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POSTPARTUM HEADACHE AND THE DIFFERENTIATION OF POSTDURAL PUNCTURE HEADACHE CEREBRAL VENOUS THROMBOSIS AND PREECLAMPSIA: A STRUCTURED NARRATIVE REVIEW

CEFALEIA NO PÓS-PARTO E A DIFERENCIAÇÃO ENTRE CEFALEIA PÓS-PUNÇÃO DURAL, TROMBOSE VENOSA CEREBRAL E PRÉ-ECLAMPSIA: UMA REVISÃO NARRATIVA ESTRUTURADA

CEFALEA POSPARTO Y DIFERENCIACIÓN ENTRE CEFALEA POSPUNCIÓN DURAL, TROMBOSIS VENOSA CEREBRAL Y PREECLAMPSIA: UNA REVISIÓN NARRATIVA ESTRUCTURADA

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ABSTRACT

Background: Headache is common after delivery, but apparently typical presentations can conceal hypertensive or cerebrovascular emergencies. Recent neuraxial anesthesia may create diagnostic anchoring and delay recognition of a second disorder. Objective: To examine the clinical differentiation of postdural puncture headache, cerebral venous thrombosis, and postpartum preeclampsia, emphasizing investigation, overlapping presentations, and time-sensitive management. Methods: A targeted PubMed-based narrative review evaluated clinical guidelines, prospective headache cohorts, retrospective obstetric studies, and contemporary cerebrovascular recommendations available through October 24, 2026. Evidence was interpreted according to population, diagnostic ascertainment, and study design; no new quantitative pooling was performed. Results: Orthostatic pain beginning after dural puncture supports postdural puncture headache, whereas new hypertension, visual symptoms, seizures, focal deficits, or a progressive or changing headache require evaluation for alternative or concurrent disease. Preeclampsia may develop after a normotensive pregnancy and does not require proteinuria when other diagnostic severe features are present. Cerebral venous thrombosis can initially present with headache alone and requires venous imaging; a normal unenhanced CT does not exclude it. Large administrative cohorts associate postdural puncture headache with rare but clinically important intracranial complications, although causality is not established. Treatment pathways differ substantially: epidural blood patch for selected disabling cerebrospinal fluid

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leakage, urgent blood pressure control and magnesium when indicated for hypertensive disease, and therapeutic anticoagulation for confirmed venous thrombosis. Conclusions: Differentiation depends on repeated clinical assessment rather than a single symptom. Persistent, atypical, or evolving postpartum headache warrants prompt multidisciplinary reassessment and appropriately selected neuroimaging.

Keywords: Postpartum Period. Headache. Postdural Puncture Headache. Cerebral Venous Thrombosis. Preeclampsia. Epidural Blood Patch.

RESUMO

Contexto: A cefaleia é comum após o parto, mas apresentações aparentemente típicas podem mascarar emergências hipertensivas ou cerebrovasculares. A anestesia neuroaxial recente pode criar uma âncora diagnóstica e atrasar o reconhecimento de uma segunda doença. Objetivo: Examinar a diferenciação clínica entre cefaleia pós-punção dural, trombose venosa cerebral e pré-eclâmpsia pós-parto, enfatizando a investigação, as apresentações sobrepostas e o manejo oportuno. Métodos: Uma revisão narrativa direcionada, baseada no PubMed, avaliou diretrizes clínicas, coortes prospectivas de cefaleia, estudos obstétricos retrospectivos e recomendações cerebrovasculares contemporâneas disponíveis até 24 de outubro de 2026. As evidências foram interpretadas de acordo com a população, a confirmação diagnóstica e o desenho do estudo; nenhuma nova análise quantitativa conjunta foi realizada. Resultados: Dor ortostática com início após a punção dural corrobora o diagnóstico de cefaleia pós-punção dural, enquanto hipertensão de início recente, sintomas visuais, convulsões, déficits focais ou cefaleia progressiva ou variável requerem avaliação para doenças alternativas ou concomitantes. A pré-eclâmpsia pode se desenvolver após uma gravidez normotensiva e não requer proteinúria quando outras características diagnósticas graves estão presentes. A trombose venosa cerebral pode se apresentar inicialmente apenas com cefaleia e requer exames de imagem venosa; uma tomografia computadorizada sem contraste normal não a exclui. Grandes coortes administrativas associam a cefaleia pós-punção dural com complicações intracranianas raras, porém clinicamente importantes, embora a causalidade não esteja estabelecida. As vias de tratamento diferem substancialmente: tampão sanguíneo epidural para vazamento de líquido cefalorraquidiano incapacitante selecionado, controle urgente da pressão arterial e magnésio quando indicado para doença hipertensiva e anticoagulação terapêutica para trombose venosa confirmada. Conclusões: A diferenciação depende da avaliação clínica repetida, e não de um único sintoma. Cefaleia pós-parto persistente, atípica ou em evolução justifica reavaliação multidisciplinar imediata e exames de neuroimagem adequadamente selecionados.

