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Intraventricular hemorrhage in a preeclamptic patient: A late and lethal maternal complication

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ABSTRACT

We report a rare case of postpartum intraventricular and intracerebral hemorrhage secondary to eclampsia in a 40-year-old multiparous woman with a history of preeclampsia. The patient underwent a term cesarean section and was discharged with antihypertensive therapy. On the 7th postpartum day, she experienced an eclamptic seizure and was admitted with confusion and elevated blood pressure. Cranial computed tomography revealed multiple parenchymal hematomas in both hemispheres, with the largest measuring 45 mm on the left, and intraventricular hemorrhage. Despite intensive care management, including intubation, mannitol therapy, levetiracetam administration, and external ventricular drainage placement, the patient developed signs of brain death on the 10th postpartum day.

Regular blood pressure monitoring during both antenatal and postnatal follow-up are essential for early detection and management of preeclampsia. Increment in the blood pressure should be considered even after discharge. Additionally, we must keep in mind that preeclampsia can lead to rupture of vessels at a lower blood pressure because of the altered cerebral autoregulation.

Keywords: intraventricular hemorrhage, pregnancy, preeclampsia.

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Introduction

Pregnancy is a physiological state associated with an increased risk of both ischemic and hemorrhagic stroke. This risk becomes particularly evident during the third trimester and the postpartum period. It has been reported in the literature that stroke accounts for more than 12% of all maternal deaths. Among patients who experience pregnancy-related stroke, 25–45% have concomitant preeclampsia or eclampsia [1].

The risk of ischemic stroke associated with preeclampsia is not limited to pregnancy and the puerperium; it may also persist beyond gestation. According to data from the Stroke Prevention in Young Women Study**,** women with a history of preeclampsia were found to have a 60% higher likelihood of experiencing ischemic stroke outside of pregnancy [2].

Cerebral autoregulation is typically preserved within a mean arterial pressure range of 60–150 mmHg. However, this mechanism can be impaired in pregnancy due to chronic hyperventilation. In association with preeclampsia and eclampsia, increased cerebral

perfusion pressures have been reported to disrupt autoregulation, leading to barotrauma and vascular injury (3).Additionally, preeclampsia promotes hemoconcentration due to fluid extravasation into the third space, while activation of the coagulation system and microthrombus formation contribute to hypoperfusion and further increase the risk of stroke [1].

Here, we present a case of intraventricular hemorrhage occurring on the 7th postpartum day secondary to eclampsia in a patient with preeclampsia.

Case Report

A 40-year-old woman, gravida 3 parity 3, with a history of three previous cesarean deliveries, underwent a term cesarean section at an outside center due to preeclampsia. Postoperatively, she had no active complaints and remained normotensive. She was discharged on the second postoperative day with blood pressure monitoring instructions and antihypertensive treatment.

On the 7th postpartum day, the patient experienced an eclamptic seizure. An emergency call was placed, and she was transferred to the hospital by ambulance. During transportation, her vital signs were: arterial blood pressure 160/90 mmHg, heart rate 86 bpm, body temperature 36.7 °C, respiratory rate 15/min. She was confused, with a Glasgow Coma Scale (GCS) score of 11, and was handed over to the obstetrics emergency team.

At the bedside initial examination, her arterial blood pressure was 120/60 mmHg, heart rate 98 bpm, body temperature 36.1 °C, and capillary glucose 174 mg/dL. Her mental status was still confused. Suprapubic ultrasonography revealed a uterus of postpartum size with a thin endometrium; no retained placenta, intra-abdominal, or pouch of Douglas free fluid was detected. Abdominal examination was soft without defense or rebound. The Pfannenstiel incision site was intact without discharge, erythema, or signs of infection. Vaginal examination revealed no active bleeding. Initial laboratory results were as follows: hemoglobin 12.7 g/dL, platelets 567 × 10^3/μL, WBC 4.18 × 10^6/μL, fibrinogen 5.04 g/L, AST/ALT 19/19 U/L, creatinine 0.51 mg/dL.

