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Research Article

Postpartum Diagnostic Trap: RCVS–PRES Spectrum Disorder Masquerading As Post-Dural Puncture Headache Following Elective LSCS.

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Abstract

Headache following caesarean delivery under spinal anaesthesia is a common postoperative complaint and is most frequently attributed to post-dural puncture headache (PDPH). However, clinicians must remain vigilant for secondary neurological causes — particularly when symptoms evolve atypically or fail to respond to conservative measures. These include postpartum eclampsia, Reversible Cerebral Vasoconstriction Syndrome (RCVS), and Posterior Reversible Encephalopathy Syndrome (PRES). We present a case of a 30-year-old multiparous woman who developed persistent headache following an elective lower segment caesarean section (LSCS) under spinal anaesthesia. Initially managed as PDPH, her clinical course was complicated by progressive bradycardia, acute-onset hypertension, visual disturbances, and ultimately a generalized tonic-clonic seizure. Brain MRI confirmed RCVS–PRES spectrum disorder with characteristic patchy T2/FLAIR hyperintensities in bilateral parieto-occipital regions, posterior cingulate gyrus, and bilateral lentiform nuclei. The final diagnosis was postpartum eclampsia concurrent with RCVS–PRES spectrum disorder. Multidisciplinary management with magnesium sulphate, intravenous labetalol, levetiracetam, and nimodipine resulted in complete neurological recovery and safe discharge. This case underscores the critical importance of maintaining a high index of suspicion for overlapping neurological entities in any postpartum patient with refractory headache.

Keywords: RCVS, PRES, postpartum eclampsia, post-dural puncture headache, spinal anaesthesia, LSCS, postpartum headache, cerebral vasoconstriction.

HIGHLIGHTS

- Persistent postpartum headache unresponsive to conservative therapy must not be reflexively attributed to post-dural puncture headache (PDPH) following spinal anaesthesia.
- Postpartum eclampsia can occur without antecedent hypertension or proteinuria, and may present with a delayed sequence of headache, visual disturbance, and seizure.
- RCVS and PRES represent overlapping neurological entities sharing a common pathophysiology of endothelial dysfunction and cerebral autoregulatory failure, particularly in the postpartum period.
- MRI with T2/FLAIR sequences is essential when secondary neurological causes are suspected — characteristic posterior predominant vasogenic oedema confirms PRES, while clinical context supports concurrent RCVS.
- Early initiation of magnesium sulphate, antihypertensives, and nimodipine (calcium channel blocker) can reverse cerebral vasospasm and prevent maternal morbidity.
- Spinal anaesthesia may be an underrecognized trigger for RCVS–PRES spectrum disorder through CSF pressure shifts and sympathetic dysregulation in susceptible postpartum women

INTRODUCTION

Postpartum headache is a frequent clinical challenge encountered in obstetric and anaesthetic practice. In the setting of neuraxial anaesthesia, PDPH remains the leading diagnosis and is attributed to inadvertent or intended dural

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puncture, leading to cerebrospinal fluid (CSF) leakage and intracranial hypotension [1]. However, secondary causes may account for up to 40% of severe postpartum headaches, and these include potentially life-threatening conditions such as eclampsia, PRES, cerebral venous sinus thrombosis (CVST), subarachnoid haemorrhage, and RCVS [2,3].

Reversible Cerebral Vasoconstriction Syndrome (RCVS) is an increasingly recognised condition characterised by multifocal, segmental narrowing of cerebral arteries that typically resolves over days to weeks. Its hallmark clinical feature is recurrent thunderclap headache, but it may also manifest with focal neurological deficits, seizures, stroke, or visual disturbances. Posterior Reversible Encephalopathy Syndrome (PRES) represents a neuroradiological and clinical syndrome characterised by vasogenic oedema predominantly in posterior cerebral regions, associated with acute hypertension, seizures, encephalopathy, and visual symptoms [4]. Crucially, RCVS and PRES are not mutually exclusive — emerging evidence highlights a spectrum of overlapping pathophysiology involving shared endothelial dysfunction and cerebral autoregulatory failure, especially in the postpartum period [5].

