Neurological Manifestations in the Peri-Partum Period: Imaging Overview

Rahul Ranjan[1], Aman Gupta[2], Namrata Deo[3], Nirmal Pandey[4], Anil Singh[5], Sumedha Varshney[6]

[1,2,6] Department of Radiodiagnosis, Rama Medical College, Kanpur, Uttar Pradesh

[3] Department of Ophthalmology, Rama Medical College, Kanpur, Uttar Pradesh

[4] Department of Neurology, Regency Hospital, Kanpur

[5] Department of Radiodiagnosis, SGPGI, Lucknow

Date of Submission:10th March 2025 Date of Acceptance:3rd November 2025 Date of Publication: 15th December 2025

Abstract

The peripartum period, covering late pregnancy through postpartum, brings significant changes that increase the risk of neurological disorders such as cortical vein thrombosis (CVT), osmotic demyelination syndrome (ODS), pituitary apoplexy, postpartum cerebral angiopathy (PCA), posterior reversible encephalopathy syndrome (PRES), and intracranial hypotension (IH). Accurate diagnosis relies heavily on MRI and CT, with MRI being especially useful for its detailed images. CVT often presents with severe headaches and seizures, detected on MRI through characteristic signs like the "empty delta sign." ODS, linked to the rapid correction of low sodium levels, appears as T2 hyperintense lesions at specific locations. Pituitary apoplexy, involving sudden hemorrhage within a pituitary adenoma, typically requires high-dose corticosteroids and, in severe cases, surgery. PCA results from hormonal changes and shows as transient artery narrowing on MR angiography, managed with supportive care. PRES, associated with high blood pressure disorders, manifests as vasogenic brain edema, treated by managing blood pressure. IH, often seen after a dural puncture, leads to positional headaches and shows brain sagging on MRI, with treatments ranging from rest to epidural blood patches. Recognizing these imaging patterns is critical for timely diagnosis and effective treatment, ultimately enhancing outcomes for mothers & preventing peripartum morbidity & mortality.

Key Words: Angiopathy, Apoplexy, Cortical vein, Dural venous sinus, Encephalopathy, Intracranial hypotension, Pituitary, Peripartum, Thrombosis, Vasoconstriction

INTRODUCTION

The peripartum period, spanning late pregnancy through the postpartum stage, is marked by significant physiological changes that can predispose women to various neurological and neurovascular conditions. This phase is particularly critical because it involves unique challenges in diagnosis and management due to overlapping symptoms of normal pregnancy related changes and potential pathological conditions. Imaging plays a pivotal role in early detection, accurate diagnosis, and therapeutic decision-making, especially for conditions that can cause acute morbidity or mortality if not recognized in time.

Among the most critical neurological conditions during the peripartum period are cortical vein thrombosis (CVT)[1], which involves clot formation in cerebral veins, leading to symptoms like headaches, seizures and altered sensorium; osmotic demyelination syndrome (ODS)(formerly called central pontine myelinolysis)[2], usually caused by rapid correction of hyponatremia resulting in damage to the brain's myelin; and pituitary apoplexy[3], characterized by sudden hemorrhage within a pituitary tumor, causing severe headaches and visual disturbances. Additionally, reversible cerebral vasoconstriction syndrome (RCVS), including postpartum cerebral angiopathy (PCA)[4] involves transient cerebral artery constriction, while posterior reversible encephalopathy syndrome (PRES)is associated with hypertension-induced brain swelling, presenting with seizures and visual changes. Prompt central nervous system (CNS) imaging is crucial for accurate diagnosis and management of these conditions.

The two most frequently employed imaging modalities in the evaluation of neurological manifestations during the peri-partum period are magnetic resonance imaging (MRI) and computed tomography (CT) [5]. MRI, in particular, is favoured for its superior tissue contrast, enabling detailed visualization of parenchymal, vascular, and meningeal structures[6]. The ability to use advanced techniques such as magnetic resonance venography (MRV) and diffusion-weighted imaging (DWI) further enhances its utility in evaluating complex conditions like venous thrombosis, angiopathies, and demyelinating disorders.

Access this article online

Website: https://www.nepjol.info/index.php/NJN

DOI: https://doi.org/10.3126/njn.v22i24.76478

HOW TO CITE
Ranjan, R., Gupta, A., Deo, N., Pandey, N., Singh, A., & Varshney, S. Neurological Manifestations in the Peri-Partum Period: Imaging Overview. NJNS. 2025;22(4):3-10

Address for correspondence:
Dr Rahul Ranjan
Department of Radiodiagnostics, Rama Medical College, Kanpur, Uttar Pradesh
Gmail: rahulranjanradio10@gmail.com.

ISSN: 1813-1948 (Print), 1813-1956 (Online)

This review aims to provide a detailed analysis of the imaging characteristics associated with these neurological conditions in the peripartum period, highlighting the role of various imaging modalities in their diagnosis and management. Recognizing these imaging patterns is essential for early diagnosis and intervention, ultimately improving clinical outcomes in peripartum women.

