

**Postpartum seizure following emergency LSCS under spinal anaesthesia: a diagnostic dilemma between post-dural puncture headache and postpartum eclampsia with PRES**

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Abstract

Background: Acute seizures in the immediate postpartum period are potentially life-threatening events requiring prompt recognition, seizure control, airway and haemodynamic stabilization, and evaluation of the underlying cause. Although eclampsia is an important consideration, postpartum seizures may also occur because of metabolic, cerebrovascular and neurological disorders.

Case Presentation: This case illustrates the diagnostic challenge of a postpartum seizure occurring after neuraxial anaesthesia with uneventful intra op events. The presence of two failed spinal attempts initially raised concern regarding a possible post-dural puncture headache/CSF leak. However, the subsequent

development of severe hypertension and generalized seizure required consideration of postpartum eclampsia and other neurological causes. The MRI was particularly important. It demonstrated bilateral parieto-occipital and cerebellar vasogenic edema without diffusion restriction or haemorrhage, and the radiologist reported the findings as consistent with PRES. Therefore, the available clinical and radiological evidence supports PRES in the setting of postpartum hypertensive/eclamptic neurological disease rather than uncomplicated PDPH as the principal explanation for the seizure. It is important, however, not to attribute causality solely from the temporal relationship with spinal anaesthesia. The two failed neuraxial attempts are clinically relevant to the differential diagnosis, but the MRI pattern and

associated acute hypertension provide stronger evidence for the PRES pathway

Conclusion: This case highlights the importance of early recognition and multidisciplinary management of acute postpartum seizures following caesarean delivery. Anaesthesiologists have a central role in airway management, oxygenation, seizure control and haemodynamic stabilization. A postpartum seizure should be evaluated systematically, with consideration of eclampsia as well as metabolic, cerebrovascular and other neurological causes.

Keywords: Postpartum Seizure, Caesarean Section, Eclampsia, Obstetric Cholestasis, Anaesthesia, Magnesium Sulfate, Levetiracetam, Intensive Care.

Introduction

Postpartum seizures represent a potentially life-threatening obstetric and neurological emergency because they can rapidly lead to maternal hypoxia, aspiration, cerebral injury and cardiovascular instability. Eclampsia remains an important cause of seizures during pregnancy and the puerperium, but seizures may also result from epilepsy, metabolic abnormalities, cerebrovascular disease and other neurological disorders.^{1,2}

The diagnostic approach to a pregnant or postpartum woman with a new-onset seizure should therefore remain broad. Cerebral venous sinus thrombosis, posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome, intracranial haemorrhage and ischemic stroke may present with overlapping neurological manifestations and should be considered when clinically appropriate.³⁻⁵ From an anaesthetic and critical-care perspective, immediate management should focus on airway protection, adequate oxygenation, circulation, termination of seizure

activity and correction of potentially reversible metabolic abnormalities. Continuous haemodynamic and neurological monitoring is particularly important when seizures occur in the immediate postoperative period.^{6,7} Magnesium sulfate has an established role in the management and prevention of seizures associated with preeclampsia and eclampsia. Evidence regarding the optimal duration of postpartum magnesium therapy continues to be evaluated, although magnesium remains central to obstetric seizure management when eclampsia is clinically suspected.⁸⁻¹⁰

The present case describes an acute generalized seizure occurring approximately 2 hours after emergency caesarean section in a term primigravida with documented obstetric cholestasis. The patient required urgent anaesthetic intervention, anticonvulsant therapy, oxygen supplementation and intensive-care monitoring, highlighting the importance of rapid multidisciplinary management of acute postpartum neurological events.

Materials and Methods

This case report was prepared from the clinical records of a term primigravida who underwent emergency lower-segment caesarean section (LSCS) and subsequently developed an acute generalized seizure in the immediate postpartum period. The clinical information included the obstetric history, indication for caesarean delivery, operative and anaesthetic records, postoperative observations, emergency seizure management, laboratory investigations, arterial blood gas analysis, intensive-care management and documented maternal and neonatal outcomes.

