

## CASE REPORT

# Eclampsia and postpartum onset of subarachnoid hemorrhage in dual setting of cerebral venous thrombosis and posterior reversible encephalopathy syndrome: A case report

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## Abstract

Subarachnoid hemorrhage (SAH) can be an initial presentation of cerebral venous thrombosis. Eclampsia and postpartum onset of SAH should prompt physicians to identify possible causes and institute prompt management to avert irreversible cerebrovascular sequelae.

## KEYWORDS

cerebral venous thrombosis, eclampsia, posterior reversible encephalopathy syndrome, subarachnoid hemorrhage

## 1 | INTRODUCTION

Subarachnoid hemorrhage (SAH) can be an initial presentation of cerebral venous thrombosis. Eclampsia with SAH secondary to postpartum cerebral venous thrombosis and posterior reversible encephalopathy syndrome. Eclampsia and postpartum onset of SAH should prompt physicians to identify possible causes and institute prompt management to avert irreversible cerebrovascular sequelae.

The global incidence of cerebral venous thrombosis (CVT) is rare at the rate of 5 per million population and accounts for <1% of all the strokes reported.<sup>1</sup> Incidence of CVT in pregnancy and puerperium vary from 11.6 to over 200 per 100 000 deliveries per year due to rarity and diverse clinical manifestations.<sup>2,3</sup> Risk factors associated with CVT include younger age, anemia, hypercoagulable state, gender-specific risk factors such as hormonal contraceptive use, hormone replacement therapy, pregnancy and puerperium, and hypertensive disorders in pregnancy.<sup>3-5</sup> In pregnancy and

puerperium, various mechanisms are postulated to increase the risk of CVT such as iron deficiency anemia, hypercoagulable state, altered platelet functions and obstetric trauma, and hemorrhage.<sup>3,6</sup>

Depending on the involvement of various dural veins, sinuses, and accompanying brain parenchymal involvement, clinical presentations are nonspecific and include a wide array of neurological features. Patients present with headache, motor and sensory impairment, seizures, altered sensorium, and secondary intracranial hemorrhage.<sup>6</sup> Mortality in pregnancy-related CVT is higher in the presence of hypertensive disorder of pregnancy, sepsis, intracranial hemorrhage, and seizures.<sup>6</sup>

## 2 | CASE PRESENTATION

A 41-year-old elderly G3P2 without any significant medical, family, or obstetric history was diagnosed with the current

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pregnancy at 13 weeks by ultrasound. There was an intramural myoma 11 × 6 cm on the left uterine wall. In view of advanced maternal age, longer interpregnancy interval (10 years), and poor placentation due to myoma uteri, oral Aspirin 150 mg daily was also started to reduce the risk of preeclampsia.

At 30-week gestation, she presented with worsening headache, facial and hand swelling, and epigastric pain over 3 days duration. She did not complain of blurring of vision, nausea, or vomiting. There was no per vaginal bleeding, and fetal movement was perceived normal. On examination, she was afebrile, conscious, not dyspneic, and facial puffiness was noted. Pulse rate was 78 beats per minute, BP-198/115 mm Hg. Respiratory rate was 16 per minute. Bilateral lungs were clear on auscultation. Per abdomen examination showed gravid uterus corresponding to 33 weeks in cephalic presentation with adequate liquor. Cardiotocograph tracing was reassuring. Preeclampsia workup showed the following reports (Table 1).

Hypertensive crisis management with IV Labetolol and prophylactic Mg SO<sub>4</sub> along with antenatal corticosteroid was carried out as per the hospital protocol.

Provisional diagnosis of preeclampsia-HELLP syndrome was made. Emergency Cesarean section was decided under spinal anesthesia and mother taken to operation room. She developed intra cesarean generalized tonic clonic convulsions. Immediate conversion to general anesthesia was performed to protect the airway and top up IV Mg SO<sub>4</sub> 2 g administered to control convulsions. Male baby weighing 800 g was delivered with Apgar score of 6 at 1 minute and was shifted to NICU. Mother was also shifted to ICU for observation. She remained drowsy and complained of persistent headache and blurring of vision. There was no focal neurological deficit, and fundoscopy revealed no papilledema or retinal detachment. Her BP remained elevated with worsening of renal and liver function test for two consecutive postoperative days despite treatment with antihypertensive drugs. She also had worsening thrombocytopenia at 31 000/mm<sup>3</sup> with gum bleeding during that period. Four units of platelet was transfused. CT brain was performed due to persistent neurological symptoms which revealed left convexal frontal lobe subarachnoid hemorrhage (SAH) (Figure 1). One time setting MRI brain showed acute left frontal lobe SAH, dural venous sinus thrombosis of left transverse, and sigmoid sinus (Figure 2). There was also T2/FLAIR hyperintensities noted in bilateral

occipital region (Figure 3). No aneurysm or constriction of cerebral vessels were detected on MR vascular studies.