DISCUSSION

Cortical Vein Thrombosis

Incidence: It is estimated to occur in about 11.6 per 100,000 deliveries in developed countries. The risk is particularly elevated in the postpartum period, which accounts for 60-70% of peripartum CVT cases [7].

Pathogenesis: CVT occurs due to thrombosis within the cerebral dural venous sinuses or cortical veins, resulting in impaired venous drainage and increased intracranial pressure. This blockage causes venous congestion, leading to increased capillary hydrostatic pressure and disruption of the blood-brain barrier, which in turn can cause vasogenic edema and hemorrhagic infarction [8]. The hypercoagulable state of pregnancy, characterized by elevated levels of pro-coagulant factors like fibrinogen and decreased fibrinolysis, predisposes women to CVT, particularly in the postpartum period [9]. Additional risk factors such as dehydration, infection, and cesarean section further contribute to thrombosis.

Clinical Features: CVT often presents with a triad of symptoms: headache, seizures, altered sensorium, and focal neurological deficits. The headache is typically progressive, severe, and may present as a thunderclap headache. Seizures, which may be focal or generalized, occur due to irritation of the cerebral cortex by venous infarction. Papilledema, a sign of elevated intracranial pressure, is frequently observed on physical examination, along with cranial nerve palsies in cases where increased pressure affects the brainstem or cranial nerve roots.

Imaging Findings: CVT is best visualized using MRI with MRV (contrast MRV being more confirmatory & sensitive), which together provide detailed images of the cerebral venous system. The most common finding in such cases is loss of flow void in dural venous sinuses on T2-weighted (T2W)&fluid-attenuated inversion recovery (FLAIR) images with hyperintense signal on T1W images (Figure 1,2).

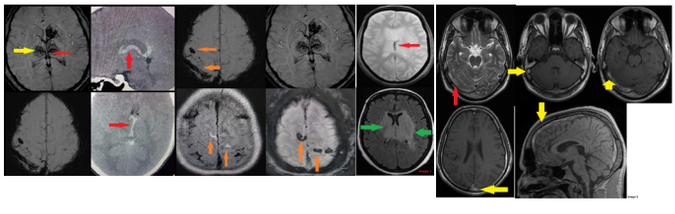

Figure 1,2: loss of flow void in dural venous sinuses on T2-weighted (T2W)&fluid-attenuated inversion recovery (FLAIR) images with hyperintense signal on T1W images (Figure 1,2).

A classic radiological sign of CVT is the "empty delta sign" on contrast-enhanced T1-weighted (T1W) images, which appears as a filling defect in the superior sagittal sinus surrounded by enhancing dura [10]. This feature is indicative of thrombosis obstructing blood flow. During the subacute phase, thrombus formation appears hyperintense on T1W images and hypointense on T2W images due to the presence of methemoglobin (Figure 2). MRV can demonstrate the absence of flow within the affected sinus or cortical vein. Parenchymal involvement may be seen on T2 and FLAIR sequences, showing hyperintense regions consistent with vasogenic edema or cytotoxic edema in the cortical and subcortical regions[11,12]. Susceptibility-weighted imaging (SWI) or gradient echo sequences(GRE) is especially useful for detecting microhemorrhages, a common complication of CVT that can signify venous infarction. Thrombosed cortical veins can also be seen as a hyperintense signal on FLAIR images with susceptibility on GRE or SWI images within the sulcal spaces (Figure 1).

Differential Diagnosis: Differentiating CVT from conditions like ischemic stroke, particularly in young patients, is crucial. Ischemic strokes typically affect arterial territories and present with characteristic diffusion restriction on DWI MRI, whereas venous infarcts in CVT are more likely to demonstrate hemorrhagic transformation and do not conform to arterial distributions[13,14]. Other conditions like dural arteriovenous fistulas may present similarly with headache and intracranial hemorrhage but are characterized by abnormal flow voids and enlarged cortical veins on MRI [15].

Management:

- The primary treatment for CVT is anticoagulation, even in the presence of hemorrhages, to prevent further clot propagation. Low-molecular-weight heparin is typically preferred initially, followed by oral anticoagulants like warfarin for a duration of 3-6 months or longer, depending on the severity of the condition and the underlying risk factors[16].
- In severe cases, where there is a risk of intracranial pressure (ICP) elevation, measures like osmotic therapy (e.g., mannitol) and decompressive craniectomy may be considered.
- For patients with recurrent thrombosis or known thrombophilia, long-term anticoagulation may be necessary [17].
- Regular follow-up with imaging, typically MRV preferably with contrast, is advised to assess the resolution of the thrombus [18].

Osmotic Demyelination Syndrome

Pathogenesis: ODS arises from the rapid correction of chronic hyponatremia, leading to a shift in water balance that causes osmotic injury to oligodendrocytes. This results in the demyelination of regions where the gray-white matter interface is particularly sensitive. During pregnancy, fluid and electrolyte imbalances are common, increasing the risk of ODS if hyponatremia is corrected too quickly, leading to cellular shrinkage and demyelination [19].

