RESEARCH PAPER

Cerebral Venous Thrombosis in Term Pregnancy Complicated by Peripartum Cardiomyopathy: A Case Report

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Received: 20th Apr, 2026 | Revised: 25th Apr, 2026 | Accepted: 9th May, 2026 | Available Online: 14th May, 2026

ABSTRACT

Introduction: Cerebral venous thrombosis (CVT) is a rare but potentially life-threatening cerebrovascular complication of pregnancy. Its presentation often mimics common obstetric emergencies, making timely diagnosis challenging. We report a case of acute CVT involving the right transverse and right sigmoid sinuses in a woman at term gestation, further complicated by peripartum cardiomyopathy (PPCM) in the postpartum period — an exceptionally rare co-occurrence with shared pathophysiological underpinnings.

Case Presentation: A 38-year-old woman (G2A1) at 37 weeks and 2 days of gestation, conceived via ovarian induction, presented with sudden-onset severe bifrontal and occipital headache unresponsive to analgesics and two episodes of projectile vomiting. Vitals were stable. Routine haematological, biochemical, and coagulation investigations were unremarkable. Urine albumin was 1+ on dipstick.

Diagnosis and Intervention: MRI brain showed normal parenchyma. MR venography (MRV) demonstrated absence of flow in the right transverse and sigmoid sinuses, confirming acute CVT. Extended thrombophilia screening and fundus examination were normal. The patient was commenced on levetiracetam 500 mg intravenously twice daily, enoxaparin 60 mg (0.6 mL) subcutaneously twice daily, and intravenous hydration. An emergency lower segment caesarean section (LSCS) was performed. Postoperatively, acenocoumarol was initiated; supratherapeutic INR on postoperative day (POD) 7 required temporary withholding of anticoagulation and administration of intravenous Vitamin K 10 mg, with subsequent dose reduction and restabilisation. The patient was discharged on POD 10 on combination enoxaparin and acenocoumarol.

Outcomes: On POD 15, the patient was readmitted with breathlessness and bilateral pedal oedema. Echocardiography confirmed severe left ventricular dysfunction, establishing a diagnosis of PPCM. She was managed by cardiology with diuretics, beta-blockers, and calcium channel blockers, with satisfactory clinical improvement and discharge.

Conclusion: This case highlights the importance of a high index of suspicion for CVT in pregnant women with progressive or thunderclap headache, regardless of the absence of focal neurological deficits. MRV is indispensable when MRI brain is non-contributory. Prompt LMWH anticoagulation, vigilant INR monitoring in the postpartum period, and early echocardiographic surveillance for PPCM are essential components of management in this setting.

Keywords: cerebral venous thrombosis, pregnancy, peripartum cardiomyopathy, low molecular weight heparin, case report

Informed Consent: Written informed consent obtained from the patient for publication.

How to cite this article: Reddy N., Cerebral Venous Thrombosis in Term Pregnancy Complicated by Peripartum Cardiomyopathy: A Case Report. Int J Drug Deliv Technol. 2026;16(44s): 452-463; DOI: 10.25258/ijddt.16.44s.48

INTRODUCTION

Cerebral venous thrombosis (CVT) is an uncommon but serious cerebrovascular disease that disproportionately affects women of reproductive age. Pregnancy and the puerperium represent well-established prothrombotic states, attributed to the physiological hypercoagulability that develops as a haemostatic adaptation to prevent excessive peripartum blood loss [1]. The incidence of CVT in pregnancy is estimated at approximately 1–2 per 10,000 deliveries in high-income countries; population-based studies from India report higher figures ranging from 4.5 to 10 per 100,000 deliveries, likely reflecting epidemiological differences as well as improved case detection [2,3]. The condition carries a case fatality rate of up to 5–10% when diagnosis or

treatment is delayed, with significant neurological morbidity in survivors [4].

The clinical presentation of CVT is heterogeneous, ranging from isolated thunderclap headache to focal neurological deficits, seizures, papilloedema, and altered consciousness [5]. In the obstetric context, this diagnostic ambiguity is compounded by symptom overlap with pre-eclampsia, eclampsia, and benign intracranial hypertension of pregnancy [6]. MRI brain combined with MR venography (MRV) is the gold standard investigation, offering superior sensitivity over CT venography, particularly for non-haemorrhagic CVT and posterior fossa sinus thrombosis [7]. The mainstay of treatment is anticoagulation with low molecular weight heparin (LMWH), which is safe in pregnancy and effective in promoting sinus recanalisation [8]. The timing and mode of delivery in CVT complicating term pregnancy remains a matter of clinical judgement in the absence of consensus guidelines [9]. The co-occurrence of peripartum cardiomyopathy (PPCM) — itself a rare entity with an incidence of 1 in 1,000–4,000 deliveries — in the setting of CVT represents an exceptionally uncommon clinical scenario with very limited published data [10]. We report such a case to highlight the diagnostic challenges, management decisions, and clinical implications of this rare co-occurrence.