Palavras-chave: Período Pós-parto. Cefaleia. Cefaleia Pós-punção Dural. Trombose Venosa Cerebral. Pré-eclâmpsia. Tampão Sanguíneo Epidural.

RESUMEN

Antecedentes: El dolor de cabeza es frecuente tras el parto; sin embargo, presentaciones aparentemente típicas pueden ocultar emergencias hipertensivas o cerebrovasculares. La administración reciente de anestesia neuroaxial puede inducir un sesgo de anclaje diagnóstico y retrasar la identificación de una patología concomitante. Objetivo: Analizar la diferenciación clínica entre la cefalea pospunción dural, la trombosis venosa cerebral y la preeclampsia posparto, haciendo hincapié en el proceso diagnóstico, las presentaciones clínicas solapadas y el manejo que requiere una intervención oportuna. Métodos: Se realizó

una revisión narrativa basada en PubMed que evaluó guías clínicas, cohortes prospectivas de pacientes con cefalea, estudios obstétricos retrospectivos y recomendaciones actuales sobre patología cerebrovascular, disponibles hasta el 24 de octubre de 2026. La evidencia se interpretó considerando la población, los criterios diagnósticos y el diseño de cada estudio; no se llevó a cabo ningún nuevo análisis cuantitativo conjunto. Resultados: La presencia de dolor ortostático tras una punción dural sugiere una cefalea pospunción dural; no obstante, la aparición de hipertensión arterial, síntomas visuales, convulsiones, déficits focales, o una cefalea que evoluciona o cambia de características, obliga a descartar otras patologías subyacentes o concomitantes. La preeclampsia puede manifestarse tras un embarazo normotensivo y no requiere la presencia de proteinuria si existen otros criterios diagnósticos de gravedad. La trombosis venosa cerebral puede presentarse inicialmente solo con cefalea, por lo que requiere estudios de imagen venosa; una tomografía computarizada (TC) simple (sin contraste) normal no permite descartarla. Grandes cohortes basadas en registros administrativos asocian la cefalea pospunción dural con complicaciones intracraneales poco frecuentes pero clínicamente relevantes, aunque no se ha establecido una relación causal. Los enfoques terapéuticos difieren considerablemente: el parche hemático epidural se reserva para casos seleccionados de fuga de líquido cefalorraquídeo que resultan incapacitantes; el control urgente de la presión arterial y la administración de magnesio están indicados en trastornos hipertensivos; y la anticoagulación terapéutica es necesaria en casos de trombosis venosa confirmada. Conclusiones: El diagnóstico diferencial depende de una evaluación clínica seriada, más que de un síntoma aislado. Ante una cefalea posparto persistente, atípica o de carácter evolutivo, es imperativo realizar una reevaluación multidisciplinaria inmediata y solicitar los estudios de neuroimagen más adecuados.

Palabras clave: Periodo Posparto. Cefalea. Cefalea Pospunción Dural. Trombosis Venosa Cerebral. Preeclampsia. Parche Hemático Epidural.

1 INTRODUCTION

Postpartum headache spans common primary headache disorders and uncommon conditions capable of causing stroke, seizures, permanent disability, or maternal death. Delivery changes the diagnostic context through rapid hormonal and circulatory shifts, sleep disruption, hypercoagulability, hypertensive disease, and exposure to neuraxial procedures. A history of migraine or an apparently explanatory dural puncture reduces neither the need to measure blood pressure nor the importance of a focused neurological examination.[1,2]

Population and referral studies produce very different estimates of secondary disease. In a prospective cohort of 985 women, 381 reported headache or neck/shoulder pain during the first postpartum week, and primary headaches accounted for more than three-quarters of postpartum headaches. By contrast, among 95 women hospitalized for headache more than 24 hours after delivery, preeclampsia/eclampsia accounted for 24% and spinal headache for 16%. These findings describe different levels of clinical selection and cannot be combined into a single probability for all postpartum patients.[3,4]

This review focuses on three frequently competing explanations: postdural puncture headache (PDPH), cerebral venous thrombosis (CVT), and postpartum preeclampsia. The objective is to identify discriminating features, explain their limitations, and connect diagnostic assessment with treatment. These conditions can coexist; assigning one diagnosis should not prevent reassessment when the course changes.