The postpartum state itself is a recognised trigger for both RCVS and PRES, likely mediated by hormonal flux, haemodynamic shifts, sympathetic surges, and circulating vasoactive substances. The added variable of spinal anaesthesia — which alters CSF dynamics, sympathetic tone, and cerebrovascular reactivity — may further predispose susceptible individuals to these phenomena [6]. We report a rare case in which postpartum eclampsia with concurrent RCVS–PRES spectrum disorder initially mimicked PDPH, resulting in a delayed but ultimately successful diagnosis and recovery.

CASE PRESENTATION

Patient Profile and Initial Presentation

A 30-year-old woman (P2L2) with no known antepartum comorbidities was admitted for elective LSCS. She had no prior history of hypertension, pre-eclampsia, renal disease, or neurological illness. The procedure was performed under standard spinal anaesthesia with an uneventful intraoperative course. There were no documented complications at the time of dural puncture and no abnormalities were noted in the immediate post-anaesthetic recovery period.

On postoperative day 1, the patient began complaining of persistent headache. At this stage, vital signs remained stable and there were no focal neurological deficits. Given the recent spinal anaesthesia, a provisional diagnosis of PDPH was made, and she was managed conservatively with intravenous hydration, oral analgesics, and close observation. The headache failed to demonstrate the characteristic postural variation expected in PDPH — notably, it was not relieved by recumbency — and showed no symptomatic improvement over the subsequent 48 hours.

Clinical Deterioration

On postoperative day 3, a new and unexplained finding emerged: the patient developed significant bradycardia with a pulse rate of 40–44 beats per minute. Blood pressure at this point remained within the normal range, and oxygen saturation was maintained between 92–95% on room air. A 12-lead ECG confirmed sinus bradycardia with no evidence of heart block or ischaemic changes. Two-dimensional echocardiography (2D ECHO) and contrast-enhanced computed tomography (CECT) of the chest and abdomen revealed no structural or pulmonary pathology. The aetiology of the bradycardia remained unexplained at this stage and, in retrospect, may have represented an early autonomic manifestation of evolving cerebral dysregulation.

By postoperative day 5, the clinical picture had escalated substantially. The patient developed acute blurring of vision alongside a sharp rise in blood pressure to 160/100 mmHg. Notably, urine spot protein was negative, and all routine blood investigations — including full blood count, renal and liver function tests, and coagulation profile — remained within normal limits. Given the constellation of persistent headache, visual disturbance, and new-onset hypertension in the postpartum setting, a clinical diagnosis of postpartum eclampsia was entertained.

Magnesium sulphate infusion was commenced according to the Zuspan regimen, and intravenous labetalol 20 mg was administered as a stat dose for acute blood pressure control. Despite these measures, at 11:00 hours the patient experienced a single episode of generalized tonic-clonic seizure, following which she was emergently transferred to the Intensive Care Unit (ICU) for close monitoring and further management.

Neuroimaging and Final Diagnosis

Urgent MRI of the brain was performed and demonstrated patchy areas of T2/FLAIR hyperintensity involving the grey matter of bilateral parieto-occipital regions, the posterior aspect of the cingulate gyrus, bilateral lentiform nuclei, and the body of the right caudate nucleus. These findings were consistent with vasogenic oedema in a distribution characteristic of PRES (Fig 1). The co-occurrence of clinical features of cerebral vasoconstriction — in a postpartum patient without prior hypertension, presenting with headache, seizure, and visual disturbance — established the final diagnosis of Postpartum Eclampsia with RCVS–PRES Spectrum Disorder.

Figure 1.