PATIENT INFORMATION

De-identified Patient Information

A 38-year-old woman (gravida 2, abortus 1; G2A1), at 37 weeks and 2 days of gestation, with Rh-negative blood group (indirect Coombs test negative), was referred from a peripheral hospital to our tertiary care centre for evaluation and management of acute-onset severe headache.

Primary Concerns and Symptoms

The patient's primary complaints were sudden-onset severe headache, bifrontal and occipital in distribution, gradually progressive over one day and unresponsive to analgesics administered at the referring facility. This was accompanied by two episodes of projectile vomiting, non-bilious and non-blood-stained. There were no complaints of visual disturbances, seizures, focal limb weakness, speech difficulty, altered sensorium, or loss of consciousness. Fetal movements were perceived satisfactorily throughout.

Medical, Family, and Psychosocial History

The current pregnancy had been conceived via ovarian induction (OI). The patient had no prior history of cerebrovascular disease, epilepsy, hypertension, diabetes mellitus, or known autoimmune disorder. There was no family history of thromboembolic disease or inherited coagulopathy. No relevant psychosocial history was elicited. The previous pregnancy had resulted in an abortion; detailed circumstances were not documented in the available records.

Relevant Past Interventions and Outcomes

During the current pregnancy, the patient had been prescribed Tab. Ecosprin 150 mcg once daily, Tab. Susten 200 mg, and Inj. Proluton Depot 500 mg once every 15 days throughout the first trimester in view of the OI conception — all without adverse events. Anti-D immunoglobulin (RAADP 300 mcg) was administered at 28 weeks as per protocol for Rh-negative mothers. Ecosprin was electively discontinued at 32 weeks. A third trimester growth scan was reported as normal.

CLINICAL FINDINGS

On general examination, the patient was conscious, oriented, and afebrile. Vital parameters were: blood pressure 120/80 mmHg, pulse rate 76 beats per minute (regular), respiratory rate 18 breaths per minute, and SpO_2 98% on room air. There was no pallor, icterus, cyanosis, clubbing, pedal oedema, or lymphadenopathy. Cardiovascular examination revealed normal heart sounds with no murmurs. Respiratory examination revealed bilaterally equal vesicular breath sounds.

Obstetric examination revealed a uterus corresponding to term gestation, relaxed and non-tender, with a longitudinal lie and cephalic presentation. Liquor was clinically adequate. Fetal heart rate was normal and fetal movements were perceived satisfactorily. Per vaginal examination findings are not documented in the available case record.

Neurological examination did not reveal focal deficits. Cranial nerve examination was unremarkable. Motor power, tone, and reflexes in all four limbs were within normal limits. Fundus examination obtained via ophthalmology consultation revealed no papilloedema, haemorrhages, or exudates — an important finding given the clinical concern for raised intracranial pressure.

TIMELINE

The chronological sequence of clinical events is summarised in Table 1. POD = postoperative day.

Table 1. Timeline of Clinical Events

Time Point	Event / Findings	Action Taken
First Trimester	OI conception confirmed. Uneventful antenatal period.	Tab. Ecosprin 150 mcg OD, Tab. Susten 200 mg, Inj. Proluton Depot 500 mg once in 15 days commenced.
28 Weeks of Gestation	Rh-negative status — no alloimmunisation (ICT negative).	Anti-D immunoglobulin (RAADP 300 mcg) administered.
32 Weeks of Gestation	Third trimester growth scan — normal fetal growth and liquor.	Tab. Ecosprin electively discontinued.
37 Weeks 2 Days — Day of Admission	Sudden-onset severe bifrontal and occipital headache (progressive, unresponsive to analgesics) with 2 episodes of projectile vomiting. Referred from peripheral hospital.	Admitted to tertiary care. History, general, obstetric, and neurological examination performed. Vitals stable.
Day of Admission — Investigations	CBC, RFT, LFT, uric acid, LDH, serum fibrinogen, peripheral smear, coagulation profile — all within normal limits. Urine albumin 1+.	Neuroimaging urgently ordered.
Day of Admission — MRI Brain	MRI brain: normal parenchyma. No infarct, haemorrhage, or mass lesion identified. (Figure 1, Figure 2)	MR venography (MRV) ordered in view of high clinical suspicion for CVT.
Day of Admission — MR Venography	MRV: absence of flow signal in right transverse sinus and right sigmoid sinus — acute CVT confirmed. (Figure 3, Figure 4)	Diagnosis established. Neurology and ophthalmology consultations obtained.
Day of Admission — Ophthalmology Review	Fundus examination — normal. No papilloedema.	Raised ICP-related changes excluded.
Day of Admission — Treatment Initiated	Acute CVT at 37 weeks 2 days gestation confirmed.	Inj. Levetiracetam 500 mg IV BD commenced. Inj. Enoxaparin 60 mg (0.6 mL) SC BD commenced. IV hydration at 75–100 mL/hour initiated.
Day of Admission	Protein C, Protein S, lupus	Acquired and hereditary

