Annals of Clinical and Medical Case Reports[R] ISSN 2639-8109 | Volume 17

Case Report Open Access

Postpartum Intracranial Hemorrhage in Hypertensive Disorders of Pregnancy: A Three-Case Series

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Received: 19 Oct 2026
Accepted: 09 Nov 2026
Published: 13 Nov 2026
J Short Name: ACMCR

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Citation:

Nikita Krishna, Postpartum Intracranial Hemorrhage in Hypertensive Disorders of Pregnancy: A Three-Case Series. Ann Clin Med Case Rep® 2026; V17(1): 1-3

Keywords:

Postpartum; Intracranial Hemorrhage; Pre-Eclampsia; HELLP Syndrome; Intraventricular Hemorrhage; Case Series

1. Abstract

1.1. Background

Intracranial hemorrhage during the postpartum period is rare and may be associated with hypertensive disorders of pregnancy. This report describes three fatal cases and discusses their clinical context.

1.2. Case Presentation

Three women aged 24–27 years with gestational hypertension or pre-eclampsia developed intracranial hemorrhage after delivery. Two had extensive intraventricular hemorrhage; one had a right parietal intraparenchymal hemorrhage with intraventricular extension and midline shift. One patient had severe thrombocytopenia and an elevated international normalized ratio in the context of HELLP syndrome. All three patients died following neurological deterioration and systemic complications.

1.3. Conclusion

The cases emphasize the need for urgent investigation of new postpartum neurological symptoms and coordinated obstetric, neurological, and critical care. As a small case series, these observations cannot establish causation or estimate risk.

2. Introduction

Intracranial hemorrhage is an uncommon but life-threatening neurological complication of pregnancy and the puerperium. Hypertensive disorders of pregnancy, including pre-eclampsia and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count), are important clinical contexts in which cerebrovascular complications may occur. Proposed mechanisms include endothelial dysfunction, altered cerebral autoregulation, blood–brain barrier disruption, and, in HELLP syndrome, thrombocytopenia and coagulation abnormalities. These mechanisms are biologically plausible but do not establish the cause of hemorrhage in an individual patient. We describe three fatal cases of postpartum intracranial hemorrhage in women with hypertensive disorders of pregnancy, highlighting the varied radiological patterns and the need for prompt evaluation of neurological deterioration.

3. Case 1

A 25-year-old woman, gravida 2 para 1, at 36 weeks and 2 days of gestation presented with gestational hypertension and preterm labor. Her blood pressure was 171/86 mmHg. She received a single 20-mg intravenous dose of labetalol; 10 minutes later, her blood pressure was 140/90 mmHg. Urinalysis showed no albuminuria. Abdominal examination showed a fundal height of 34 weeks, cephalic presentation, average amniotic fluid volume, and mild uterine contractions. Vaginal examination showed 5-cm cervical dilatation and 80% effacement. Artificial rupture of membranes produced thin meconium-stained liquor. Labor progressed, and she delivered a 2.1-kg female infant within seven hours of labor onset; Apgar scores were 8 and 9. Evaluation for pre-eclampsia was reported as unremarkable. Oral amlodipine 5 mg once daily was initiated, and subsequent blood-pressure recordings were stable.

On postpartum day 3, she developed marked lethargy and refused oral intake, followed several hours later by a generalized tonic–clonic seizure. Vital signs were reported as normal. Complete blood count, coagulation profile, electrocardiogram, and arterial blood gas analysis were reported as normal. She was intubated, transferred to the intensive care unit, and started on broad-spectrum antibiotics. Non-contrast computed tomography

(CT) of the head showed extensive intraventricular hemorrhage involving the lateral and third ventricles, with extension into the quadrigeminal cistern and marked ventricular dilatation. The cerebral parenchyma was unremarkable, with no midline shift or extra-axial collection. A diagnosis of gestational hypertension complicated by acute intraventricular hemorrhage was made.

By postpartum day 5, the total leukocyte count had increased to 29,300/mm³, and renal function had deteriorated, with serum creatinine reaching 4.4 mg/dL. Progressive hypoxemia required tracheostomy. On postpartum day 8, she sustained a cardiac arrest and died despite resuscitation.