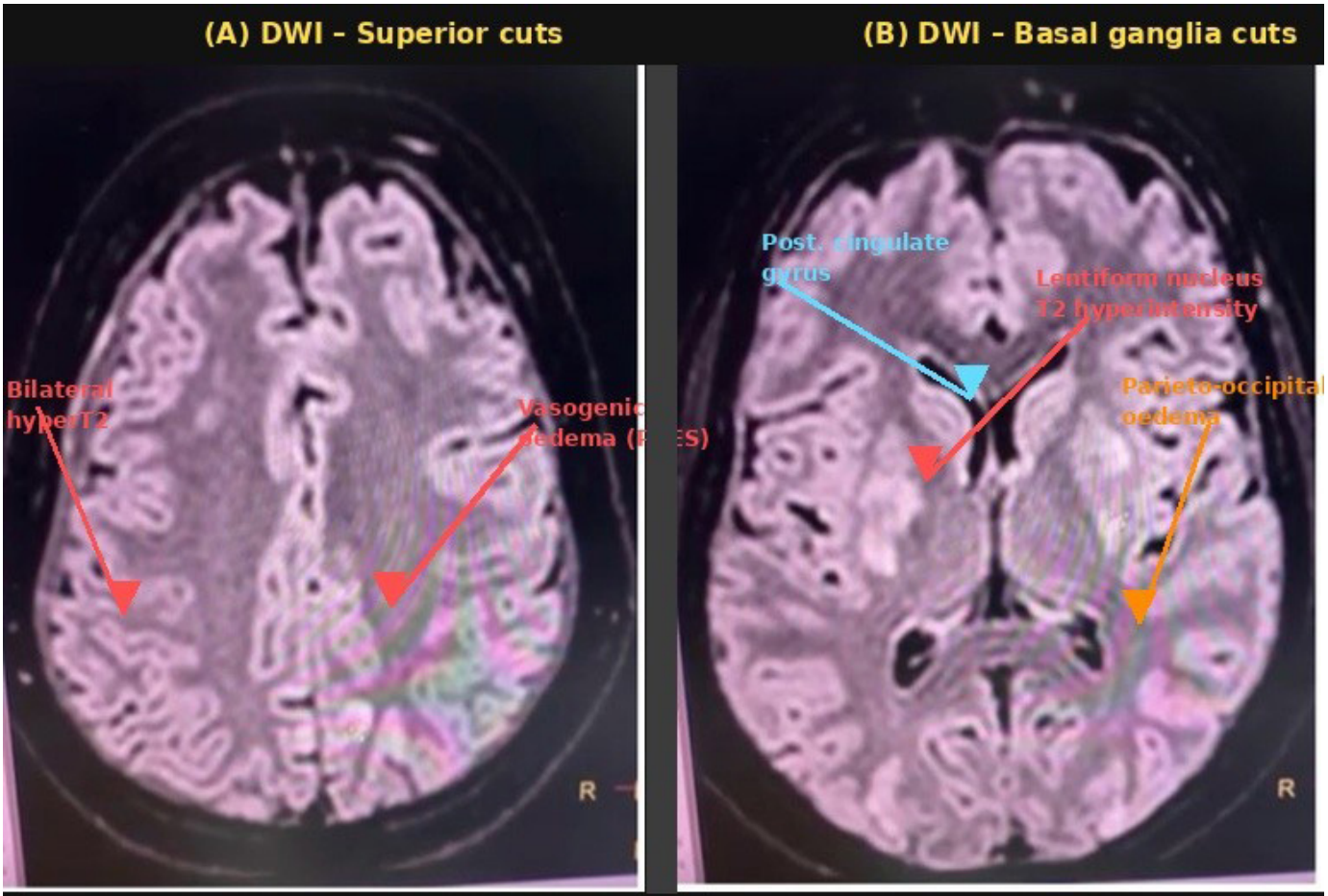


Fig. 1 MRI Brain (DWI): Patchy T2/FLAIR hyperintensities consistent with vasogenic oedema in RCVS–PRES spectrum