Time Point	Event / Findings	Action Taken
— Thrombophilia Screen	anticoagulant, homocysteine, antithrombin III, beta-2 glycoprotein IgG and IgM — all negative.	thrombophilia excluded.
Day of Admission — Delivery Decision	Acute CVT at term gestation. Multidisciplinary decision taken.	Emergency LSCS performed. Intraoperative period uneventful. Estimated blood loss 500 mL.
POD 0	No neurological symptoms. Vitals stable. Urine output adequate.	Inj. Enoxaparin 60 mg (0.6 mL) SC restarted 6 hours post-surgery. Inj. Levetiracetam 500 mg IV BD continued.
POD 5	Clinical condition stable. Transition to oral anticoagulation initiated.	Tab. Acenocoumarol 2 mg BD commenced.
POD 7	Serial INR monitoring — supratherapeutic values noted.	Inj. Enoxaparin and Tab. Acenocoumarol both withheld.
POD 8	INR elevated on repeat monitoring.	Inj. Vitamin K 10 mg IV administered. INR corrected.
		Enoxaparin and Acenocoumarol restarted at reduced doses.
POD 10	Patient clinically stable. Neurologically intact.	Discharged on Inj. Enoxaparin 60 mg (0.6 mL) SC BD + Tab. Acenocoumarol 1 mg and 1.5 mg on alternate nights.
POD 15 (Post-discharge)	Patient presented with 2-day history of breathlessness and bilateral pedal oedema.	Admitted under Cardiology. Echocardiography performed.
POD 15 — Cardiology Admission	Echocardiography: severe LV dysfunction. Diagnosis: Peripartum Cardiomyopathy (PPCM).	Managed with diuretics, beta-blockers, and calcium channel blockers. Clinical improvement achieved. Discharged with medications.

DIAGNOSTIC ASSESSMENT

Diagnostic Testing

A comprehensive panel of investigations was undertaken on admission to evaluate the aetiology of symptoms and to exclude common obstetric causes of headache and neurological deterioration in the third trimester.

Laboratory Investigations

Complete blood count, peripheral blood smear, and coagulation profile were within normal limits. Serum

fibrinogen was not elevated. Renal function tests, liver function tests, serum uric acid, and lactate dehydrogenase (LDH) - markers employed to screen for pre-eclampsia with severe features and HELLP syndrome - were unremarkable. Urine dipstick showed albumin 1+, which did not meet the diagnostic threshold for pre-eclampsia in the absence of hypertension or other supportive features.

Neuroimaging

MRI brain was performed as the first-line neuroimaging modality. T2-weighted and FLAIR axial sequences revealed normal cerebral parenchyma with no infarction, haemorrhage, oedema, or mass lesion (Figure 1, Figure 2). In view of persistent clinical suspicion for CVT, MR venography (MRV) was performed using time-of-flight (TOF) sequences and demonstrated absence of flow signal in the right transverse sinus and right sigmoid sinus, confirming acute cerebral venous sinus thrombosis (Figure 3, Figure 4).

Ophthalmological Assessment

Fundus examination by an ophthalmologist was normal bilaterally, with no disc oedema, haemorrhages, or exudates, effectively excluding raised intracranial pressure-related changes.

Thrombophilia Screening

An extended thrombophilia panel was performed to identify hereditary or acquired prothrombotic predisposition. The panel included Protein C activity, Protein S activity, antithrombin III levels, lupus anticoagulant, serum homocysteine, antithrombin 3 IgG and IgM, and beta-2 glycoprotein IgG and IgM. All parameters were negative, excluding both inherited thrombophilia and antiphospholipid antibody syndrome.

Diagnostic Challenges

The primary diagnostic challenge was the absence of focal neurological signs. Headache and vomiting in the obstetric context are frequently attributed to migraine, tension-type headache, or hypertensive disorders of pregnancy. Normal blood pressure and unremarkable pre-eclampsia markers directed suspicion towards an intracranial aetiology. Critically, a normal MRI brain could have prematurely reassured the treating team had MRV not been pursued, underlining that a normal MRI does not exclude CVT, and dedicated venographic imaging must be obtained whenever clinical suspicion persists. The co-existence of mild proteinuria (urine albumin 1+) introduced further diagnostic ambiguity requiring careful contextualisation.