The patient was approximately 40 weeks pregnant and had documented obstetric cholestasis. The pregnancy was also complicated by suspected fetal growth restriction, oligohydramnios and a non-reassuring non-

stress test. Emergency LSCS was performed under anaesthetic care, and a live female infant weighing 2600 g was delivered at 4:05 PM.

Postoperatively, the patient was monitored closely. Approximately 2 hours after delivery, she developed a generalized convulsive episode characterized by teeth clenching, upward rolling of the eyes, frothing from the mouth and jerky movements involving all four limbs. Her blood pressure during the episode was documented as 164/100 mmHg.

Immediate assessment and management were undertaken by the anaesthesia and medical teams. Oxygen supplementation, airway and respiratory assessment, magnesium sulfate and anticonvulsant therapy were administered, followed by continued intensive monitoring. Laboratory investigations included blood glucose, complete blood count, serum electrolytes, renal and liver-function assessment and arterial blood gas analysis.

Arterial blood gas analysis during the acute event demonstrated a pH of 7.27, PaO₂ of 178.7 mmHg, bicarbonate of 9.06 mmol/L, base excess of -16.5 and lactate of 4.59 mmol/L. Following stabilization, the patient was managed in the intensive-care unit with continuous neurological and haemodynamic monitoring. Levetiracetam was administered, and fluid management with consideration of vasopressor support was documented according to haemodynamic status.

The clinical course was reviewed from the available hospital documentation, including postoperative observations and neurological assessment. The case was evaluated with emphasis on the acute anaesthetic management, seizure control, haemodynamic stabilization and multidisciplinary critical-care management. The available records did not provide

sufficient information to establish a definitive underlying neurological aetiology or complete long-term maternal follow-up; therefore, these aspects were not inferred beyond the documented clinical findings.

Acute Postpartum Neurological Event

Approximately **2 hours after caesarean delivery**, the patient developed shivering which was managed medically by Obs and gynae team (Inj. Dexamethasone and Inj. Avil) consequently after the medical management by obs and gynae team at around **6:32 PM**, the patient developed her first documented generalized seizure

The clinical episode was described by:

- Clenching of the teeth
- Upward rolling of the eyes
- Frothing from the mouth
- Jerky movements involving both upper and lower limbs
- Associated headache documented in the ICU record

During the episode, her recorded blood pressure was **164/100 mmHg**. Pulse was approximately 62/min, she was afebrile, and oxygen saturation was approximately 98% on room air.

The sudden neurological deterioration prompted an urgent anaesthetic and medical response.

Emergency Management after call from Team of Obs and Gynae to Team Anesthesia and Medicine

The anaesthesia team was called while the seizure was ongoing. Immediate management focused on stabilization and seizure control.

The documented emergency management included:

1. Oxygen supplementation
2. Airway and respiratory assessment
3. Magnesium sulfate therapy
4. Intravenous anticonvulsant therapy

5. Urgent anaesthetic assessment
 6. Continuous vital-sign monitoring
 7. Blood glucose assessment
 8. Electrolyte, renal and liver-function assessment
 9. Arterial blood gas analysis
 10. ECG and chest assessment as clinically indicated
- The patient remained agitated after initial treatment and required continued medical management and close observation.

Arterial Blood Gas Findings

Arterial blood gas analysis obtained during the acute episode demonstrated:

Parameter	Recorded value
pH	7.27
PaO ₂	178.7 mmHg
HCO ₃ ⁻	9.06 mmol/L
Base excess	-16.5
Lactate	4.59 mmol/L

These findings demonstrated significant metabolic derangement during the acute neurological event. The low bicarbonate and negative base excess were accompanied by elevated lactate, which occurred in the setting of generalized convulsive activity.