Interdisciplinary consultation with medical team advised Tab Nimodipine 30 mg 6 hourly to prevent cerebral vasospasm-related parenchymal injuries. Intravenous Hydralazine and Labetolol was used to achieve optimal control of blood pressure and prevent further worsening of PRES. In view of thrombocytopenia related bleeding, low molecular weight heparin (LMWH) was not used to treat CVT. Anticoagulation with Warfarin 3 mg daily was started and dose adjusted based on the PT/INR value at 2-3. Her blood pressure and neurological symptoms improved with the treatment and objective assessment with repeat CT brain in 1 week time showed resolving SAH. We are not able to study the inherited thrombophilia profile in our case as protocol issues persist in sending the blood samples abroad due to current COVID pandemic. We decided to anticoagulate for 6-month postpartum in the presence of provoking factor and presumed absence of inherited thrombophilic status as per AHA/ASA guidelines 2014. Follow-up MRI studies at 6-week postpartum revealed no residual CVT or SAH.

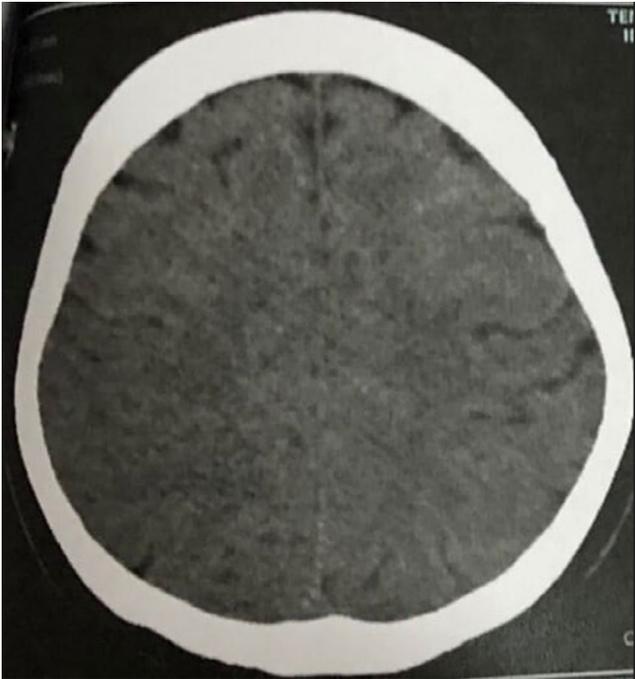
### 3 | DISCUSSION

There is a considerable overlap in the clinical presentation of CVT and SAH. Aneurysmal SAH constitutes about 85%.<sup>7</sup> The remaining nonaneurysmal SAH is divided radiologically into convexal and perimesencephalic. Possible mechanisms to look for pregnancy-related convexal SAH include posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome (RCVS), and CVT.<sup>8</sup> Azeemuddin et al<sup>9</sup> found out that SAH as initial presentation in cerebral venous thrombosis is about 10%. Therefore, when radiological findings are suggestive of non aneurysmal convexal SAH, further evaluation is warranted to look for possible causes.<sup>10</sup> This is of paramount importance as management is different in each setting.

Our case did not present with the classical acute onset headache of aneurysmal SAH nor did she have risk factors such as chronic hypertension, smoking, drug abuse, or alcohol use. Her initial headache was subacute onset, frontoparietal, and throbbing in nature which favor more toward CVT as found out by Botta et al.<sup>11</sup> Probably, sepsis as shown by neutrophil leucocytosis and worsening thrombocytopenia

**TABLE 1** Showing laboratory investigation report

|                      |  |
|----------------------|--|
| Urine albumin        | 4+   |
| Complete blood count | TLC 27 760/mm <sup>3</sup> , Neutrophil-84%, Hemoglobin-11 g/dL, platelet-85 000/mm <sup>3</sup> |
| Renal function test  | Serum creatinine-1.6 mg/dL, urea-73 mg/dL  |
| Liver function test  | Serum AST-314/ALT-221 IU/L (5-40), serum LDH-2387 IU/L (180-360)                                 |