Diagnosis

Primary Diagnosis: Acute cerebral venous sinus thrombosis- right transverse sinus and right sigmoid sinus thrombosis- at 37 weeks 2 days of gestation.

Differential diagnoses considered and systematically excluded are presented in Table 2.

Table 2. Differential Diagnoses Considered and Excluded

Differential Diagnosis	Basis for Consideration	Reason for Exclusion
Pre-eclampsia with severe features	Headache, vomiting, proteinuria 1+	BP normal; LFT, RFT, uric acid, LDH, platelet count all normal
Hypertensive encephalopathy	Headache, vomiting	No hypertension documented at any point
Migraine / Tension headache	Bifrontal headache	Sudden onset, progressive, unresponsive to analgesics; neuroimaging abnormal
Benign intracranial hypertension	Headache, vomiting, pregnancy	No papilloedema; MRV confirms sinus thrombosis as aetiology
Meningitis / Encephalitis	Headache, vomiting	Afebrile; no meningeal signs; normal MRI parenchyma
Subarachnoid haemorrhage	Thunderclap headache	No haemorrhage

Differential Diagnosis	Basis for Consideration	Reason for Exclusion
		on MRI; MRV confirms venous pathology
Intracranial space-occupying lesion	Progressive headache	Normal MRI parenchyma

Prognosis

At diagnosis, the patient had no focal neurological deficits, no papilloedema, and no haemorrhagic transformation on MRI brain. GCS was full. Thrombosis was confined to the right transverse and sigmoid sinuses, with the superior sagittal sinus and deep venous system remaining patent. Thrombophilia screening was negative. These features collectively indicated a clinically stable presentation with a favourable short-term prognosis as assessed by the treating team.

THERAPEUTIC INTERVENTION

Types of Therapeutic Intervention

The patient received a combination of pharmacological and surgical interventions, instituted in a stepwise and clinically reasoned manner across the antepartum, intraoperative, and postoperative periods.

Pharmacological Interventions

Anticoagulation with enoxaparin (LMWH) was initiated as the primary treatment for acute CVT and continued through the postoperative period with subsequent transition to oral anticoagulation. Levetiracetam was commenced prophylactically given the risk of seizures associated with intracranial sinus thrombosis. Intravenous isotonic fluid therapy was administered for cerebral venous hydration and reduction of blood viscosity. Acenocoumarol, a vitamin K antagonist, was introduced postoperatively for long-term anticoagulation with INR-guided dose titration. Intravenous Vitamin K was administered in response to supratherapeutic INR.

Surgical Intervention

Emergency lower segment caesarean section (LSCS) was performed in view of the acute CVT at term gestation, with the dual objective of securing fetal wellbeing and enabling safer postoperative anticoagulation without the haemorrhagic risks associated with labour and vaginal delivery.

Post-discharge Cardiac Management

Diuretics, beta-blockers, and calcium channel blockers were instituted by the cardiology team following the diagnosis of PPCM with severe left ventricular (LV) dysfunction on POD 15.

Administration of Therapeutic Interventions

The complete pharmacotherapy regimen is detailed in Table 3.

Table 3. Summary of Therapeutic Agents — Dosage, Route, Frequency, and Duration

Drug	Dose	Route	Frequency	Duration / Remarks
Levetiracetam (Levipil)	500 mg	Intravenous	Twice daily (BD)	Commenced on day of admission; continued postoperatively
Enoxaparin (Clexane)	60 mg (0.6 mL)	Subcutaneous	Twice daily (BD)	Commenced on day of admission; withheld perioperatively; restarted 6 hours post-LSCS
IV Fluids (Isotonic)	75–100 mL/hour	Intravenous	Continuous infusion	Commenced on day of admission for cerebral venous

Drug	Dose	Route	Frequency	Duration / Remarks
				hydration
Acenocoumarol (Acitrom)	2 mg	Oral	Twice daily (BD)	Started on POD 5
Acenocoumarol (Acitrom)	0.5 mg	Oral	At night (HS)	Restarted at reduced dose on POD 8 following Vitamin K correction
Vitamin K	10 mg	Intravenous	Single dose	Administered on POD 8 for supratherapeutic INR
Enoxaparin (Clexane)	60 mg (0.6 mL)	Subcutaneous	Twice daily (BD)	Restarted on POD 8; continued at discharge
Acenocoumarol (Acitrom)	1 mg / 1.5 mg	Oral	Alternate nights (HS)	Discharge prescription — alternating dose regimen
Beta-blocker	Not specified	Oral	As prescribed	Initiated by Cardiology for PPCM
Calcium channel blocker	Not specified	Oral	As prescribed	Initiated by Cardiology for PPCM
Diuretic	Not specified	Oral	As prescribed	Initiated by Cardiology for PPCM

Changes in Therapeutic Intervention (with Rationale)

Modification 1 - Perioperative enoxaparin withholding: Enoxaparin was withheld in the immediate perioperative period to minimise surgical haemorrhage risk. It was recommenced at 60 mg (0.6 mL) subcutaneously twice daily at 6 hours post-LSCS, once haemostasis was confirmed and estimated blood loss (500 mL) was deemed acceptable.