The patient was admitted to the intensive care unit, and the anesthesia and reanimation team was informed. During evaluation, GCS was 4, arterial blood pressure 240/110 mmHg, pulse 108 bpm, oxygen saturation 95% with 6 L/min oxygen by reservoir mask. Arterial blood gas analysis revealed: pH 7.42, pCO_2 41.5 mmHg, pO_2 278 mmHg, lactate 2.2 mmol/L, HCO_3 26.6 mmol/L. She was intubated at the bedside.

Neurological examination revealed a confused and disoriented patient with bilateral positive light reflexes. No eye-opening to painful stimuli was observed, and only mild withdrawal was noted in the right extremity. A non-contrast cranial computed tomography (CT) was performed under perlinganit infusion. CT revealed multiple parenchymal hematomas in both cerebral hemispheres, the largest being 45 mm in diameter in the left hemisphere. Intraventricular hemorrhage was present with mild compression in the left lateral ventricle. Bilateral white matter hypodensities were observed. No midline shift was noted. (Figure 1).

A follow-up non-contrast cranial CT demonstrated extensive bilateral intracerebral hemorrhages, with EVD catheters extending into both frontal lobes. Hemorrhagic sedimentation was noted within cerebrospinal fluid (CSF) spaces, accompanied by bilateral subarachnoid hemorrhage. (Figure 2).

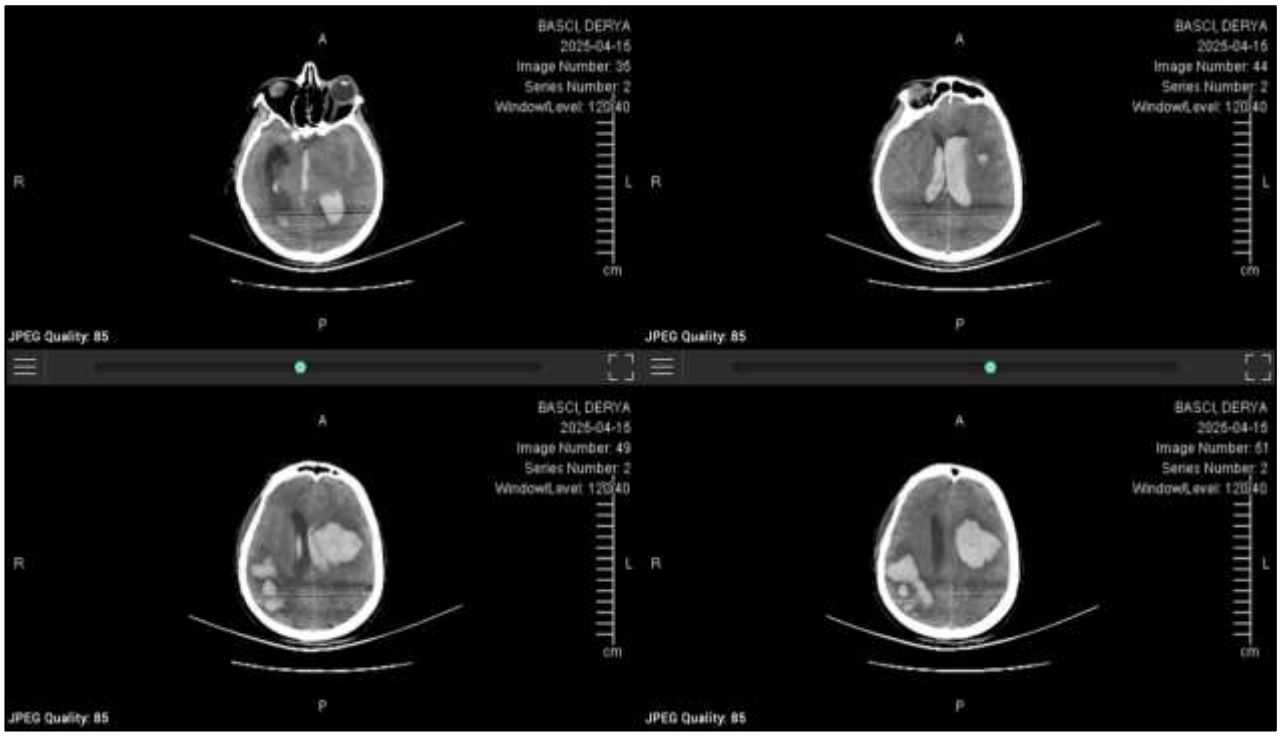

Figure 1. Non-contrast cranial CT demonstrating bilateral parenchymal hematomas, the largest measuring 45 mm in the left hemisphere.

