O, Striano P, Tortora F, Striano S, Tufano R (2007) Posterior reversible encephalopathy syndrome in intensive care medicine. Intensive Care Med 33:230–236. https://doi.org/10.1007/s00134-006-0459-0
62. Shah R, Kubisz-Pudelko A, Reid J (2014) Posterior reversible encephalopathy syndrome following an inadvertent dural puncture during an emergency laparotomy for ischemic colitis - a case report. Local Reg Anesth 7:1–4. https://doi.org/10.2147/LRA.S57660
63. Shaharir SS, Remli R, Marwan AA, Said MS, Kong NC (2013) Posterior reversible encephalopathy syndrome in systemic lupus erythematosus: pooled analysis of the literature reviews and report of six new cases. Lupus 22:492–496. https://doi.org/10.1177/0961203313478303
64. Shields LB, Johnson JR, Shields CB (2016) Posterior reversible encephalopathy syndrome following a thoracic discectomy-induced dural leak: case report. J Neurosurg Spine 25:586–590. https://doi.org/10.3171/2016.4.SPINE1623
65. Takahashi H, Matsubara T, Makino S, Horie K, Matsubara S (2017) Color vision abnormality as the sole manifestation of posterior reversible encephalopathy due to post-partum HELLP syndrome. J Obstet Gynaecol Res 43:592–594. https://doi.org/10.1111/jog.13241

66. Tanaka K, Matsushima M, Matsuzawa Y, Wachi Y, Izawa T, Sakai K, Kobayashi Y, Iwashita M (2015) Antepartum reversible cerebral vasoconstriction syndrome with pre-eclampsia and reversible posterior leukoencephalopathy. J Obstet Gynaecol Res 41:1843–1847. https://doi.org/10.1111/jog.12788
67. Torrillo TM, Bronster DJ, Beilin Y (2007) Delayed diagnosis of posterior reversible encephalopathy syndrome (PRES) in a parturient with preeclampsia after inadvertent dural puncture. Int J Obstet Anesth 16:171–174. https://doi.org/10.1016/j.ijoa.2006.08.015
68. Yi JH, Ha SH, Kim YK, Choi EM (2011) Posterior reversible encephalopathy syndrome in an untreated hypertensive patient after spinal surgery under general anesthesia -a case report. Korean J Anesthesiol 60:369–372. https://doi.org/10.4097/kjae.2011.60.5.369
69. Youssoufa M, Callixte KT, Christian N (2013) Occipital lobe epilepsy secondary to posterior reversible encephalopathy syndrome (PRES) during a post-partum eclampsia in Mali (West Africa). BMC Res Notes 6:321. https://doi.org/10.1186/1756-0500-6-321
70. Zhang M, Lian Y, Liu XH, Peng W, Wang HQ (2014) Could extreme emotional stress be a potential precipitating factor associated with posterior reversible encephalopathy syndrome in postpartum woman? A case report. Neurol Sci 35:317–318. https://doi.org/10.1007/s10072-013-1534-2
71. Zhang L, Wang Y, Shi L, Cao J, Li Z, Wang YX (2015) Late postpartum eclampsia complicated with posterior reversible encephalopathy syndrome: a case report and a literature review. Quant Imaging Med Surg 5:909–916. https://doi.org/10.3978/j.issn.2223-4292.2015.12.04
72. Zimering JH, Mesfin A (2018) Posterior reversible encephalopathy syndrome following elevated mean arterial pressures for cervical spinal cord injury. J Spinal Cord Med 41:111–114. https://doi.org/10.1080/10790268.2016.1250030
73. Zis P, Tavernarakis A (2013) Headache and status epilepticus in the postpartum period; posterior reversible encephalopathy syndrome or cerebral venous thrombosis? Case Rep Emerg Med 2013:680327. https://doi.org/10.1155/2013/680327