Modification 2 - Supratherapeutic INR on POD 7: Following introduction of acenocoumarol 2 mg twice daily on POD 5, serial INR monitoring on POD 7 revealed supratherapeutic values. Both acenocoumarol and enoxaparin were withheld. On POD 8, intravenous Vitamin K 10 mg was administered, normalising the INR. Anticoagulation was recommenced at a reduced acenocoumarol dose (0.5 mg nightly) alongside enoxaparin 60 mg (0.6 mL) twice daily. The supratherapeutic response likely reflects the well-recognised heightened sensitivity to vitamin K antagonists in the early postpartum period, when rapid physiological reversal of procoagulant changes amplifies anticoagulant effect.

Modification 3 — Transition to alternating acenocoumarol at discharge: At POD 10, the acenocoumarol regimen was adjusted to an alternating dose schedule (1 mg and 1.5 mg on alternate nights) to

achieve stable therapeutic INR on an outpatient basis, with enoxaparin continued as bridging cover pending INR stabilisation.

FOLLOW-UP AND OUTCOMES

Clinician and Patient-Assessed Outcomes

The immediate maternal outcome was favourable. Following emergency LSCS, the patient remained haemodynamically stable with no recurrence or progression of neurological symptoms. No seizure activity was observed at any point during the hospital stay. At discharge on POD 10, the patient was conscious, oriented, and ambulatory with no focal neurological deficits. Neonatal outcome is not documented in the available records.

The postoperative course was complicated by PPCM with severe LV dysfunction, diagnosed on POD 15 following emergency readmission for breathlessness and bilateral pedal oedema. Following cardiology-directed management, satisfactory clinical improvement was achieved with good diuresis, resolution of breathlessness, and reduction in oedema prior to the second discharge.

Important Follow-up Diagnostic and Other Test Results

Serial INR values and corresponding management decisions are outlined in Table 4.

Table 4. Postoperative INR Monitoring and Corresponding Clinical Actions

Day	INR Status	Action Taken
POD 5	Within normal range	Tab. Acenocoumarol 2 mg BD commenced
POD 7	Supratherapeutic — elevated beyond target range	Enoxaparin and Acenocoumarol withheld
POD 8	Elevated — persisting supratherapeutic levels	Inj. Vitamin K 10 mg IV administered; INR corrected
POD 8 (post-Vitamin K)	Within normal limits	Enoxaparin and Acenocoumarol restarted at reduced doses
POD 10 (Discharge)	Stable — within therapeutic range	Discharged on alternating acenocoumarol + enoxaparin

Echocardiography during the second admission revealed severe left ventricular systolic dysfunction consistent with PPCM. Specific left ventricular ejection fraction (LVEF) values are not available in the case records; however, the severity was classified as severe by the cardiology team. Follow-up MRV to document sinus recanalisation was not performed during the available follow-up period.

Intervention Adherence and Tolerability

The patient demonstrated satisfactory adherence to the prescribed anticoagulation regimen, as evidenced by regular enoxaparin administration and INR monitoring during the inpatient stay. The INR fluctuation on POD 7–8 represented a pharmacokinetic phenomenon rather than non-adherence. Enoxaparin was well tolerated with no documented injection site reactions, thrombocytopaenia, or allergic responses. Levetiracetam was tolerated without adverse effects. Cardiac medications during the second admission were tolerated with good clinical response.

Adverse and Unanticipated Events

Event 1 - Supratherapeutic INR (POD 7): Supratherapeutic INR developed following initiation of acenocoumarol 2 mg twice daily on POD 5. Both acenocoumarol and enoxaparin were withheld. Vitamin K 10 mg intravenously on POD 8 resulted in INR correction without haemorrhagic complication. This is attributable to postpartum pharmacokinetic amplification of vitamin K antagonist effect.

Event 2 - Peripartum Cardiomyopathy (POD 15): The patient presented to the emergency department on POD 15 with a two-day history of progressive breathlessness and bilateral pedal oedema. As cardiovascular examination had been unremarkable throughout the index admission, this was an unanticipated development. Echocardiography confirmed severe LV systolic dysfunction meeting diagnostic criteria for PPCM. The pathophysiological relationship between CVT and PPCM in this patient is discussed further in the Discussion section.