2 METHODS

Targeted PubMed searches combined postpartum or puerperium with headache, dural puncture, cerebral venous thrombosis, preeclampsia, diagnosis, and guideline. Additional searches retrieved foundational prospective cohorts and major obstetric and stroke recommendations. Publications available through October 24, 2026 were considered. Bibliographic details of every cited reference were verified in PubMed. Guidelines were prioritized for clinical pathways and primary cohorts for frequency and association estimates.

This is a structured narrative review rather than a systematic review. Searches were selective, no independent duplicate screening or formal risk-of-bias scoring was conducted, and no new meta-analysis was undertaken. Administrative diagnosis codes, referral enrichment, different postpartum windows, and inconsistent headache definitions were considered when interpreting results. Management recommendations are presented as clinical synthesis supported by published guidance, not as a validated diagnostic score.

2.1 INITIAL ASSESSMENT AND DIAGNOSTIC PRIORITIES

The first assessment should establish vital signs, level of consciousness, headache onset and evolution, and whether an immediate neurological or hypertensive emergency is present. Blood pressure should be measured with an appropriate cuff and repeated when abnormal. A focused examination should assess pupils, eye movements, visual function, speech, limb power, sensation, coordination, reflexes, and gait when safe. Fundus examination can reveal optic disc edema, although its absence does not exclude acute CVT or other intracranial disease.[1,5]

The history should document the exact delivery date, antenatal and postpartum blood pressures, neuraxial procedure details, recognized accidental dural puncture, anticoagulant exposure, fever, fluid loss, thrombosis history, and previous headache phenotype. The onset pattern should be characterized as thunderclap, rapidly progressive, gradually progressive, or recurrent. Positional dependence, response to lying flat, visual disturbance, vomiting, neck pain, seizures, and focal symptoms are particularly relevant. Response to an analgesic or caffeine is insufficient to establish a benign diagnosis.

Immediate escalation is warranted for thunderclap headache, seizure, altered consciousness, focal deficit, severe or persistent hypertension, meningeal features, or rapidly worsening pain. A new persistent headache that differs from prior migraine also deserves caution, even if the neurological examination is initially normal. Stabilization and urgent treatment should proceed in parallel with investigation when eclampsia or hypertensive encephalopathy is suspected.[1,6]

2.2 POSTDURAL PUNCTURE HEADACHE

2.2.1 Mechanism and clinical phenotype

PDPH results from cerebrospinal fluid leakage after intentional or unintentional dural puncture. Reduced intracranial cerebrospinal fluid volume can produce traction on pain-sensitive structures and compensatory venous changes. Typical pain begins within five days of puncture, worsens upright, and improves supine; neck stiffness, auditory symptoms, nausea, and diplopia may accompany it. A puncture may have gone unrecognized during epidural placement, so absence of a documented accident does not exclude PDPH.[7,8]

The postural component is strongly informative but not obligatory. Conversely, CVT and other causes can initially resemble a low-pressure headache. The most useful question is whether the complete trajectory remains consistent with uncomplicated cerebrospinal fluid

leakage. Loss of positional dependence, development of persistent nonorthostatic pain, a new focal deficit, seizure, or failure to improve should trigger reconsideration rather than repeated treatment under the same assumption.[7,9]

2.2.2 Investigation and initial treatment

Routine imaging is not required before treatment of a clinically typical PDPH after a known puncture when examination and evolution are reassuring. Imaging becomes important when symptoms are atypical, changing, severe despite treatment, or accompanied by neurological findings. The purpose is to identify alternative disease or complications such as subdural collection, hemorrhage, CVT, or posterior reversible encephalopathy syndrome (PRES).[7]

Initial treatment can include appropriate hydration, simple analgesia, and a limited trial of caffeine when suitable, accounting for breastfeeding and concurrent intake. Excessive intravenous hydration does not seal a dural defect, and prolonged bed rest has burdens that are particularly relevant in the puerperium. Symptom control should be accompanied by planned reassessment and an anesthesia review rather than discharge with an open-ended conservative regimen.[7,8]