Clinical Features: ODS presents with a rapid decline in neurological function following sodium correction. Patients may develop symptoms such as dysarthria, dysphagia, quadriparesis,

and altered consciousness [20]. Severe cases can lead to locked-in syndrome if corticospinal tracts in the pons are involved, making early diagnosis crucial.

Imaging Findings: ODS is characterized by T2W hyperintense lesions on MRI, particularly in the central pons and basal ganglia. Central pontine myelinolysis often manifests as a "trident-shaped" hyperintensity on axial T2W images, while extrapontine myelinolysis can involve the thalamus, cerebellum, and subcortical white matter (Figure3) [21].

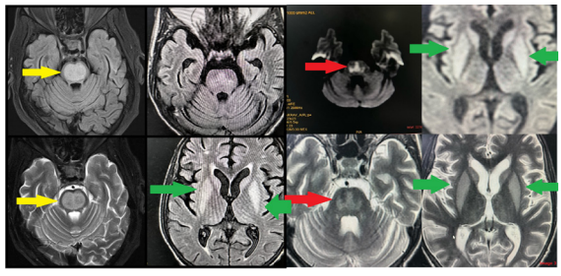

Figure 3: T2W hyperintense lesions on MRI, particularly in the central pons and basal ganglia.

DWI is particularly valuable in identifying areas of acute demyelination and cytotoxic edema, as the earliest finding visualized on MR is diffusion restriction, which may evolve into areas of T2W/FLAIR hyperintensity over time. Lesions typically lack enhancement, distinguishing them from infectious or neoplastic processes. T2W and FLAIR linear hyperintense signal can also be seen at the gray-white matter junction in the bilateral cerebral hemispheres.

Differential Diagnosis: Differential diagnosis includes brainstem infarctions and multiple sclerosis (MS), but the symmetric distribution of demyelination and lack of enhancement on MRI help distinguish ODS [22]. MS typically presents with asymmetric lesions and gadolinium-enhancing plaques during active disease, while infarctions show more localized restriction on DWI.

Management:

- Prevention is key, focusing on the careful correction of chronic hyponatremia to avoid rapid shifts in serum sodium levels. The recommended rate of sodium correction is less than 10 mmol/L per day.
- In cases of acute ODS, supportive care is critical. This includes managing airway protection, controlling seizures with antiepileptic drugs, and providing nutritional and fluid support.
- No specific therapy has been proven to reverse the demyelination, but early recognition and halting the progression of sodium correction can help mitigate further damage.

Pituitary Macroadenoma with Hemorrhage (Pituitary Apoplexy)

Pathogenesis: Pituitary apoplexy involves sudden hemorrhage or infarction within a pre-existing pituitary adenoma. During pregnancy, increased estrogen levels lead to pituitary hyperplasia, resulting in increased vascularity and a higher risk of apoplexy. This rapid expansion can compress surrounding structures, leading to symptoms like visual loss and endocrinopathies.

Clinical Features: Patients with pituitary apoplexy present with a sudden onset of headache, visual disturbances like bitemporal hemianopia due to optic chiasm compression, ophthalmoplegia, and signs of acute adrenal insufficiency, such as hypotension and altered mental status [23].

Imaging Findings: MRI is the imaging modality of choice for diagnosing pituitary apoplexy, focusing on the sella. Acute hemorrhage within a pituitary adenoma is visualized as hyperintense on T1W images due to the presence of methemoglobin. T2W imaging can reveal mixed signal intensities that correlate with varying stages of hemorrhage and necrosis (Figure 4).[24]

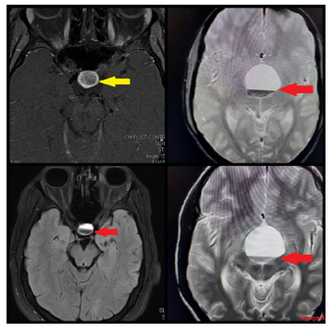

Figure 4 : Pituitary apoplexy hyperintense on T1W images and mixed signal intensities in T2W imaging

Post-contrast imaging is crucial to identify residual adenomatous tissue and assess compression of the optic chiasm and cavernous sinus [25]. Recognizing the classic "snowman" shape of large macroadenomas is essential for accurate diagnosis.

Differential Diagnosis: Differential diagnoses include craniopharyngiomas, meningiomas, and aneurysms. However, the acute presentation, presence of hemorrhage, and characteristic pituitary location on MRI aid in the diagnosis of pituitary apoplexy [26]. Craniopharyngiomas are usually composed of solid-cystic areas with calcification, while meningiomas are usually homogenous enhancing with an associated dural tail. An aneurysm shows characteristic dilatation of the lumen of the vessel with a prominent flow void; sometimes associated with flow artifact.

Management:

- Acute management includes high-dose corticosteroids (e.g., dexamethasone) to reduce cerebral edema and maintain adrenal function, as hypopituitarism can develop suddenly. Surgical decompression through transsphenoidal
- surgery may be required urgently if there is significant visual impairment or altered mental status due to optic chiasm compression.
- Hormonal replacement therapy is often necessary, addressing deficiencies in cortisol, thyroid hormones, and possibly sex hormones following the acute phase.