DISCUSSION

CVT in pregnancy is a rare but well-recognised complication of the gestational hypercoagulable state. The physiological increase in clotting factors V, VIII, X, and fibrinogen, combined with reduced Protein S activity and impaired fibrinolysis, creates a prothrombotic milieu persisting until approximately six weeks postpartum [1]. While CVT can occur at any gestational age, the majority of pregnancy-associated cases are reported in the third trimester and early postpartum period, consistent with the presentation in this case at 37 weeks [2,3].

CVT incidence in pregnancy ranges from 1 to 2 per 10,000 deliveries globally, with South Asian populations reporting rates of 4.5 to 10 per 100,000, attributed to nutritional deficiencies, anaemia, dehydration, and higher rates of inherited thrombophilia [2,3]. The present case involved a woman of advanced maternal age (38 years) with an OI conception — both independently recognised risk factors for thromboembolic disease in pregnancy [4].

Headache is the most common symptom of CVT, reported in over 90% of cases [5]. The sudden-onset severe bifrontal and occipital headache unresponsive to analgesics with projectile vomiting in this case is consistent with raised intracranial pressure from venous outflow obstruction. The absence of focal neurological deficits at presentation, though reassuring, is well documented in CVT and must not reduce clinical suspicion [5]. The mild proteinuria in this case introduced diagnostic overlap with pre-eclampsia — a recognised pitfall in obstetric CVT [6].

MRI combined with MRV is the imaging modality of choice, offering superior sensitivity over CT venography for posterior fossa sinus thrombosis and non-haemorrhagic infarcts [7]. Normal MRI parenchyma does not exclude CVT; up to 30% of cases may have normal conventional sequences, with diagnosis resting entirely on venographic demonstration of absent flow [7]. MRV in this case confirmed absence of flow in the right transverse and sigmoid sinuses (Figure 3, Figure 4), with the superior sagittal sinus and deep venous system remaining patent — a finding associated with relatively favourable prognosis.

LMWH anticoagulation is the cornerstone of CVT management in pregnancy and is recommended even in the presence of haemorrhagic infarction, based on evidence from randomised trials demonstrating reduced mortality and dependency without increased haemorrhagic risk [8]. Enoxaparin was commenced promptly at therapeutic doses following diagnosis. Transition to acenocoumarol from POD 5, detailed in Table 3, is consistent with current anticoagulation guidelines [8].

The decision for emergency LSCS in this case merits discussion. No randomised trials or consensus guidelines exist for the optimal mode of delivery in CVT at term. While vaginal delivery under epidural analgesia is feasible in neurologically stable patients, emergency LSCS is appropriate when clinical urgency precludes a trial of labour [9]. The uneventful intraoperative course with an estimated blood loss of 500 mL reflects appropriate perioperative anticoagulation management.

The supratherapeutic INR on POD 7 is a clinically instructive finding (Table 4). Rapid postpartum reversal of pregnancy-associated procoagulant changes — including falling clotting factor levels and recovering Protein S activity — dramatically amplifies the effect of vitamin K antagonists [8]. The initial acenocoumarol dose of 2 mg twice daily was likely excessive in this physiological context. Successful INR correction with intravenous Vitamin K 10 mg without haemorrhagic complication reflects timely clinical recognition and appropriate escalation.

The most academically distinctive aspect of this case is the development of PPCM with severe LV dysfunction on POD 15. PPCM is an idiopathic cardiomyopathy presenting with heart failure secondary to LV systolic dysfunction at the end of pregnancy or within months of delivery, in the absence of another identifiable cause [10]. Its incidence ranges from 1 in 1,000 to 1 in 4,000 deliveries, with higher rates in older multiparous women — consistent with the profile of this patient [10]. The co-occurrence of CVT and PPCM within the peripartum period is exceptionally rare and has not been well characterised in published literature. Both conditions share a common pathophysiological substrate: endothelial dysfunction, systemic inflammation, oxidative stress, and microvascular dysregulation — mechanisms that may explain their temporal clustering in susceptible individuals [11,12]. This shared vulnerability underscores the importance of maintaining vigilance for cardiac decompensation in patients presenting with peripartum CVT, particularly those with additional risk factors such as advanced maternal age and ovarian induction. The concurrent anticoagulation

requirement for CVT and the need for heart failure therapy in PPCM introduces therapeutic complexity, as agents such as angiotensin-converting enzyme inhibitors are contraindicated in the breastfeeding period and must be substituted with compatible alternatives [11].

Strengths and Limitations

This case adds to the sparse published literature on the rare co-occurrence of CVT and PPCM within the peripartum period. The systematic diagnostic workup — including extended thrombophilia screening, MRI brain, MRV, fundus examination, and echocardiography — illustrates a methodical clinical approach. The detailed anticoagulation record, including the INR fluctuation and its management, offers instructive insight into the pharmacokinetic vulnerabilities unique to the postpartum period. High-quality MRI and MRV images add significant illustrative value.