An epidural blood patch should be considered when headache substantially limits mobility, daily functioning, or infant care, or when conservative treatment is inadequate. Timing should reflect severity and the likelihood of needing a repeat procedure; severely disabling symptoms should not be left untreated solely to satisfy an arbitrary waiting period. Infection, coagulation status, platelet abnormalities, and anticoagulant timing must be reviewed before intervention. Incomplete relief is not automatically evidence that the first diagnosis was wrong, but an atypical course or no meaningful response warrants renewed assessment before repeated patching.[7]

2.2.3 Why a dural puncture does not end the diagnostic process

In an administrative cohort of 1,003,803 women receiving neuraxial anesthesia, 4,808 had coded PDPH. The combined incidence of CVT or subdural hematoma was 3.12 per 1,000 in women with PDPH versus 0.16 per 1,000 without it; the adjusted odds ratio for that composite was 19.0. Most identified intracranial events occurred during readmission. The composite should not be reported as a CVT-only rate, and coding-based associations do not prove that PDPH caused the complications.[10]

A separate national cohort of more than 22 million deliveries found subdural hematoma to be rare overall, while its absolute frequency was higher among women with presumed PDPH. Together, these studies support explicit follow-up and low thresholds for reassessment when a headache changes. They do not justify routine imaging of every uncomplicated postural headache or establish that an early blood patch prevents all neurological complications.[11]

2.3 CEREBRAL VENOUS THROMBOSIS

2.3.1 Risk and presenting features

The puerperium is a clinically important prothrombotic period. In a case-control study, postpartum status was associated with CVT, with the strongest association during the first six weeks after delivery: adjusted odds ratio 18.7 (95% confidence interval, 8.3–41.9). This relative association does not imply a high absolute probability in every postpartum patient, but it changes the threshold for investigating a compatible syndrome.[12]

CVT may present with isolated headache, intracranial hypertension, papilledema, focal deficits, encephalopathy, or seizures. Pain may be diffuse, progressive, migraine-like, or occasionally thunderclap. No single headache quality excludes it. A normal examination early in the illness is therefore insufficient reassurance when the headache is persistent, evolving, or accompanied by meaningful thrombotic risk.[5,13]

Potential contributors include thrombophilia, prior venous thrombosis, infection, dehydration, and other postpartum complications. Recent dural puncture can be an additional source of diagnostic confusion; intracranial hypotension and venous abnormalities may coexist. The practical implication is to investigate suspicious evolution rather than require the patient to fit a stereotyped thrombotic presentation.[10,14]

2.3.2 Choosing the correct imaging examination

Diagnosis requires demonstration of venous thrombosis with MRI/MR venography or CT/CT venography, chosen according to availability, stability, and the clinical question. An unenhanced CT can identify hemorrhage or a mass effect but may be normal in CVT. Ordering a brain scan without the appropriate venous study can therefore produce false reassurance. Equivocal venous findings should be reviewed by an experienced neuroradiologist because anatomical variants and flow artifacts can resemble thrombosis.[5,13]

D-dimer should not be used as a stand-alone exclusion test in a postpartum patient with concerning symptoms. Physiological postpartum elevation limits specificity, and a negative result cannot reliably resolve a high-suspicion presentation, particularly with isolated or prolonged headache. Lumbar puncture is not the primary diagnostic test for CVT and should not precede adequate assessment of intracranial pathology when red flags are present.[13]

2.3.3 Treatment implications

Confirmed acute CVT generally requires therapeutic anticoagulation, commonly with a heparin-based regimen initially. Venous hemorrhagic infarction does not automatically prohibit anticoagulation, because ongoing venous thrombosis is itself the cause of congestion and hemorrhage. The decision requires specialist assessment of hemorrhage type, clinical stability, obstetric bleeding, recent procedures, and other contraindications. Seizures, intracranial hypertension, visual risk, and threatening mass effect require simultaneous management.[5,13]

Postpartum anticoagulant selection must account for breastfeeding and recent neuraxial procedures. Low-molecular-weight heparin and warfarin are established options in lactation, whereas direct oral anticoagulants should not be assumed interchangeable in breastfeeding patients. Treatment duration depends on whether the event was transiently provoked, recurrent, or associated with persistent thrombophilia. Endovascular therapy and decompressive surgery are reserved for selected severe cases, particularly deterioration despite appropriate medical treatment or impending herniation.[13,14]