Management

As soon as the Anaesthesia Team arrived, the patient was found restless

1. For her restlessness, Inj Midazolam (3mg) IV STAT was given
2. For Diagnosed Metabolic Acidosis according to ABG, Inj Soda Bicarb was given according to the norms
3. For elevated Lactate levels, proper fluid resuscitation was done

4. For further management and monitoring the patient was shifted to SICU
 5. The Obgy team sent a neurosurgery reference in view of the seizure activity after which the neuro team advised investigations (CECT Head and MRI Brain) and for seizures Inj. Leverta was advised
- After 24 Hours In SICU, patient developed the following episodes

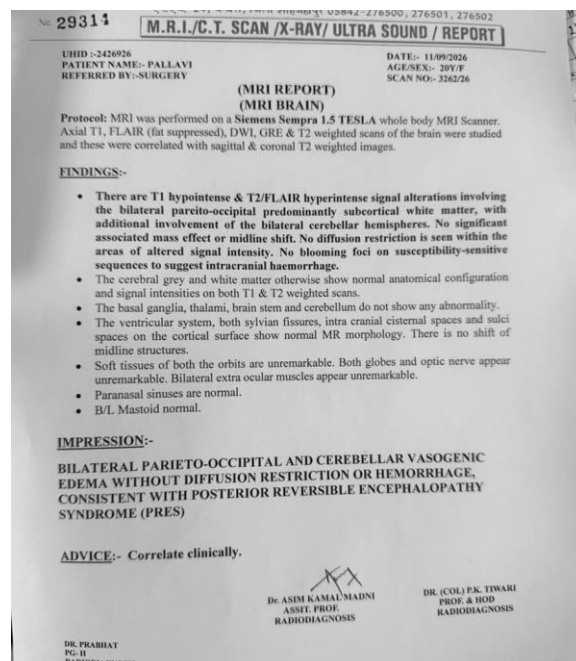
1. Blurring of vision
2. Headache
3. Raised MAP and Blood Pressure along with Tachycardia

Management

1. Inj. Midazolam (2mg) IV STAT
2. Inj. Labetalol (10 mg) IV STAT
3. Inj. PCM (1 gm) IV STAT

After stabilizing the patient, Neuro team was informed for this consequent episode in the patient, for which they visited and after the thorough general and systemic examination MRI Head was planned as a further investigation

MRI Findings



Case Report

Age: 20 years Obstetric status: Primigravida Gestational age: 40 weeks + 1 day Date of admission: 10/09/2026 Procedure: Emergency LSCS under spinal anaesthesia Indication: Non-reassuring NST with oligohydramnios and suspected IUGR Antenatal history: Obstetric cholestasis with generalized itching during the 8th month of pregnancy Admission BP: 120/70 mmHg Pulse: 94/min SpO₂: 99% on room air

Clinical Presentation and Preoperative Course

A 20-year-old primigravida at 40+1 weeks of gestation presented with amenorrhoea of approximately 9 months. She had a history of generalized itching during the 8th month of pregnancy and was diagnosed with obstetric cholestasis.

On evaluation, she was diagnosed with:

- Term pregnancy at 40+1 weeks
- Suspected IUGR
- Oligohydramnios
- Non-reassuring NST
- Obstetric cholestasis

Her initial blood pressure was 120/70 mmHg, with no documented history of antenatal hypertension or pre-eclampsia. In view of the non-reassuring fetal status and oligohydramnios, emergency LSCS was planned.

Anaesthetic Management

Emergency LSCS was performed under spinal anaesthesia on 10/09/2026. There were two failed attempts at spinal anaesthesia, following which successful spinal anaesthesia was achieved. The intraoperative period was uneventful.

Immediately following surgery:

- BP: 130/80 mmHg
- HR: 60/min
- SpO₂: 98% on room air

- Temperature: 96.5°F
- Urine output after OT: approximately 500 mL

Postoperative GICU Transfer

After completion of surgery, the patient was shifted to GICU for postoperative monitoring and care. At this stage, her postoperative vital parameters were stable.

After 2 Hours in GICU – Development of Postoperative Neurological Symptoms

While in the postoperative period, the patient developed headache and blurring of vision.