The limitations include: (1) single case report design precluding generalisable conclusions; (2) absence of specific LVEF values limiting objective quantification of cardiac dysfunction; (3) no follow-up MRV to assess sinus recanalisation; (4) unavailability of long-term neurological and cardiac outcome data; and (5) absence of neonatal outcome documentation.

Scientific Rationale for Conclusions

The temporal co-occurrence of CVT and PPCM in this patient is unlikely coincidental. Both conditions share pathophysiological mechanisms of endothelial injury, inflammatory cytokine activation, and microvascular dysfunction [11,12]. Advanced maternal age, OI conception, and late pregnancy hypercoagulability likely acted as converging risk factors [4,10]. Negative thrombophilia screening suggests a provoked thrombotic event attributable to the pregnancy-associated prothrombotic state, with implications for counselling regarding anticoagulation duration and recurrence risk in future pregnancies [8].

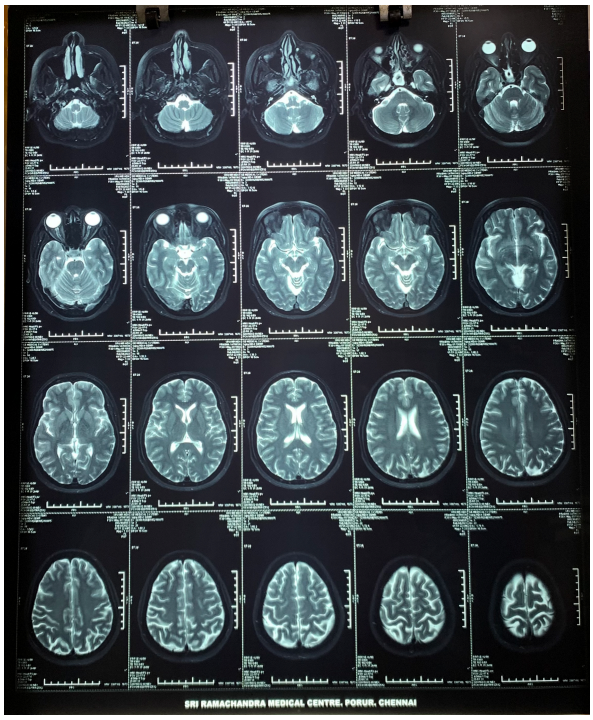

Figure 1. *MRI Brain — T2-weighted axial sequences demonstrating normal cerebral parenchyma with no infarction, haemorrhagic transformation, perilesional oedema, or mass effect. Ventricular system is normal in size and configuration. Study performed at Sri Ramachandra Medical Centre, Porur, Chennai.*

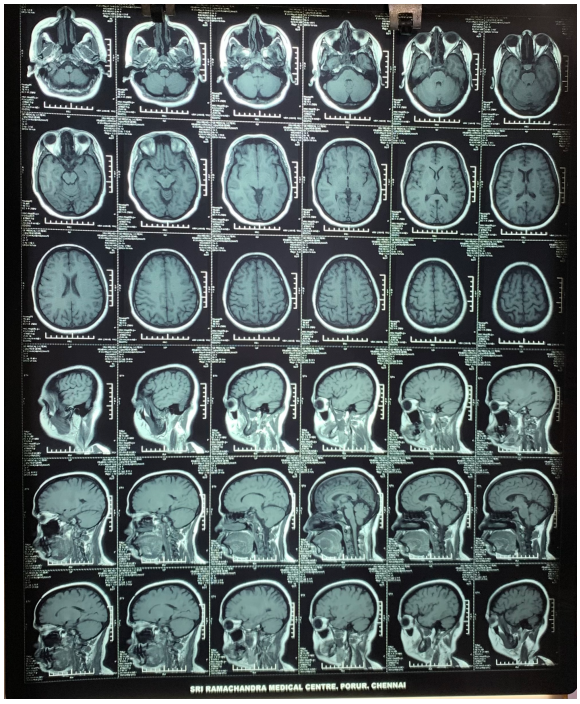

Figure 2. *MRI Brain — T1-weighted axial and sagittal sequences demonstrating normal cerebral parenchyma with preserved cortical gyri and sulci. No*

haemorrhagic signal or venous infarction identified. Study performed at Sri Ramachandra Medical Centre, Porur, Chennai.