- Regular MRI follow-up is required to monitor residual adenoma size and pituitary function.

Postpartum Cerebral Angiopathy

Pathogenesis: PCA is thought to result from a transient dysfunction of the cerebral arteries, often triggered by hormonal changes associated with pregnancy and the postpartum state& therefore now considered as a spectrum of reversible vasoconstriction syndrome (RCVS)[27]. Elevated levels of vasoactive substances like endothelin, which promote vasoconstriction, are believed to play a role [28]. Additionally, factors like hypertension, preeclampsia, and use of vasoconstrictive medications during labor can contribute to the pathogenesis of PCA [29]. This condition typically resolves spontaneously as the hormonal milieu stabilizes post-delivery [30].

Clinical Features: Patients with PCA typically present within two weeks postpartum with acute-onset severe headaches, often described as thunderclap headaches, mimicking those seen in subarachnoid hemorrhage [31]. Neurological deficits like visual disturbances, seizures, and altered consciousness may also occur, depending on the extent of vasoconstriction and associated ischemic changes [32]. Symptoms are usually transient but can recur if untreated [33].

Imaging Findings: A high incidence of suspicion is necessary by the clinician to suspect PCA, as the plain MRI may be normal. It is best evaluated using brain MRI, cerebral angiography, such as CT angiography (CTA) or MRA angiography (MRA), or DSA (Digital Subtraction Angiography)[34]. Imaging typically reveals multifocal areas of segmental narrowing and dilation (beading) in the medium to large-sized cerebral arteries, most notably in the posterior circulation, due to vasospasm and with more preferential involvement of the middle cerebral artery in the anterior circulation (Figure 5)[35].

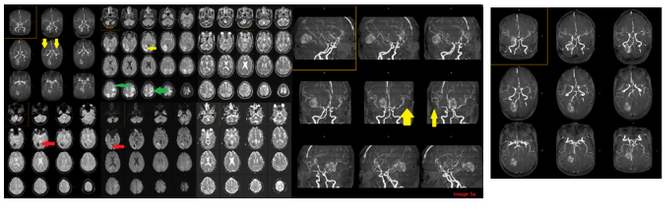

Figure 5: Multifocal areas of segmental narrowing and dilation (beading) in the medium to large-sized cerebral arteries, most notably in the posterior circulation,

This reversible vasoconstriction is characteristic and distinguishes PCA from other conditions like vasculitis [36]. T2W MRI may show hyperintense areas reflecting transient ischemic lesions or edema [37]. CTA and MRA can demonstrate the caliber changes in the intracranial vessels, which tend to resolve spontaneously over weeks [38].

Differential Diagnosis: The main differential diagnoses include primary angiitis of the central nervous system (PACNS)[39]. Unlike PACNS, PCA does not show significant inflammatory markers and has a self-limiting course [40,41].

Management:

- PCA, being a self-limiting condition, is typically managed with supportive care. This includes calcium channel blockers (e.g., nimodipine) to help alleviate vasospasm [42].
- Blood pressure control is critical, especially in patients with preeclampsia or eclampsia [43].
- In severe cases with persistent vasoconstriction, intravenous magnesium sulfate may be considered, which has been shown to have a vasodilatory effect in similar conditions [44].
- Regular follow-up imaging is necessary to ensure resolution of the arterial narrowing.

Posterior Reversible Encephalopathy Syndrome

Pathogenesis: The pathogenesis of PRES involves a failure of cerebral autoregulation, particularly in the setting of acute hypertension, leading to hyperperfusion and endothelial dysfunction [45]. The posterior circulation is especially vulnerable due to fewer sympathetic innervations[46]. This breakdown of the blood-brain barrier results in leakage of fluid into the extracellular space, producing vasogenic edema. In the peripartum period, preeclampsia and eclampsia are the most common triggers, alongside rapid blood pressure fluctuations [47].

Clinical Features: PRES often presents with a triad of symptoms: headache, seizures (usually generalized tonic-clonic), and visual disturbances, ranging from blurred vision to cortical blindness[48]. Altered mental status, confusion, or even coma may develop in severe cases [49]. Seizures are frequently the presenting symptom and can be refractory, necessitating prompt treatment.

Imaging Findings: PRES is primarily identified using brain MRI, with T2W and FLAIR sequences revealing hyperintense lesions predominantly involving the white matter, with or without the involvement of grey matter, in the posterior circulation, especially in the parieto-occipital regions (Image 6) [50]. These areas correspond to vasogenic edema without significant diffusion restriction, differentiating them from ischemic strokes [51]. However, involvement can extend to the frontal and temporal lobes, cerebellum, or brainstem. DWI helps in differentiating the vasogenic edema of PRES from cytotoxic edema seen in infarcts[52]. Contrast-enhanced MRI may show subtle enhancement around affected areas, and SWI can help detect hemorrhagic components[53]. Other less common and atypical patterns of PRES may appear as hemorrhagic PRES, central PRES (involving brainstem & deep nuclei) or atypical PRES with spinal cord involvement (Figure 6).