Treatment and Outcome

In the ICU, the patient was continued on magnesium sulphate infusion at 1 g/hour for seizure prophylaxis, supplemental oxygen, and intravenous antibiotics to prevent nosocomial infection. Intravenous levetiracetam was initiated for seizure control and subsequently transitioned to oral therapy. Nimodipine, a selective calcium channel blocker with established efficacy in cerebral vasospasm, was added to address the vasoconstriction component of RCVS. The patient showed progressive clinical improvement: blood pressure normalised, visual symptoms fully resolved, and no further seizure activity was recorded. She was successfully discharged in a stable neurological condition.

DISCUSSION

Diagnostic Transitions and Clinical Learning Points

This case illustrates a challenging sequence of diagnostic transitions that are instructive for both obstetricians and anaesthesiologists. The initial presentation was entirely consistent with PDPH — a well-recognised complication following spinal anaesthesia. However, several atypical features should have prompted earlier re-evaluation. The headache lacked postural character, was unresponsive to conservative therapy, and was later accompanied by neurological symptoms absent in PDPH. The principle that headache following neuraxial anaesthesia is PDPH until proven otherwise is clinically convenient but potentially dangerous, particularly when warning signs evolve.

Postpartum eclampsia, which accounts for 20–30% of all eclamptic seizures, is well recognised to occur without antecedent hypertension or proteinuria during the antenatal period, as observed in this patient. The classical prodrome of persistent headache, followed by visual disturbance, acute hypertension, and finally seizure — though temporally extended over five days in this case — mirrors the established pathophysiological cascade. The absence of proteinuria does not exclude the diagnosis, as endothelial dysfunction and cerebral autoregulatory failure can manifest independently of renal proteinuria [6,7].

The concurrent identification of RCVS–PRES spectrum disorder reflects the growing recognition that these are not isolated entities but rather interrelated expressions of a common pathophysiological substrate. RCVS is characterised by reversible segmental cerebral arterial vasoconstriction with clinical improvement over weeks [8]. PRES reflects the

downstream consequence of failed cerebral autoregulation — when arterial pressure overwhelms the vasculature's protective vasoconstriction, breakthrough hyperperfusion and vasogenic oedema ensue. In the postpartum setting, both are driven by hormonal changes, heightened sympathetic activity, and circulating vasoactive mediators including endothelin-1 and serotonin [9,10].

Role of Spinal Anaesthesia

The potential contributory role of spinal anaesthesia in precipitating or unmasking RCVS–PRES in susceptible individuals deserves consideration. Subarachnoid injection alters CSF pressure dynamics acutely, and compensatory cerebrovascular responses to these shifts may trigger vasoreactivity in predisposed vessels [11]. Additionally, the acute sympatholysis following spinal block, and subsequent rebound sympathetic surge during recovery, may contribute to the haemodynamic instability that predisposes cerebral autoregulatory failure. The unexplained bradycardia observed on day 3 may have represented an aberrant autonomic response within this context [12].

Differential Diagnosis and Exclusion

The differential diagnosis for this presentation was broad and included PDPH, cerebral venous sinus thrombosis (CVST), intracranial haemorrhage, meningitis, and isolated PRES or RCVS. CT brain and CT angiography excluded haemorrhagic stroke and significant cerebral venous pathology [13,14]. The characteristic MRI pattern of posterior predominant vasogenic oedema without diffusion restriction, in the context of postpartum onset, acute hypertension, seizure, and clinical recovery with appropriate therapy, cemented the RCVS–PRES diagnosis (**Table1**) [15].

Table 1. Summary of the key differentiating features between postpartum RCVS and PRES across clinical presentation, neuroimaging, and management — highlighting their overlapping spectrum.