2.4 POSTPARTUM PREECLAMPSIA AND ECLAMPSIA

2.4.1 Recognition after an apparently uncomplicated pregnancy

Hypertensive disease can persist after delivery or develop de novo. Delayed-onset postpartum preeclampsia is commonly considered between 48 hours and six weeks after delivery, while immediate postpartum disease can occur earlier. Many delayed presentations occur in the first 7–10 days. A previously normotensive pregnancy, discharge without antihypertensive medication, or absence of a prior preeclampsia diagnosis does not exclude it.[15]

In a cohort of 152 women readmitted with delayed postpartum preeclampsia, 96 had no antecedent hypertensive diagnosis, headache was the presenting symptom in 105, and

22 developed eclampsia. These proportions reflect a selected affected population, not the incidence among all postpartum women. They nevertheless show why new headache requires a blood pressure check even when an alternative explanation appears plausible.[16]

2.4.2 Diagnostic findings and neurological complications

New blood pressure of at least 140/90 mmHg requires confirmation and evaluation in context. Severe hypertension, at least 160 mmHg systolic or 110 mmHg diastolic, should be confirmed promptly and treated urgently rather than waiting several hours for repeat measurements. Preeclampsia can be diagnosed without proteinuria when qualifying organ dysfunction is present. Platelet reduction, renal or liver abnormalities, pulmonary edema, and persistent neurological symptoms guide assessment.[6,15]

Headache in this setting is often persistent and poorly responsive to usual medication, sometimes accompanied by visual symptoms. However, headache alone is nonspecific and should not substitute for a full clinical assessment. Conversely, normal urine protein does not negate severe hypertension or other severe features. Initial tests usually include platelet count, creatinine, liver enzymes, and urine protein assessment, with additional testing directed by symptoms and examination.[6]

PRES may produce headache, visual disturbance, seizures, and encephalopathy, with vasogenic edema on imaging. Eclampsia is a clinical emergency, and treatment should not await MRI confirmation. Persistent neurological symptoms despite blood pressure control, an atypical seizure pattern, focal findings, or concern for hemorrhage or thrombosis warrant neuroimaging even when preeclampsia is already diagnosed. Hypertension and CVT are not mutually exclusive explanations.[1,15]

2.4.3 Immediate management

Sustained severe hypertension should be treated promptly using established obstetric protocols, commonly with intravenous labetalol, intravenous hydralazine, or immediate-release oral nifedipine when appropriate. Magnesium sulfate is the standard treatment for eclamptic seizures and is considered for seizure prophylaxis in postpartum preeclampsia with neurological symptoms or other high-risk features. Renal function, urine output, respiration, and reflexes influence monitoring and dosing. Magnesium is not a substitute for antihypertensive therapy.[6,15]

Volume assessment is important because postpartum preeclampsia can include fluid overload and pulmonary edema. Diuresis is appropriate when clinically indicated rather than as an automatic response to every headache. Management should involve obstetrics, anesthesia, and neurology according to the presentation, with critical care input when organ dysfunction or neurological instability is present. After stabilization, follow-up must address blood pressure, medication adjustment, and the longer-term cardiovascular implications of hypertensive pregnancy disorders.[2,15]

Table 1

Clinical differentiation of the three principal diagnoses

Condition	Supporting presentation	Assessment that changes management	Initial management implications
Postdural puncture headache	Recent dural puncture; typically orthostatic pain beginning within five days; possible neck or auditory symptoms.	Review neuraxial history, blood pressure, examination, and trajectory. Image atypical or changing presentations.	Analgesia and limited conservative treatment; anesthesia assessment and epidural blood patch when disabling or persistent. [7]
Cerebral venous thrombosis	Persistent or progressive headache; may initially be isolated. Papilledema, seizures, focal deficits, or encephalopathy increase concern.	MRI with MR venography or CT with CT venography. Normal unenhanced CT does not exclude CVT.	Therapeutic anticoagulation when appropriate; treat seizures, intracranial hypertension, and threatening mass effect. [5,13]
Postpartum preeclampsia	New or persistent hypertension, often with refractory headache or visual symptoms; can follow a normotensive pregnancy.	Repeat blood pressure; platelets, creatinine, liver enzymes, and urine protein. Assess neurological symptoms and need for imaging.	Urgent treatment of sustained severe hypertension; magnesium for eclampsia and selected high-risk presentations. Proteinuria is not obligatory when other qualifying features are present. [6,15]

CT, computed tomography; CVT, cerebral venous thrombosis; MRI, magnetic resonance imaging. Features overlap and conditions can coexist. This table is a clinical synthesis, not a validated exclusion rule.