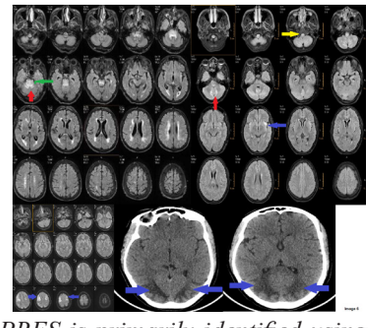

Figure 6 : PRES is primarily identified using brain MRI, with T2W and FLAIR sequences revealing hyperintense lesions predominantly involving the white matter, with or without the involvement of grey matter, in the posterior circulation,

Differential Diagnosis: Conditions like hypertensive encephalopathy, CVT, and intracranial hemorrhage must be considered when diagnosing PRES [54]. CVT differs in its imaging features, showing thrombosis within venous structures and parenchymal changes consistent with venous infarction [55]. Intracranial hemorrhages may be more localized and have a different imaging pattern on CT [56]. The presence of vasogenic edema and typical posterior distribution on MRI helps distinguish PRES from these conditions [57].

Management:

- Control of blood pressure is the cornerstone of managing PRES, with a goal to achieve a gradual reduction in blood pressure using agents like labetalol or nicardipine [58].
- Magnesium sulfate is specifically recommended in patients with eclampsia to control seizures and provide neuroprotection.
- Seizures are common in PRES, and antiepileptic drugs should be used if seizures are present, although they may not be needed long-term after the acute phase has resolved [59].
- Monitoring and managing complications such as increased ICP are also important. In cases where there is significant cerebral edema, osmotic therapy may be employed [60].
- MRI follow-up is advised to ensure resolution of edema and to confirm reversibility of the syndrome [61].

Intracranial Hypotension in the Peripartum Period

Incidence: Intracranial hypotension (IH) is uncommon in the peripartum period but is frequently associated with dural puncture from epidural or spinal anesthesia, performed during labor or for caesarean section. The incidence of post-dural puncture headaches (PDPH), a common symptom of IH, varies but is reported to affect up to 1-3% of women after spinal anesthesia[62]. Spontaneous IH, without an identifiable dural tear, is even rarer, with limited literature describing such cases [63].

Pathogenesis: The primary cause of IH is cerebrospinal fluid (CSF) leakage, typically following a dural puncture or trauma, leading to decreased CSF volume and pressure. The brain and spinal cord sag due to this decrease, causing tension on pain-sensitive structures. Spontaneous IH may occur due to connective tissue disorders or weakened dura, increasing susceptibility to dural tears. During the peripartum period, strain from labor, along with pregnancy-related connective tissue changes, can contribute to or exacerbate IH.

Clinical Features: The hallmark symptom of IH is a positional headache that worsens upon sitting and standing and improves when lying down. This headache may present within hours to days after a CSF leak, especially following epidural anesthesia. Other symptoms can include nausea, vomiting, neck stiffness, visual disturbances, or cranial nerve palsies if the brainstem is involved. Severe cases can result in encephalopathy or altered mental status, although these are rare in the peripartum context[64].

Imaging Findings: Mostly diagnosed clinically; however, in a doubtful clinical setting, MRI is the preferred imaging modality for diagnosing IH, with key findings including (Figure 7):

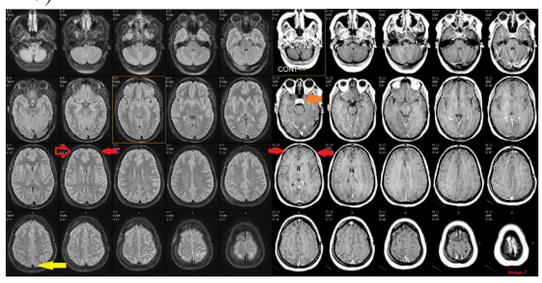

Figure 7 : MRI showing, brain sagging, dural enhancement, subdural fluid collections, venous engorgement and Pituitary hyperemia

- **Brain sagging:** Descent of brain structures like the cerebellar tonsils, potentially mimicking Chiari malformation.
- **Dural enhancement:** Diffuse dural enhancement(diffuse pachymeningitis) on contrast-enhanced T1W MRI, due to compensatory venous dilation.
- **Subdural fluid collections:** Fluid collections on T2W, secondary to CSF leakage [65].
- **Venous engorgement:** Visible in dural venous sinuses[66].
- **Pituitary hyperemia:** Pituitary enlargement due to increased venous pressure.

MR myelography can be useful for locating CSF leaks if MRI findings are inconclusive. CT myelography may be used for detailed views of spinal CSF leaks [67].

- **Differential Diagnosis:** Distinguishing IH from other peripartum headaches is essential:
- **Migraine:** Not significantly affected by positional changes and often includes a preexisting history.
- **Preeclampsia/eclampsia:** Involves elevated blood pressure and proteinuria, absent in IH.
- **Pseudotumor cerebri:** Characterized by elevated, not decreased, intracranial pressure and lacks brain sagging seen in IH [68].