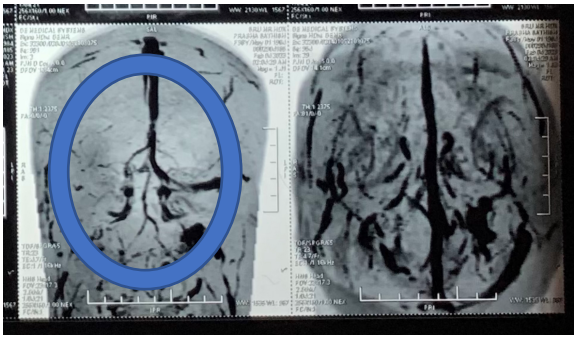

Figure 3. *MR Venography — Coronal projections demonstrating markedly diminished flow signal in the right transverse sinus, consistent with acute thrombotic occlusion. The superior sagittal sinus and left transverse sinus show preserved flow signal. Study performed at Sri Ramachandra Medical Centre, Porur, Chennai.*

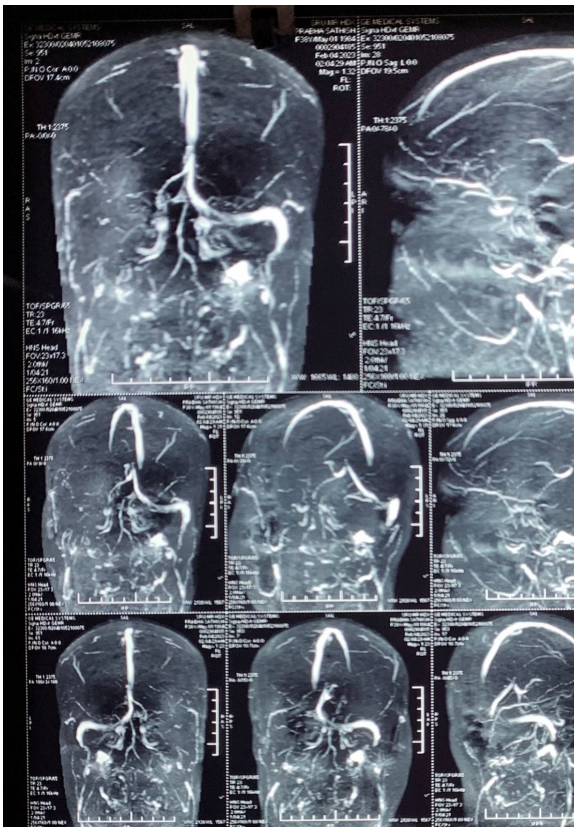

Figure 4. *MR Venography — Time-of-flight (TOF) sequences in coronal and sagittal projections demonstrating absence of flow signal in the right transverse sinus and right sigmoid sinus, confirming acute thrombotic occlusion of the right-sided posterior venous drainage pathway. The superior sagittal sinus, straight sinus, left transverse sinus, and deep venous system appear patent. Study performed at Sri Ramachandra Medical Centre, Porur, Chennai.*

CONCLUSION

This case of acute CVT involving the right transverse and sigmoid sinuses in a term pregnancy, complicated by PPCM with severe LV dysfunction, illustrates the diagnostic and therapeutic complexity that can arise at the intersection of rare obstetric and neurological emergencies. CVT must be considered in any pregnant woman presenting with progressive or thunderclap headache unresponsive to analgesics, irrespective of normal blood pressure, absence of focal deficits, or normal MRI parenchyma. MRV is an indispensable second-line investigation. Prompt LMWH therapy is safe and effective. Mode of delivery requires individualised multidisciplinary decision-making. Vitamin K antagonists in the early postpartum period demand vigilant INR monitoring. Most critically, this case highlights that PPCM may co-occur with CVT within the peripartum period, sharing a common substrate of endothelial vulnerability — a rare but clinically significant association that mandates early echocardiographic evaluation and coordinated care across obstetrics, neurology, and cardiology.

PATIENT PERSPECTIVE

The patient described the onset of her headache as sudden and unlike any previous experience — severe, unrelenting, and unresponsive to medications at the referring hospital. She expressed considerable anxiety on arrival at the tertiary centre, particularly regarding fetal wellbeing given the history of prior pregnancy loss and the current assisted conception. The period between neuroimaging and the decision for emergency caesarean section was described as deeply distressing, though she felt reassured by clear communication from the treating team regarding the diagnosis and surgical rationale.

In the postoperative period, she noted gradual improvement in headache symptoms following initiation of anticoagulation and hydration. She was compliant with prescribed medications and INR monitoring during the inpatient stay. She expressed relief at discharge on POD 10 in a stable condition. The onset of breathlessness and leg swelling within two weeks of discharge caused considerable alarm; she presented promptly to the emergency department, attributing this decision to counselling received during

her index admission regarding the importance of early reporting of new symptoms. Following cardiology management for PPCM, she reported progressive symptomatic improvement and expressed gratitude for the coordinated multidisciplinary care received.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images, including MRI brain and MR venography sequences. The patient was informed of the academic and educational purpose of this publication and was assured of complete anonymity, with all identifying information de-identified in accordance with institutional guidelines and the Declaration of Helsinki. A copy of the signed consent form is available for review upon request by the editorial office.

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