3 OTHER DIAGNOSES AND COMMON DIAGNOSTIC ERRORS

The focused differential should not exclude subarachnoid hemorrhage, intracerebral hemorrhage, reversible cerebral vasoconstriction syndrome, arterial dissection, meningitis, pituitary apoplexy, or primary migraine. Thunderclap headache requires urgent assessment even when it follows neuraxial anesthesia. Fever or meningeal signs suggest an infectious process, and prominent visual or endocrine abnormalities may direct investigation toward the pituitary. The original headache label should be reconsidered whenever a new finding emerges.[1,17]

Common errors include diagnosing uncomplicated PDPH despite worsening positional pain, excluding preeclampsia because antenatal blood pressure or urine protein was normal, and excluding CVT after unenhanced CT. Assessment should instead integrate the trajectory, repeated blood pressure, examination, laboratory findings, and imaging selected for the suspected mechanism.

4 FOLLOW UP AND EVIDENCE LIMITATIONS

Before discharge, patients should receive a clear reassessment plan and instructions to seek urgent care for worsening or nonpostural headache, visual change, seizure, weakness, confusion, fever, or severe blood pressure elevation. Anesthesia-related headache should have documented follow-up until resolution or an alternative diagnosis is established. Education should include the possibility of delayed hypertensive disease after a previously reassuring pregnancy.[7,16]

Study setting determines observed frequency. In the Stella cohort, 15/22 selected patients undergoing imaging had abnormalities; that yield does not apply to routine scanning of all postpartum headaches. A prospective study of 900 women recorded headache in 241 during the first month, with primary disorders predominating.[4,18]

The evidence base remains limited by observational designs, diagnostic coding, changing anesthetic techniques, and small numbers of rare outcomes. There is no single validated bedside rule that safely distinguishes the three target conditions. Future studies should measure changes in phenotype, timing of reassessment, imaging appropriateness, and delays to treatment, rather than rely only on a final diagnostic code. Table 2 summarizes representative studies and the limits of their application.

Table 2

Selected studies informing frequency and diagnostic vigilance

Study	Population	Principal finding	Interpretive limitation
Goldszmidt et al. [3]	Prospective cohort of 985 women in the first postpartum week.	381 reported headache or neck/shoulder pain; primary disorders accounted for >75% of postpartum headaches.	Combined symptom frequency should not be relabeled as headache-only incidence; findings differ from hospitalized cohorts.

Study	Population	Principal finding	Interpretive limitation
Stella et al. [4]	95 women hospitalized for headache >24 hours after delivery.	Preeclampsia/eclampsia 24%; spinal headache 16%. Imaging abnormal in 15/22 selected patients.	Referral and imaging selection enrich secondary pathology. The 68% imaging yield does not apply to all postpartum headaches.
Guglielminotti et al. [10]	1,003,803 women receiving neuraxial anesthesia; administrative cohort.	CVT/subdural hematoma composite: 3.12/1,000 with PDPH versus 0.16/1,000 without; adjusted OR 19.0.	Composite outcome, diagnosis codes, and observational design. Association is not proof of causation.
Silvis et al. [12]	Case-control comparison of CVT cases and controls.	First six weeks postpartum: adjusted OR 18.7 (95% CI 8.3–41.9) for CVT.	Relative odds do not provide the absolute CVT probability for a patient presenting with headache.

CI, confidence interval; CVT, cerebral venous thrombosis; OR, odds ratio; PDPH, postdural puncture headache. Different populations, endpoints, and time windows prevent informal pooling.

5 CONCLUSION

Postpartum headache requires a mechanism-based differential that remains open to concurrent disease. Orthostatic symptoms after puncture support PDPH, but changing pain or neurological findings demand renewed investigation. New hypertension should prompt evaluation for postpartum preeclampsia regardless of antenatal history or proteinuria, and suspected CVT requires dedicated venous imaging. Prompt recognition directs substantially different treatments and reduces the risk that a familiar symptom conceals an evolving obstetric or neurological emergency.

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