Her blood pressure subsequently increased to:

164/100 mmHg

She then developed a generalized convulsive seizure lasting approximately 1 minute, characterized by:

- Clenching of teeth
- Upward rolling of eyes
- Frothing from the mouth
- Jerky movements involving both upper and lower limbs

Immediate ABC assessment and stabilization were performed.

Immediate Management

- Airway, breathing and circulation assessed and stabilized
- MgSO₄ loading dose administered
- Labetalol 20 mg IV slowly
- Urgent anaesthesia and medicine consultations
- Midazolam 2 mg IV
- ABG performed
- ECG monitoring
- Chest X-ray advised
- Neuroimaging advised
- Neurology/neurosurgery consultation obtained
- Levetiracetam 1 g IV stat, followed by 500 mg IV 12-hourly

ABG showed metabolic acidosis, and sodium bicarbonate was administered. IV fluids and electrolyte monitoring were continued.

Diagnostic Dilemma

The development of severe headache and seizure after spinal anaesthesia created an important diagnostic dilemma.

1. Anaesthesia-Related Complication

Because the patient had undergone two failed spinal attempts, a post-dural puncture complication, particularly PDPH due to CSF leakage, was considered.

A post-dural puncture headache can occur following inadvertent dural puncture and is characteristically postural. However, in this patient, the clinical picture included:

- Severe headache
- Acute hypertension
- Generalized seizure
- Recurrent episodes of hypertension
- MRI evidence of vasogenic cerebral edema

These findings were not typical of uncomplicated PDPH alone.

2. Obstetric Neurological Complication

The alternative diagnosis was postpartum eclampsia with PRES.

The patient had no documented antenatal hypertension, but postpartum eclampsia can present after delivery, including in patients without previously documented hypertension.

The combination of severe headache, acute hypertension and generalized seizure raised strong clinical suspicion for a hypertensive neurological complication of pregnancy.

Subsequent Clinical Course

On 11/09/2026, the patient had another episode of elevated blood pressure:

BP: 160/108 mmHg HR: 114/min

This episode was associated with agitation and restlessness, but no further seizure was documented.

She was managed with:

- Midazolam IV 1 mg
- Labetalol 10 mg IV slowly

Following treatment, her BP improved to approximately 130/90 mmHg.

Serum electrolytes were reported to be within normal limits. Serum calcium was 8.0 mg/dL, and calcium supplementation was advised.

Neurology/neurosurgery consultation advised continuation of levetiracetam 500 mg IV 12-hourly and MRI brain evaluation, which subsequently demonstrated the following findings.

MRI Brain Findings

MRI brain was performed on 11/09/2026.

MRI findings:

- T1 hypointense and T2/FLAIR hyperintense signal alterations involving the bilateral parieto-occipital predominantly subcortical white matter
- Additional involvement of the bilateral cerebellar hemispheres
- No significant mass effect or midline shift
- No diffusion restriction within the areas of altered signal
- No blooming focus on susceptibility-sensitive sequences to suggest intracranial haemorrhage
- Basal ganglia, thalami, brainstem and cerebellum otherwise without focal abnormality
- Ventricular system and intracranial CSF spaces were normal

MRI Impression

Bilateral parieto-occipital and cerebellar vasogenic edema without diffusion restriction or haemorrhage, consistent with Posterior Reversible Encephalopathy Syndrome (PRES).

This MRI finding provides important radiological evidence supporting PRES as the explanation for the patient's neurological presentation.

Urine output remained adequate, approximately 2410 mL over 24 hours.