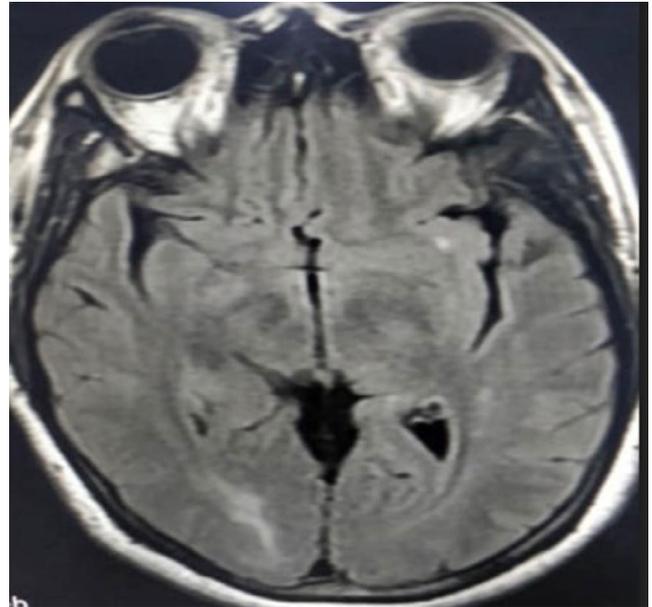


**FIGURE 1** CT brain showing left convexal frontal subarachnoid hemorrhage



**FIGURE 2** MRV showing dural venous thrombosis of left transverse and sigmoid sinus

might have played a synergistic role in pathogenesis of PRES in our case.<sup>12</sup> The concomitant development of postpartum PRES in our case might have synergistic effect on SAH as reported by Sharma et al.<sup>12</sup> The possible mechanisms explained in their study were either vasogenic or cytotoxic in nature. The pathophysiology of aneurysmal SAH in CVT



**FIGURE 3** MRI brain showing T2/FLAIR hyperintensities in occipital lobes

is not completely understood but hypothesized as venous congestion and hypertension leading to rupture of emissary cortical veins.<sup>10</sup> How can we explain the role of PRES in development SAH? Most literature on pathogenesis of PRES revolve round the vasogenic theory of cerebral arterial hyperperfusion and hypertension. One animal model study and a case report has shed some light on the findings of cerebral venous congestion or hypertension in Eclampsia-PRES.<sup>13,14</sup> Our hypothetic explanation revolves round the cerebral venous congestion or hypertension that has not gained enough scientific exploration. Our postulation of PRES and CVT as cause of SAH in this case is supported by the MRI findings which showed CVT and evidence of vasogenic edema in the occipital region.<sup>15</sup>

Our case discussion is limited by the inability to conduct thrombophilic profile. However, the final pathway in pathogenesis of SAH in this case is eclampsia-PRES and CVT rather than thrombocytopenia alone. The PIERS study group has demonstrated that although thrombocytopenia in setting of pre eclampsia is a risk factor for adverse maternal outcome including stroke, its utility is limited by low sensitivity and positive predictive value of 16% and 20%, respectively.<sup>16</sup> Thrombocytopenia and SAH likely occurred as part of disease process in eclampsia, probable sepsis, and initial cerebral vasospasm.

#### 4 | CONCLUSION

Eclampsia and postpartum onset of SAH should prompt physicians to look for possible causes and institute prompt management to avert irreversible cerebrovascular sequelae.

## ACKNOWLEDGMENT

Published with written consent of the patient.

## CONFLICT OF INTEREST

None declared.

## AUTHOR CONTRIBUTION

ST was involved in conception and design, acquisition of data, analysis and interpretation of data, revising it critically for important intellectual content, final approval of the version to be published, and agreed to be accountable for all aspects of the work. SY was involved in conception and design, acquisition of data, revising it critically for important intellectual content, final approval of the version to be published, and agreed to be accountable for all aspects of the work. DP was involved in conception and design, acquisition of data, interpretation of data, revising it critically for important intellectual content, final approval of the version to be published, and agreed to be accountable for all aspects of the work.

## ETHICAL APPROVAL

Ethical approval is not needed for case report in de-identified patients.

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