



Case Report

Cerebral venous thrombosis complicating severe preeclampsia in the postpartum period: a diagnostic challenge



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Abstract A 26 year old primigravida with preeclampsia was diagnosed with a cerebral venous thrombosis 6 days following Cesarean section. The diagnosis was initially challenging due to the patient's history of migraines, the preeclampsia, multiple attempts at spinal anesthesia for Cesarean section, and a dural puncture while performing epidural blood patch.

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1. Introduction

Cerebral venous thrombosis (CVT) is a rare cause of stroke in the general population, occurring when a blood clot forms in any of the cerebral veins [1]. Cerebral venous thrombosis leads to cerebral venous obstruction, which is responsible for the clinical findings associated with this condition [1,2]. The symptoms and clinical course are highly variable and may include headache, nausea, mental status change, seizures, and focal neurologic deficits [1,3,4]. Cerebral venous thrombosis may occur at any time, but women are particularly vulnerable during the third trimester and the postpartum period because of the hypercoagulable

state that accompanies pregnancy [2]. Diagnosis of CVT may be challenging because the symptoms are common and nonspecific, especially in the context of the peripartum state.

2. Case report

A 26 year old, 103.5 kg (body mass index 42.6 kg/m²), 156 cm, Hispanic primigravida woman at 35 weeks, 6 days' gestation, who had a history of migraines and obesity, presented to the obstetric clinic complaining of headache and "spotted" vision, as well as epigastric and bilateral flank pain beginning a few hours earlier. Her pregnancy had been uneventful until 4 days earlier, when she was seen for leg pain due to swelling. She had gained excessive weight over the last two weeks, which was attributed to the dependent edema. A pregnancy-induced hypertension panel and a 24-hour urine-protein analysis were ordered.

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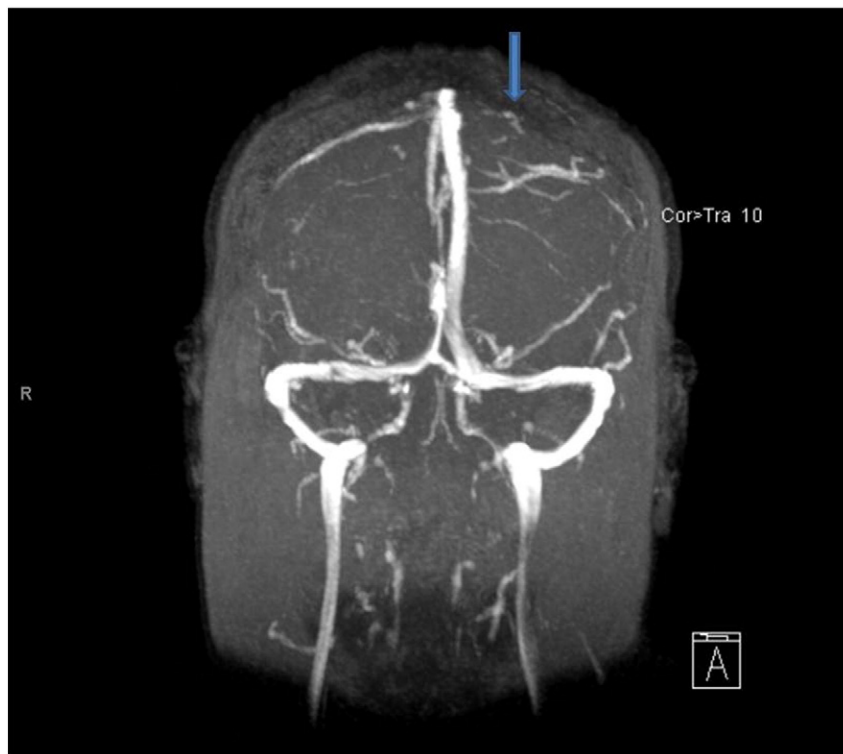


Fig. 1 Magnetic resonance image demonstrating a left superficial cortical vein thrombosis (arrow).

On admission, physical examination showed a blood pressure (BP) of 193/104 mmHg, heart rate of 74 beats per minute (bpm), temperature of 37.5° C, and 2+ pitting edema in all extremities. Total weight gain during pregnancy was 21.7 kg. Fetal heart tracing was category I. Other parts of the physical examination were unremarkable. Laboratory results demonstrated an elevated uric acid level (8.2 mg/dL) and alanine aminotransferase (ALT; 77 U/L); 24-hour urine protein was greater than 6 grams. Treatment with magnesium, labetalol, and hydralazine was initiated and labor induction was started. Three hours after induction the fetus developed a nonreassuring heart tracing; the obstetric team opted for Cesarean delivery. Spinal anesthesia was attempted three times, followed by several attempts using a combined spinal-epidural technique, all without success. The case proceeded uneventfully during general anesthesia.

On postoperative day (POD) 1, the patient complained of a severe occipital headache that was exacerbated by ambulation and improved when supine. This headache was unlike her previous migraine symptoms. She denied nausea, vomiting, and visual changes. On examination, she had neither focal neurological deficits nor new physical findings. She was treated symptomatically with acetaminophen, sumatriptan, ibuprofen, caffeinated drinks, hydration, and bed rest for presumed postdural puncture headache (PDPH), though the previous attempts at neuraxial anesthesia never yielded cerebrospinal fluid (CSF). Her headache did not improve and she agreed to an epidural blood patch on POD 3.

In the course of performing the blood patch, a dural puncture occurred. The Tuohy needle was retracted until CSF return stopped; an epidural catheter was inserted 3 cm into the epidural space. Aspiration was negative for CSF, a test dose was negative, and 12 mL of autologous blood was injected via the catheter. The patient reported immediate improvement of her headache. She was discharged home on POD 4 with a new prescription for nifedipine.

On the following morning, she returned to the emergency department with headache and acute onset of right-sided facial, arm, and leg numbness and weakness. Her BP was 165/108 mmHg. She had not taken the prescribed antihypertensive. Examination demonstrated diminished strength in the right leg as well as a positive Babinski reflex. Laboratory tests were normal. Computed tomography (CT) of the head without contrast showed left parietal subarachnoid hemorrhage. She was admitted for BP management and further imaging. Magnetic resonance venography (MRV) showed a left superficial cortical vein thrombosis with associated left superior frontal-parietal lobe venous infarct (Fig. 1); intravenous heparin was initiated. She remained in the hospital for one week, until the international normalized ratio (INR) reached therapeutic values on oral warfarin.

Two weeks later, her weakness and numbness resolved but she still had mild exaggeration of deep tendon reflexes. In addition, no abnormalities were found after evaluation for thrombophilic and hypercoagulable disorders. She was instructed to continue warfarin therapy for 6 months.

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