4. Case 2

A 24-year-old unbooked and unregistered woman, gravida 3 para 2, presented with fever, lower abdominal pain, breathlessness, and absence of fetal movements for one day. She had a history of pregnancy-induced hypertension in both previous pregnancies; records and further details were unavailable. No history of eclampsia was reported. On examination, her temperature was 95.5°F, blood pressure 160/100 mmHg, pulse 100/min, and respiratory rate 44/min. Urine albumin was 1+. Air entry was reduced on the right. Fundal height corresponded to 26 weeks; the uterus was irritable but not tense or tender. Vaginal examination showed a 3-cm dilated cervix, 40–50% effacement, and bulging membranes. Investigations showed hemoglobin 13.4 g/dL, total leukocyte count 11,300/mm³, platelet count 16,000/mm³, lactate dehydrogenase 1,198 U/L, and international normalized ratio 1.84; D-dimer was negative. Electrocardiography showed right-axis deviation with a strain pattern. Ultrasonography showed intrauterine fetal demise with oligohydramnios, mild maternal hepatomegaly, and moderate ascites. Severe pre-eclampsia with HELLP syndrome was diagnosed. She received four units of fresh frozen plasma and 12 units of platelets. She subsequently became drowsy and developed right basal crepitations; intravenous furosemide 40 mg was administered, and she was transferred to the intensive care unit. She then delivered a preterm stillborn fetus spontaneously.

After delivery, she developed high-grade fever and became comatose. Non-contrast CT of the head showed a 4.1 × 2.1 × 3.9-cm right parietal intraparenchymal hemorrhage with extension into the ipsilateral lateral, third, and fourth ventricles, cerebral edema, and 6.6-mm leftward midline shift. Despite intensive resuscitative management and maximal inotropic support, she remained in refractory shock with persistent desaturation. She sustained a cardiac arrest on hospital day 2 and died despite resuscitation.

5. Case 3

A 27-year-old primigravida at 35 weeks and 2 days of gestation presented with abdominal pain and headache of one day's duration. Her blood pressure was 172/108 mmHg, and urine albumin was 2+. She received intravenous labetalol 20 mg followed by 40 mg; blood pressure subsequently decreased to 148/96 mmHg. Fundal height was 32–34 weeks, the fetus was in breech presentation, and amniotic fluid volume was average. Emergency cesarean delivery was performed for severe pre-eclampsia with impending complications. The intraoperative course was reported as uneventful, with blood pressure 148/94 mmHg. A 1.9-kg male infant was delivered; an Apgar score of 8 was reported. Postoperatively, she was started on amlodipine 10 mg and telmisartan 40 mg.

On postoperative day 3, she developed marked lethargy and refused oral intake, followed several hours later by a generalized tonic–clonic seizure. Her blood pressure was 118/68 mmHg, pulse 98/min, respiratory rate 24–26/min, and oxygen saturation 88%. She was intubated, transferred to the intensive care unit, and started on broad-spectrum antibiotics. Non-contrast CT of the head showed extensive intraventricular hemorrhage involving the lateral and third ventricles, with midline shift. The case was diagnosed as severe pre-eclampsia with HELLP syndrome complicated by acute intraventricular hemorrhage. By postoperative day 7, the total leukocyte count had increased to 33,350/mm³, and serum creatinine had risen to 6.4 mg/dL. She sustained a cardiac arrest on postoperative day 8 and died despite resuscitation.

6. Review of the Literature

Pregnancy-associated intracerebral hemorrhage (ICH) is rare, and estimates vary according to case definitions, data sources, and whether the denominator is deliveries or person-time. In a US Nationwide Inpatient Sample analysis of 423 pregnancy-related ICH admissions from 1993 to 2002, Bateman et al. estimated 6.1 cases per 100,000 deliveries and an in-hospital mortality of 20.3%; the study also reported that the excess risk was concentrated in the postpartum period. These estimates are based on administrative data and should not be directly generalized to other health systems or populations [1].