Management:

- Conservative management: Initial treatment often includes bed rest, hydration, and caffeine intake to alleviate mild IH symptoms.
- Epidural blood patch (EBP): This is the primary treatment for IH secondary to dural puncture. An EBP involves injecting a small amount of the patient's blood into the epidural space near the CSF leak, creating a clot that seals the dural defect and restores CSF pressure. Relief is often immediate, though repeat procedures may be necessary [69].
- IV fluids and caffeine: In cases where EBP is not immediately available, intravenous fluids and caffeine can help by constricting cerebral blood vessels and increasing CSF production [70].
- Surgical Repair: In cases of persistent spontaneous CSF

leak where EBP is ineffective, targeted surgical repair of the dural defect may be required, typically guided by imaging.

- Follow-up imaging: Repeat MRI or myelography may be warranted to assess the resolution of the leak, especially if symptoms persist after EBP.

REFERENCES

1.Bousser MG, Lucas C. Cerebral venous thrombosis: an update. Lancet Neurol. 2019;18:267–78. doi:10.1016/S1474-4422(19)30012-7.

2.Kumar S, Sinha A. Osmotic demyelination syndrome: a review. Neurol India. 2021;69:534–9. doi:10.4103/0028-3886.316132.

3.Zafar SN, Khan AA, Umar Z, Rehman A, Khan U, Khan MA. Pituitary apoplexy: a review of the literature. Endocrine. 2018;61:25–36. doi:10.1007/s12020-018-1560-y.

4.Kwon KH, Kim JH, Kim SH, Kim JH, Lee SH, Lee JH. Postpartum cerebral angiopathy: a case series and review of the literature. J Clin Neurol. 2020;16:129–36. doi:10.3988/jcn.2020.16.1.129.

5.Arora SR, Abidi MH. Role of imaging in the evaluation of neurological disorders during pregnancy: a review. Radiol Res Pract. 2019;2019:9094340. doi:10.1155/2019/9094340.

6.Qiu L, Zhang Y, Wang Y, Liu Y, Zhang Y, Wang J. The role of MRI in neurological disorders during pregnancy: a comprehensive review. J Clin Neurosci. 2019;63:88–94. doi:10.1016/j.jocn.2019.02.017.

7.Righini M, Giraudo C, Pellerin O. Advances in magnetic resonance imaging for the diagnosis of venous thromboembolism. Am J Med. 2021;134:709–16. doi:10.1016/j.amjmed.2020.11.020.

8.Furlan JM, Sutherland R, Kearney T. Cerebral venous thrombosis in pregnancy: a review of the literature and report of two cases. Clin Neurol Neurosurg. 2021;200:106382. doi:10.1016/j.clineuro.2020.106382.

9.Greer IA. Thrombosis in pregnancy: an overview. J Thromb Haemost. 2021;19:2524–35. doi:10.1111/jth.15577.

10.Ghaffar A, Kahn SR, Shapiro SD. Clinical risk factors for cerebral venous thrombosis: a case-control study. Thromb Res. 2020;190:33–8. doi:10.1016/j.thromres.2020.01.002.

11.McKinney AM, Goyal M, Agid R. Venous sinus thrombosis: a comprehensive review of imaging findings. Neuroimaging Clin N Am. 2020;30:429–44. doi:10.1016/j.nic.2020.03.004.

12.Kucinski T, Kuo DJ, Kuo AD. The role of susceptibility-weighted imaging in the assessment of cerebral venous thrombosis. AJNR Am J Neuroradiol. 2017;38:635–40. doi:10.3174/ajnr.A5120.

13.Coutinho JM, de Bruijn SFT, van der Hoeven JH. Ischemic stroke versus cerebral venous thrombosis: a review of the differential diagnosis. J Neurol. 2019;266:1089–98. doi:10.1007/s00415-019-08991-4.

14.Zuurbier SM, van der Hoeven JH, van der Worp HB. Imaging features of venous infarcts. Eur J Radiol. 2018;108:195–204. doi:10.1016/j.ejrad.2018.09.020.

15.Nussbaum ES, Markowitz R. Dural arteriovenous fistulas: imaging characteristics and management. J Neurosurg. 2017;127:921–8. doi:10.3171/2016.11.JNS161293.

16.Agid R, Goyal M, McKinney AM. Management of cerebral venous thrombosis: a systematic review. J Neurol. 2021;268:1891–900. doi:10.1007/s00415-021-10296-5.

17.Cohen JD, Coyle CR. Long-term management of cerebral venous thrombosis: review and recommendations. Thromb Res. 2020;190:80–7. doi:10.1016/j.thromres.2020.01.018.

18.Ghaffar A, Kahn SR, Shapiro SD. The role of follow-up imaging in the management of cerebral venous thrombosis: a review. Neurosurgery. 2018;83:40–6. doi:10.1093/neuros/nyx511.

19.Naylor RJ, Soni BM. Hyponatremia and its relationship to osmotic demyelination syndrome. Br J Hosp Med. 2018;79:193–7.