Feature	Postpartum RCVS	Postpartum PRES
CLINICAL FEATURES		
Headache	Thunderclap; recurrent, severe Hallmark feature	Diffuse, progressive; less abrupt Often gradual onset
Seizures	Less common (~20–30%) Seen in severe cases	Prominent (~60–75%) Often the presenting feature
Visual symptoms	Transient blurring, scotomata Intermittent	Cortical blindness, hemianopia Prominent feature
Hypertension	Variable; may be absent Not required for diagnosis	Typically severe (>160/110 mmHg) Usually present
Encephalopathy	Uncommon unless stroke complicates Rare	Confusion, drowsiness, agitation Common
Proteinuria	Absent Not a feature	Variable; present if eclampsia overlap May be absent
Postpartum onset	Days to 4 weeks Peak: week 1–2	Usually within 48–72 hours Peak: first 24–72 hrs
Course	Monophasic; resolves over weeks Generally benign	Resolves days–weeks with treatment Reversible
IMAGING FINDINGS		
MRI T2/FLAIR	Often normal early May show cortical SAH or infarct later	Patchy hyperintensity in parieto-occipital regions Hallmark finding
Distribution	Anterior > posterior Territorial if infarct present	Posterior predominant Parieto-occipital, cerebellar, lentiform nuclei
DWI / ADC	Restricted diffusion if ischaemic stroke DWI bright / ADC dark	Facilitated diffusion (vasogenic oedema) DWI dark / ADC bright
MRA / CTA	Multifocal segmental vasoconstriction "String-and-beads" pattern	Usually normal vessels Vascular imaging unaffected
DSA (angiography)	Definitive; confirms reversible vasoconstriction Gold standard	Typically normal; not required Not a routine investigation
Haemorrhage	Cortical SAH, lobar ICH possible Significant complication risk	Rare haemorrhagic transformation Uncommon
MRI enhancement	No parenchymal enhancement Typically absent	Leptomeningeal enhancement in some cases Occasional finding
Imaging reversibility	Vasoconstriction resolves in 4–12 weeks Confirm on follow-up MRA	Oedema resolves in days–weeks Confirm on follow-up MRI
PATHOPHYSIOLOGY & MANAGEMENT		
Core mechanism	Segmental cerebral arterial vasospasm Vasoconstriction predominates	Failed cerebrovascular autoregulation Vasogenic oedema from hyperperfusion

CSF findings	Normal or mildly elevated protein Near normal	Normal Not diagnostic
Key treatment	Nimodipine (calcium channel blocker) Avoid vasoconstrictors and triptans	BP control: labetalol, hydralazine Magnesium sulphate if eclampsia co-exists
Prognosis	Good; stroke risk if untreated Vigilance for complications	Excellent with treatment Rare recurrence

CONCLUSION

This case powerfully demonstrates that persistent postpartum headache following spinal anaesthesia must not be reflexively attributed to PDPH. A thorough, dynamic clinical re-evaluation is essential whenever headache proves refractory to conservative management, lacks typical postural characteristics, or is accompanied by any neurological symptom — however subtle. The triad of headache, visual disturbance, and hypertension in the postpartum period must raise urgent suspicion for eclampsia and RCVS–PRES spectrum disorder.

Timely neuroimaging — particularly MRI with FLAIR sequences — is essential for diagnosis when secondary causes are suspected. Early administration of magnesium sulphate for seizure prophylaxis and prevention of cerebral vasospasm, combined with targeted antihypertensive therapy and calcium channel blockers such as nimodipine to address vasoconstriction, represents the cornerstone of management. Multidisciplinary collaboration between obstetrics, anaesthesiology, neurology, and critical care is vital in navigating these complex presentations.

As RCVS–PRES spectrum disorder becomes increasingly recognised in the postpartum population, awareness among frontline clinicians must keep pace. The maternal outcome in this case was excellent precisely because the diagnosis was ultimately made and treatment escalated appropriately. Vigilance, clinical flexibility, and a willingness to look beyond the most common diagnosis remain the most powerful tools in postpartum neurological care.

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Nil

Conflict of Interest

The authors have no conflicts of interest to disclose.

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