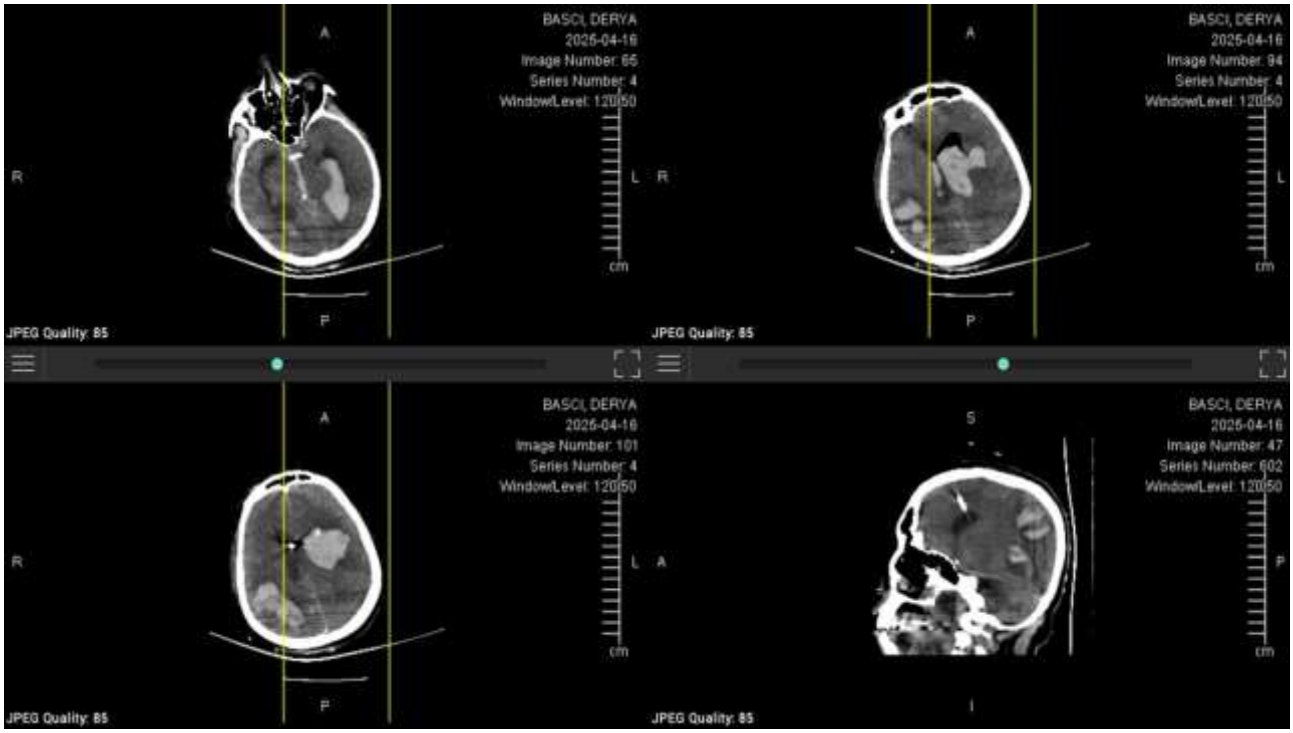

Figure 1. Control non-contrast cranial CT showing external ventricular drainage catheters and extensive bilateral hemorrhage with subarachnoid involvement.

Despite interventions, on the first ICU day, after discontinuation of sedation, neurological evaluation revealed absent pupillary light reflexes, no spontaneous respiration, and no gag reflex. Hemodynamic status remained unstable despite inotropic support. Following normalization of temperature and blood gas parameters, an apnea test was performed as part of the brain death protocol. Initial arterial blood gas showed pCO_2 43.6 mmHg, which increased to 65.3 mmHg at the 10th minute without any spontaneous respiratory effort, confirming a positive apnea test.