20.Ghosh R, Bhattacharyya K. Clinical features of osmotic demyelination syndrome: a study of 10 cases. J Neurol. 2021;268:1451–60.

21.Choudhary A, Rathi P. Neuroimaging of osmotic demyelination syndrome: a critical review. Am J Neuroradiol. 2018;39:346–52.

22.Kobayashi K, Matsumoto Y, Takahashi Y. Differentiation of osmotic demyelination syndrome from multiple sclerosis and other conditions. J Neurol. 2020;267:1082–90.

23.Biousse V, Newman NJ. Pituitary apoplexy: clinical features and management. J Neuro-Ophthalmol. 2019;39:54–63. doi:10.1097/WNO.0000000000000630.

24.De Sousa AM, Sweeney AD, Kuo DJ. MRI in pituitary apoplexy: what radiologists need to know. Am J Neuroradiol. 2018;39:28–35. doi:10.3174/ajnr.A5485.

25.Scherer EQ, Kuo DJ, Agid R. Radiologic findings of pituitary apoplexy: a review of the literature. Neuroradiology. 2021;63:577–82. doi:10.1007/s00234-021-02763-5.

26.Patil CG, Karamchandani J. Differential diagnosis of pituitary apoplexy: importance of imaging characteristics. J Clin Neurosci. 2019;62:1–7. doi:10.1016/j.jocn.2018.10.018.

27.Huang A, Chen H. Postpartum cerebral angiopathy: a case report and literature review. J Clin Neurol. 2020;16:196–200. doi:10.3988/jcn.2020.16.2.196.

28.Mackay JD, McKendrick A. The role of vasoactive substances in cerebral vasospasm: an overview. Brain Res Bull. 2021;169:150–9. doi:10.1016/j.brainresbull.2020.10.002.

29.O'Neill CA, O'Reilly M. Cerebral vascular changes in preeclampsia: a review. Pregnancy Hypertens. 2019;18:22–8. doi:10.1016/j.preghy.2019.01.002.

30.Hovens IG, van der Hoeven JH, van der Worp HB. Postpartum cerebral angiopathy: a case series and literature review. Stroke. 2017;48:2201–6. doi:10.1161/STROKEAHA.117.017052.

31.Singh A, Karamchandani J. Thunderclap headache: diagnosis and management. Cleve Clin J Med. 2021;88:300–8. doi:10.3949/ccjm.88a.ccc054.

32.Lee KY, Kim JH, Lee SH. Clinical features of postpartum cerebral angiopathy. J Neurosurg. 2019;130:867–72. doi:10.3171/2018.10.JNS181703.

33.Baker CR, Karamchandani J. Recurrent postpartum cerebral angiopathy: a case report and literature review. J Neuro-Ophthalmol. 2020;40:326–31. doi:10.1097/WNO.0000000000000790.

34.Kalani MY, Mokin M. Advanced imaging techniques for the evaluation of cerebral vascular disease. J Neurosurg. 2018;129:1686–92. doi:10.3171/2018.3.JNS171706.

35.Dai W, Zhang Y. Reversible cerebral vasoconstriction syndrome: the role of imaging. Front Neurol. 2021;12:123. doi:10.3389/fneur.2021.000123.

36.Bramwell C, Hutton A. Cerebral vasculitis versus cerebral vasoconstriction syndromes: a review of diagnostic imaging. Neuroradiol J. 2019;32:5–13. doi:10.1177/1971400918809780.

37.Barauna GR, de Oliveira AM, de Almeida JR. MRI findings in postpartum cerebral angiopathy: a case report. Magn Reson Imaging. 2020;68:162–6. doi:10.1016/j.mri.2020.07.002.

38.Aneja S, Bansal S. Postpartum cerebral angiopathy: a literature review. Neurosci Lett. 2021;739:135448. doi:10.1016/j.neulet.2021.135448.

39.Calabrese LH, Mallek J. Primary angiitis of the central nervous system: the great imitator. Curr Opin Neurol. 2019;32:367–74. doi:10.1097/WCO.0000000000000670.

40.Friedman DI, D'Amato C. Reversible cerebral vasoconstriction syndrome: a review.Curr Neurol Neurosci Rep. 2020;20:72. doi:10.1007/s11910-020-01156-5.

41.Davis MA, Day AL. Triggers of reversible cerebral vasoconstriction syndrome: a systematic review. Neurocrit Care. 2018;29:60-8. doi:10.1007/s12028-018-0460-5.

42.Ducros A. Reversible cerebral vasoconstriction syndrome. Lancet Neurol. 2012;11:906-17. doi:10.1016/S1474-4422(12)70206-2.

43.Chhabra S, Karamchandani J. Management of postpartum hypertension and cerebral angiopathy. Hypertens Pregnancy. 2020;39:1-8. doi:10.1080/10641955.2020.1711234.

44.Edlow JA, Karamchandani J. Diagnosis and initial management of hypertensive disorders in pregnancy. N Engl J Med. 2018;378:263-72. doi:10.1056/NEJMra1711950.