Differential Diagnosis

Differential	Supporting features	Features against/limitations
Postpartum eclampsia/PRES	Severe headache, acute hypertension, generalized seizure, MRI showing bilateral parieto-occipital/cerebellar vasogenic edema	No documented antenatal hypertension
PDPH/post-dural puncture complication	Two failed spinal attempts and postoperative headache	Does not adequately explain acute hypertension, seizure and characteristic PRES MRI findings
Cerebral venous sinus thrombosis	Postpartum state, headache and seizure	MRI reported findings consistent with PRES
Metabolic cause	Metabolic acidosis was present	Electrolytes were reported normal and MRI demonstrated PRES-type edema
Drug/toxin-related event	History of tobacco-containing toothpaste consumption was subsequently revealed	Does not explain the characteristic MRI findings

The MRI brain demonstrates bilateral parieto-occipital and cerebellar areas of abnormal signal intensity, appearing hypointense on T1-weighted images and hyperintense on T2/FLAIR sequences, predominantly involving the subcortical white matter. There is no

significant mass effect or midline shift, and the affected areas show no diffusion restriction, suggesting the absence of acute infarction. Susceptibility-sensitive sequences show no evidence of intracranial hemorrhage. The remaining cerebral gray and white matter, basal ganglia, thalami, brainstem, ventricles, cortical sulci, cisternal spaces, orbits, optic nerves, paranasal sinuses, and bilateral mastoid regions are otherwise unremarkable. Overall, the radiological findings are consistent with bilateral parieto-occipital and cerebellar vasogenic edema, without diffusion restriction or hemorrhage, and are suggestive of Posterior Reversible Encephalopathy Syndrome (PRES). Clinical correlation, particularly with blood pressure, neurological symptoms, renal function, pregnancy/postpartum status, medications, or other potential precipitating factors, is recommended.

Discussion

This case illustrates the diagnostic challenge of a postpartum seizure occurring after neuraxial anaesthesia. The presence of two failed spinal attempts initially raised concern regarding a possible post-dural puncture headache/CSF leak. However, the subsequent development of severe hypertension and generalized seizure required consideration of postpartum eclampsia and other neurological causes. The MRI was particularly important. It demonstrated bilateral parieto-occipital and cerebellar vasogenic edema without diffusion restriction or haemorrhage, and the radiologist reported the findings as consistent with PRES. Therefore, the available clinical and radiological evidence supports PRES in the setting of postpartum hypertensive/eclamptic neurological disease rather than uncomplicated PDPH as the principal explanation for the seizure. It is important, however, not to attribute causality solely from the

temporal relationship with spinal anaesthesia. The two failed neuraxial attempts are clinically relevant to the differential diagnosis, but the MRI pattern and associated acute hypertension provide stronger evidence for the PRES pathway.

In the present case, the seizure occurred approximately 2 hours after caesarean delivery and was characterized by generalized jerky movements, teeth clenching, upward rolling of the eyes and frothing from the mouth. The simultaneous blood pressure elevation to 164/100 mmHg raised concern for a hypertensive obstetric neurological event. However, the available records do not document a definitive diagnosis of eclampsia. Therefore, the episode is appropriately described as an acute postpartum seizure while maintaining a broad differential diagnosis.⁴⁻⁶

The immediate response in this patient involved anaesthesia, obstetrics, medicine and critical-care teams. From an anaesthetic perspective, management of an ongoing generalized seizure requires attention to airway patency, oxygenation, ventilation and circulation while simultaneously treating the convulsive activity. Continuous monitoring is important because prolonged seizures may produce hypoxia, aspiration, acidosis and cardiovascular instability.^{7,8}

Magnesium sulfate was administered during the acute event. Magnesium sulfate remains the recommended treatment for eclamptic seizures and is also recommended by the World Health Organization for treatment of eclampsia in preference to other anticonvulsants.^{9,10} In patients with postpartum hypertension accompanied by neurological symptoms, magnesium sulfate may be particularly relevant when an eclamptic seizure is clinically suspected.¹¹

The patient subsequently received levetiracetam as anticonvulsant therapy. The use of an additional

anticonvulsant in an acute seizure requires consideration of the clinical context and the possibility of causes other than eclampsia. Importantly, the documentation in this case does not establish a chronic seizure disorder or a definitive neurological diagnosis, so the anticonvulsant treatment should be interpreted as part of the acute clinical management rather than evidence of epilepsy.