On the second ICU day, neurological examination demonstrated a GCS score of 3, non-reactive mid-dilated pupils, absent corneal reflexes, and absent oculocephalic and oculovestibular reflexes. CT angiography was performed, revealing no contrast opacification in the supraclinoid segments of both internal carotid arteries, consistent with cerebral circulatory arrest.

Based on clinical and radiological findings, the patient was declared brain dead on the 10th postpartum day (ICU day 2).

Discussion

Intracranial hemorrhage (ICH) is a rare but devastating complication of hypertensive disorders of pregnancy and remains a leading cause of maternal morbidity and mortality. Large population-based studies show that the risk of ICH increases during late pregnancy and peaks during the early postpartum period, with a more than nine-fold rise in the first 12 weeks after delivery compared with non-pregnant intervals [4]. Our case, occurring on postpartum day 7, aligns precisely with this window of heightened susceptibility.

The mechanisms linking preeclampsia/eclampsia and ICH are multifactorial. Preeclampsia is characterized by endothelial dysfunction, loss of cerebrovascular autoregulation, and reduced integrity of the blood–brain barrier. These alterations make cerebral vessels more susceptible to pressure-induced rupture, even at blood pressure levels that may not be considered severely elevated in non-pregnant women [5].

Literature consistently emphasizes that in preeclampsia/eclampsia, vascular injury may develop despite moderate hypertension, due to endothelial activation, microthrombi formation, and hyperperfusion injury—all of which predispose to parenchymal and intraventricular bleeding [5].

Our patient's sudden neurological deterioration, progression to coma, and subsequent imaging showing multiple bilateral parenchymal hematomas with intraventricular extension indicate a fulminant cerebrovascular event. Similar cases in the literature describe ICH presenting with seizures, altered mental status, and rapid neurological decline, underscoring the difficulty in early recognition and timely intervention [6].

In contrast to cases managed conservatively with good recovery outcomes, as reported by Mohamed et al., the extensive bilateral involvement and mass effect in our patient portended a poor prognosis from the outset [6]. While rapid control of blood pressure, seizure management (typically magnesium sulfate), and neurosurgical procedures such as external ventricular drainage are standard components of acute management, mortality remains high. Numerous studies highlight that intracranial hemorrhage (ICH) is one of the leading causes of maternal death in hypertensive disorders of pregnancy, and that preeclampsia increases the risk of stroke by up to nine times compared to pregnancies with normal blood pressure. [4]. Even in tertiary care settings, outcomes depend heavily on the hemorrhage location, size, availability of timely neurosurgical

intervention, and reversibility of cerebral edema.

This case highlights several critical clinical insights. The risk of severe neurological complications clearly persists after discharge, underscoring the importance of continued postpartum blood pressure surveillance and patient education. Any new neurological symptom in women with preeclampsia should prompt urgent neuroimaging, as headache, confusion, or seizures may signal catastrophic cerebrovascular injury rather than isolated eclamptic activity. Moreover, impaired vascular integrity in preeclampsia allows hemorrhage to occur at relatively modest blood pressure levels, reinforcing the need for aggressive blood pressure control and rapid escalation of care. Finally, the occurrence of extensive intracranial hemorrhage in the absence of coagulopathy emphasizes that auto regulatory failure, rather than hematologic abnormality, is the dominant mechanism in such cases.

Despite optimal management—including intubation, antihypertensive therapy, osmotherapy, antiepileptic's, and EVD placement—our patient progressed to brain death, reflecting the severity of bilateral hemorrhagic injury. Similar fatal courses have been described in the literature, particularly in cases with extensive parenchymal involvement and intraventricular extension [7].

Conclusion

This case underscores the ongoing vulnerability of women with hypertensive disorders of pregnancy during the early postpartum period, emphasizing the need for extended blood pressure surveillance, rapid neuroimaging in the presence of any neurological symptoms, and coordinated multidisciplinary care. Patient-centered strategies that improve adherence to postpartum monitoring are particularly critical, as many catastrophic events occur after hospital discharge. As postpartum eclampsia and its cerebrovascular sequelae continue to represent preventable causes of maternal mortality worldwide, early identification and aggressive management of preeclampsia remain essential to reducing the risk of severe neurological complications.

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