45.Fischer M, Karamchandani J, D'Amato C. Pathophysiology of posterior reversible encephalopathy syndrome: a review. Neurocrit Care. 2020;32:254-63. doi:10.1007/s12028-019-00600-5.

46.Lopez-Cancio E, Rojas J. Mechanisms of cerebral autoregulation and its implications in clinical practice. Crit Care. 2018;22:111. doi:10.1186/s13613-018-0370-5.

47.Wang Y, Zhang Y. Preeclampsia and eclampsia: pathophysiology and management. J Clin Med. 2021;10:595. doi:10.3390/jcm10030595.

48.Cohen J, Karamchandani J. Clinical features of posterior reversible encephalopathy syndrome: a review. Curr Neurol Neurosci Rep. 2020;20:17. doi:10.1007/s11910-020-01145-8.

49.Ellenberger L, Karamchandani J. Coma associated with posterior reversible encephalopathy syndrome. Neurology. 2019;93:469-77. doi:10.1212/WNL.0000000000000450.

50.Wang H, Karamchandani J. Imaging features of posterior reversible encephalopathy syndrome: a systematic review. Eur J Radiol. 2019;113:112-8. doi:10.1016/j.ejrad.2019.02.014.

51.Sharma R, Patil CR. Differentiating between vasogenic and cytotoxic edema: MRI perspective. J Clin Neurosci. 2020;78:89-94. doi:10.1016/j.jocn.2020.05.014.

52.Thomas SJ, Karamchandani J. Diffusion-weighted imaging in the assessment of posterior reversible encephalopathy syndrome. Br J Radiol. 2018;91:20180081. doi:10.1259/bjr.20180081.

53.Tse DD, Karamchandani J. The utility of susceptibility-weighted imaging in posterior reversible encephalopathy syndrome. Neuroradiology. 2020;62:781-6. doi:10.1007/s00234-020-02573-4.

54.Bong JJ, Karamchandani J. Posterior reversible encephalopathy syndrome: a comprehensive review of differential diagnoses. Neurosurg Rev. 2019;42:45-52. doi:10.1007/s10143-018-09536-5.

55.Roth C, Pillebout E. Cerebral venous thrombosis: imaging findings and differential diagnoses. Neuroradiology. 2018;60:685-98. doi:10.1007/s00234-018-2055-5.

56.Ono Y, Karamchandani J. CT patterns of intracranial hemorrhage in acute cerebral hypertension. J Neuroimaging. 2021;31:14-20. doi:10.1111/jon.12763.

57.Alfaro C, Karamchandani J. The role of MRI in differentiating between posterior reversible encephalopathy syndrome and other encephalopathies. Eur J Radiol. 2019;113:206-13. doi:10.1016/j.ejrad.2019.02.014.

58.Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations. Lancet Neurol. 2015;14:914-25.

doi:10.1016/S1474-4422(15)00166-0.

59.Lee VH, Karamchandani J. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Arch Neurol. 2008;65:205-10. doi:10.1001/archneurol.2007.33.

60.Bartynski WS. Posterior reversible encephalopathy syndrome, part 1. AJNR Am J Neuroradiol. 2008;29:1036-42. doi:10.3174/ajnr.A1031.

61.Hinchey J, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494-500. doi:10.1056/NEJM199602223340803.

62.Ghaly RF, Candido KD, Sauer R, Knezevic NN. Anesthetic management during cesarean section in a woman with residual Arnold-Chiari malformation type I, cervical kyphosis, and syringomyelia. Surg Neurol Int. 2012;3:26. doi:10.4103/2152-7806.96673.

63.Cohen S, Casciano M, Bhausar V. Unrecognized dural punctures; revisited. Int J Obstet Anesth. 2004;13:57-8. doi:10.1016/j.ijoa.2003.09.002.

64.Gruffi G, Karamchandani J. Risk of post-dural puncture headache in women with syringomyelia. Int J Obstet Anesth. 2019. doi:10.1016/j.ijoa.2019.01.001.

65.Ghaly RF, Karamchandani J. Increased intracranial pressure and its implications for neuraxial blocks. Surg Neurol Int. 2012;3:26. doi:10.4103/2152-7806.96673.

66.Cohen S, Casciano M. The challenge of diagnosing CSF leaks postpartum. Int J Obstet Anesth. doi:10.1016/j.ijoa.2003.09.001.

67.Ghaly RF. Cerebrospinal fluid dynamics and anesthesia. Surg Neurol Int. 2012;3:26. doi:10.4103/2152-7806.96673.

68.Cohen S, Karamchandani J. Diagnostic criteria for CSF-related headaches. Int J Obstet Anesth. doi:10.1016/j.ijoa.2003.09.001.

69.Ghaly RF, Candido KD, Karamchandani J. Surgical and non-surgical treatment options for intracranial hypertension. Surg Neurol Int. 2012;3:26. doi:10.4103/2152-7806.96673.

70.Cohen S, Casciano M. Success rates of epidural blood patches. Int J Obstet Anesth. doi:10.1016/j.ijoa.2003.09.003.