The arterial blood gas obtained during the seizure demonstrated pH 7.27, HCO_3^- 9.06 mmol/L, base excess -16.5 and lactate 4.59 mmol/L, indicating substantial metabolic disturbance during the acute event. Generalized convulsive activity can be accompanied by transient lactate elevation and metabolic abnormalities, making reassessment after stabilization important. The patient's subsequent clinical monitoring therefore appropriately included glucose, electrolyte, renal and liver-function assessment.

A broad neurological differential diagnosis is essential in postpartum women presenting with seizures. Cerebral venous sinus thrombosis (CVST) is particularly relevant because pregnancy and the puerperium are associated with increased thrombotic risk, and CVST may present with headache, seizures, altered consciousness or focal neurological findings.¹²⁻¹⁴

The relationship between postpartum neurological symptoms and neuraxial anaesthesia also deserves consideration. CVST can mimic post-dural-puncture headache, and a change in headache characteristics or the development of neurological symptoms should prompt reconsideration of the diagnosis and appropriate neuroimaging. A review of reported postpartum CVST cases following neuraxial blockade found headache to be the predominant presenting symptom, with seizures occurring in a substantial proportion of cases.¹⁵

Other neurological conditions, including posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome, ischemic stroke and intracranial haemorrhage, can also occur during pregnancy and the postpartum period. Hypertension and the preeclampsia/eclampsia spectrum are important risk factors for pregnancy-associated cerebrovascular disease.¹⁶⁻¹⁸

Neuroimaging is therefore an important component of evaluation when the clinical presentation is atypical, severe, recurrent or associated with persistent headache, focal neurological abnormalities or altered consciousness. The literature emphasizes that several pregnancy-associated neurological disorders may overlap clinically and radiologically, making specialist neurological assessment particularly important.^{19,20}

Final Diagnosis Postpartum eclampsia with Posterior Reversible Encephalopathy Syndrome (PRES) following emergency LSCS under spinal anaesthesia Diagnostic consideration.

Overall, the major lesson from this case is the importance of rapid recognition, immediate anaesthetic stabilization, seizure control, haemodynamic support and multidisciplinary neurological evaluation. The case also demonstrates why a postpartum seizure should not automatically be assigned a single cause without adequate evidence. In this patient, the combination of postpartum timing, elevated blood pressure, generalized seizure, metabolic abnormalities and subsequent haemodynamic instability justified intensive monitoring and continued investigation. The available documentation does not establish a definitive alternative neurological diagnosis, and therefore the final diagnosis should remain appropriately descriptive unless

additional neuroimaging, EEG or specialist records confirm the underlying cause.

Conclusion

This case highlights the importance of early recognition and immediate multidisciplinary management of an acute postpartum generalized seizure following emergency caesarean section. In this patient, the seizure was associated with transient hypertension and subsequent haemodynamic instability, requiring urgent anaesthetic assessment, oxygenation, anticonvulsant therapy, magnesium sulfate and intensive-care monitoring. The available documentation does not establish a definitive cause such as eclampsia, and therefore a broad neurological differential should be maintained. Postpartum women presenting with seizures require systematic evaluation for hypertensive, metabolic, cerebrovascular and other neurological causes. The case emphasizes the central role of anaesthesia and critical care in airway management, seizure control, haemodynamic stabilization and prevention of secondary complications, together with timely neurological evaluation and appropriate neuroimaging when indicated.

Learning Points

1. Postpartum seizures are medical and obstetric emergencies requiring immediate intervention.
2. Initial management should prioritize airway, breathing, circulation and seizure control.
3. Anaesthetic involvement is particularly important when airway, respiratory or haemodynamic compromise accompanies the seizure.
4. Magnesium sulfate is an important therapy when an obstetric hypertensive seizure/eclampsia is clinically suspected.

5. Persistent or recurrent seizures require neurological assessment and consideration of alternative causes.
6. ABG, glucose, electrolyte, renal and liver-function assessment can help identify associated metabolic abnormalities.
7. Post-seizure hypotension requires close haemodynamic monitoring and timely fluid or vasopressor support when indicated.
8. Multidisciplinary coordination between Obstetrics, Anaesthesia, Medicine, Neurology and Critical Care is essential.