In a cohort-crossover study of 3,314,945 pregnancies, Meeks et al. found that primary ICH rates were elevated in the third trimester and during the first 12 postpartum weeks compared with the same women during a later unexposed period. The postpartum rate ratio was 9.15 (95% confidence interval [CI], 5.16–16.23); no significant elevation was observed from 12 to 24 weeks postpartum. These findings support continued clinical vigilance beyond hospital discharge, although they do not imply that ICH is common in absolute terms [2].

Hypertensive disorders of pregnancy are important risk contexts for maternal cerebrovascular disease. Preeclampsia has been associated with endothelial dysfunction, altered cerebral blood-flow autoregulation, and blood–brain barrier disruption; these changes may contribute to cerebral edema, seizures, and stroke. The mechanisms are complex and remain incompletely understood, and they cannot be inferred from clinical presentation alone. HELLP syndrome may add thrombocytopenia and other hemostatic abnormalities, increasing concern for bleeding, but

a causal contribution in an individual case requires supporting laboratory and diagnostic evidence [3,4].

7. Discussion

This case series describes three young women with hypertensive disorders of pregnancy who developed fatal intracranial hemorrhage during the early postpartum period. Two cases were reported as predominantly intraventricular hemorrhage, while the third involved a right parietal intraparenchymal hematoma with intraventricular extension, edema, and midline shift. All three patients experienced neurological deterioration after delivery and died following progressive systemic complications. These observations illustrate severity, but a three-case series cannot estimate frequency, compare risks, or establish causation.

The timing is consistent with population evidence that pregnancy-associated ICH risk is elevated in late pregnancy and the early puerperium [1,2]. Postpartum neurological symptoms can arise despite previously stable blood pressure or after delivery; therefore, new lethargy, persistent headache, altered mental status, seizures, focal deficits, or visual symptoms warrant urgent assessment. In these cases, lethargy preceded seizures in two patients, while the third became comatose after delivery. Once acute intracranial hemorrhage is suspected, non-contrast head CT provides rapid diagnosis. CT angiography or other vascular imaging, and MRI with appropriate venous imaging where clinically feasible, may help evaluate structural vascular lesions, cerebral venous thrombosis, posterior reversible encephalopathy syndrome, or other causes. The choice and timing of imaging should be individualized to the clinical condition and local resources [4,5].

The reported hypertensive disorders and, in one patient, profound thrombocytopenia with an elevated INR are clinically relevant. However, the available records do not establish the precise mechanism of hemorrhage in any case. Severe hypertension may contribute to cerebrovascular injury, while pre-eclampsia-related endothelial and autoregulatory dysfunction may increase cerebral vulnerability; thrombocytopenia and coagulopathy may compound bleeding risk. These are plausible pathways rather than proven explanations for the events described. Acute management requires coordinated obstetric, neurological, neurocritical-care, hematology, and neurosurgical input as indicated. Priorities include stabilization, controlled management of severe blood pressure, evaluation and correction of clinically significant coagulopathy, management of raised intracranial pressure and hydrocephalus, and timely neurosurgical consultation. Current maternal-stroke guidance emphasizes that pregnancy or postpartum status should not delay urgent evidence-based stroke evaluation and treatment [4,5].

8. Limitations

This case series is limited by its small size, lack of a comparator group, and reliance on the information available in the clinical records. It cannot establish causality or estimate incidence. Detailed serial blood-pressure and laboratory data, vascular imaging, MRI and venography, intracranial-pressure monitoring, neurosurgical assessments, and complete intensive-care timelines were not available. These gaps limit etiologic classification and interpretation of potentially modifiable factors.

9. Conclusion

Postpartum intracranial hemorrhage is an uncommon but potentially catastrophic complication in patients with hypertensive disorders of pregnancy. This three-case series underscores the need to recognize acute neurological change promptly, obtain urgent brain imaging, and coordinate multidisciplinary care. Population studies support vigilance during the early postpartum period, while the present cases cannot determine mechanism or treatment effectiveness. Strengthening postpartum blood-pressure follow-up and ensuring rapid access to neurological assessment may support earlier recognition; further studies are needed to define optimal surveillance and management strategies.

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