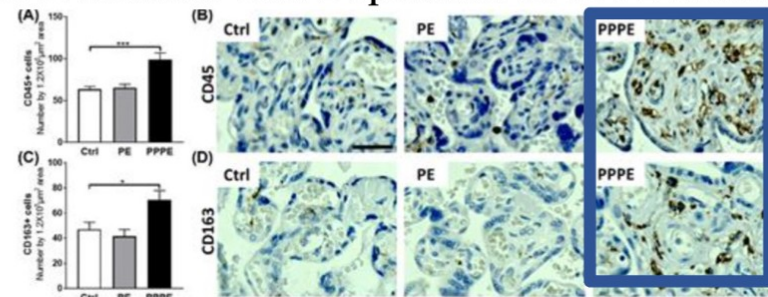


Introduction

- The second most common direct cause of maternal mortality is hypertension (HTN) related diseases.¹
- A study conducted in 2022 showed planned delivery for women with late term onset preeclampsia (PEC) was safer than expectant management and did not greatly affect neonatal outcomes.²
- There are extensive risk factors for delayed onset PEC including: elevated body mass index (BMI), advanced maternal age, obesity, and non-Hispanic black race.³
- The most common presenting symptom of postpartum preeclampsia (PPPE) is postpartum headache (HA).³⁻⁵
- In a study conducted by Wen et al., only 1 in 687 women without previous history of HTN were readmitted to the hospital within 60 days of childbirth.⁶
- The pathogenesis of PEC remains unclear. Some of the oldest hypotheses suggested abnormal spiral artery remodeling and placentation were to blame. Although this theory is from the early 1900s, it is still referred to as the likely etiology.⁷
- Newer studies suggested an exaggerated inflammatory response.
- For example, as illustrated in Figure 1, Brien et al. found increased CD45+ immune cell deposition in the placentas of women who developed PPPE.
- There was also found to be an inflammatory molecular pattern, high mobility group box 1 (HMGB1), which was identified to be elevated in placentas and maternal blood in women who developed preeclampsia postpartum, compared to intrapartum.⁸

Figure 1: Immune Cell Deposition in PPPE Placenta



Adapted from [Biology of Reproduction]^F

Criteria for Preeclampsia

- New onset HTN after 20 weeks gestation.
- Blood pressure (BP) ≥ 140 systolic and/or ≥ 90 diastolic on two occasions in addition to:
- Presence of ≥ 1 of the following: proteinuria, acute kidney injury, pulmonary edema, neurologic complications, decreased platelets, elevated transaminases, or uteroplacental dysfunction.
- Severe HTN is classified as a systolic BP ≥ 160 and/or diastolic ≥ 110 .⁹

Case Description

History

- A 23-year-old gravida 1 para 0 black female with no significant past medical history presented to the hospital for induction of labor in the setting of untreated gestational diabetes mellitus (GDM).
- She had been normotensive throughout her life. Her pre-pregnancy BMI was 34.7 kg/m².
- Obstetrics history: G1-Besides GDM, she had no other complications during pregnancy. No previous pregnancies, abortions, no abnormal pap smears, or personal/family history of gynecological malignancies.
- Social history: good support system, stable housing, no use of tobacco, alcohol, marijuana, or illicit drugs
- She received an epidural during the active stage of labor and gave birth vaginally at 39 weeks plus 2 days gestation to a healthy baby girl weighing 3490g. Apgar scores were 8 at 1 minute and 9 at 5 minutes. She lost 200cc of blood.
- Day one postpartum she complained of a positional headache and was treated with bedrest and butalbital/acetaminophen/caffeine after failing conservative management. She was discharged home with a scheduled outpatient post partum visit.
- 48 hours later she returned to the hospital with an 8/10 headache radiating from the back to front of her head.
- She had one episode of white spots appearing in her vision, but stated she did not have any diplopia, episodes of vision loss, dizziness, tingling in extremities, abdominal pain, nausea, vomiting, or edema in her face or hands.

Physical Exam Upon Readmission

BP: 159/78 mmHg Pulse: 55 Respiratory rate: 14 breaths/minute
Pulse oximetry: 98% Temperature: 97.4 °F BMI at time of delivery: 37kg/m²
General: no acute distress, appears uncomfortable
Skin: pink warm and dry, capillary refill <2 seconds, no tenting present, trace lower extremity edema, no rashes or ecchymosis
Eyes: pupils equal, round, and reactive to light, no nystagmus
Cardiopulmonary exam: unremarkable
Neuro: no focal neural deficits, headache unchanged in supine position, normal reflexes, no clonus
Abdomen: soft, not tender or distended, no pain with right upper quadrant palpation, no organomegaly noted
Uterus: firm, nontender, palpated 2cm below umbilicus, appropriate amount of lochia

Diagnostic Testing

- CBC: remarkable for WBC 16,000/microliter, and hemoglobin of 8.3g/dL.
- Chemistry: within normal limits (WNL)
- Liver function tests (in units/L): AST-**112**, ALT-**194**, and alkaline phosphatase-82
- Lactate dehydrogenase: **299** IU/L
- Urinalysis: no proteinuria
- Creatinine: 0.7 \rightarrow **0.9mg/dL**
- Protein-creatinine ratio: WNL

Differential Diagnosis



Neurologic: Spinal HA, tension HA, migraine, cluster HA



Pregnancy-related: Preeclampsia/eclampsia, postpartum hemorrhage, HELLP syndrome



Metabolic: dehydration, hyperosmolar state, electrolyte abnormalities

Patient Management/Outcome

- She was given two doses of 20mg intravenous (IV) labetalol and one dose of 40mg IV labetalol.
- Her BP starting to normalize and nifedipine 30mg extended release (ER) was added for more control.
- The patient was also placed on 24 hours of an IV magnesium drip for seizure prophylaxis, which she tolerated well. Her lower extremity reflexes remained intact throughout treatment course.
- Her headache improved significantly but did not completely resolve so the anesthesiologist placed a blood patch prior to discharge, which abated the headache.
- She followed up outpatient two weeks later and was recovering uneventfully, with blood pressure within normal limits.
- The patient remained on nifedipine XR for three weeks after discharge.
- At her six-week post partum visit her blood pressure was 124/83mmHg, without medication.

Table 1: Blood Pressures

Time (hours)	1310	1340	1425	1550	1610	1620
BP (mmHg)	159/78	171/91	188/10	173/98	159/75	139/66

Discussion

- It is recommended to initiate aspirin therapy prior to 16 weeks gestation in women with high risk for preeclampsia.^{4,9}
- A case control study published in 2019 showed that women who delivered via cesarean section had the highest odds ratio for developing postpartum preeclampsia.³
- Interesting components of this case presentation included new onset HTN three days after vaginal delivery in the setting of bradycardia, and young maternal age.
- A retrospective cohort study by Ravid et al. showed a link between decreased heart rate in post partum preeclampsia, compared to controls, and suggested this was potentially due to baroreceptor response.¹⁰
- Another interesting component of this admission, in conjunction with the research from Brien et al., is the presence of leukocytosis without infectious etiology, which could support the hypothesized immunological etiology to preeclampsia.⁸

Conclusion

- Consider common risk factors of preeclampsia when providing prenatal and antenatal care.
- Be suspicious for post partum preeclampsia in women who present with a headache, especially those with significant risk factors.
- Screen patients early in gestation to promptly identify and treat patients who would benefit for aspirin therapy for prevention.

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