References

1. Miller EC, Vollbracht S. Neurology of preeclampsia and related disorders. *Curr Opin Neurol.* 2021;34(1):130-137.
2. Katz VL, Farmer RM, Kuller JA. Preeclampsia into eclampsia: toward a new paradigm. *Am J Obstet Gynecol.* 2000;182(6):1389-1396.
3. Bushnell C, Chireau M. Preeclampsia and stroke: risks during and after pregnancy. *Stroke Res Treat.* 2011;2011:858134.
4. Katz VL, Kuller JA, McMahon MJ, Warren MA, Wells SR. Pre-eclampsia into eclampsia: toward a new paradigm. *Am J Obstet Gynecol.* 2000;182(6):1389-96.
5. Stella CL, Jodicke CD, How HY, Hibbard JU. Postpartum headache: is your work-up complete? *Am J Obstet Gynecol.* 2003;188(3):830-5.
6. Katz VL, Dotters DJ, Droegemueller W. Peripartum seizures: a continuing diagnostic dilemma. *Am J Obstet Gynecol.* 1989;161(3):1021-4.
7. Edlow JA, Caplan LR, O'Brien K, Tibbles CD. Diagnosis of acute neurologic emergencies in pregnant and postpartum women. *Emerg Med Clin North Am.* 2016;34(4):985-1001. doi:10.1016/j.emc.2016.06.014.
8. Miller EC. Neurological complications of pregnancy. *J Neurol.* 2020;267(6):1857-1864.
9. World Health Organization. WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia. Geneva: World Health Organization; 2011.
10. Duley L, Gülmezoglu AM, Henderson-Smart DJ, Chou D. Magnesium sulphate and other anticonvulsants for women with pre-eclampsia. *Cochrane Database Syst Rev.* 2010;(11):CD000025.
11. American College of Obstetricians and Gynecologists. Gestational hypertension and preeclampsia: ACOG Practice Bulletin No. 222. *Obstet Gynecol.* 2020;135(6):e237-e260.
12. Hennawy M, Zhou LW, Field TS. Cerebral venous thrombosis should be considered in a postpartum patient with headache and seizure. *CMAJ.* 2023;195(48):E1663. doi:10.1503/cmaj.149620.
13. Silvis SM, de Bruijn SF, Kapelle LJ. Cerebral venous thrombosis and thrombophilia in pregnancy. *Semin Thromb Hemost.* 2011;37(7):813-8.
14. Saposnik G, Bushnell C, Coutinho JM, et al. Diagnosis and management of cerebral venous thrombosis: a scientific statement from the American Heart Association. *Stroke.* 2011;42(4):1158-92.
15. Bhatia K, Jones H, Miller S, et al. Postpartum cerebral venous sinus thrombosis following obstetric neuraxial blockade: a literature review with analysis of 58 case reports. *Int J Obstet Anesth.* 2021;48:103218. doi:10.1016/j.ijoa.2021.103218.
16. Tawati DA, Chan WS. A systematic review of posterior reversible encephalopathy syndrome in pregnant women with severe preeclampsia and

eclampsia. *Obstet Med.* 2023;16(4):236-241.
doi:10.1177/1753495X221150302.

17. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol.* 2015;14(9):914-25.
18. Williams TL, Ford L, Morris S, et al. Posterior reversible encephalopathy syndrome in pregnancy and postpartum period. *Obstet Med.* 2020;13(2):63-69.
19. Miller EC, Gatollari HJ, Too G, et al. Risk of pregnancy-associated stroke across age groups in women with hypertensive disorders of pregnancy. *Stroke.* 2017;48(2):260-5.
20. Zhang X, Fang K, Zhang Y, Song J, Wang R, Ji X, et al. Cerebral venous thrombosis during pregnancy and postpartum: a systematic review and meta-analysis. *Int J Stroke.* 2025;20(10):1188-1200.
doi:10.1177/17474930251355751.