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RESEARCH ARTICLE

CHALLENGES IN THE MANAGEMENT OF POST-PARTUM PREECLAMPSIA WITH DELAYED PRESENTATION

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Abstract

Hypertension in the postpartum period is common in women with prior hypertensive disorders during pregnancy but can also appear newly for the first time after childbirth. Whether postpartum preeclampsia or eclampsia is distinct from its antepartum counterpart needs to be studied in future research. Definitions wise, postpartum preeclampsia should be considered in women who develop new hypertension between 48 hours and 6 weeks postpartum. This condition is understudied, with limited guidelines for diagnosis and management. Most cases present within 7 to 10 days after delivery, most commonly with headaches. New-onset hypertension with severe features should be diagnosed as postpartum preeclampsia after excluding other neurological, cardiovascular or metabolic causes, in order to institute timely management of the condition. Unlike antepartum Pre-eclampsia where delivery of the fetus remains the cornerstone of management, the treatment in postpartum period mainly involves antihypertensive agents, magnesium, and diuretics. Postpartum preeclampsia may pose a higher risk of maternal morbidity than preeclampsia of antepartum onset and delayed onset postpartum preeclampsia is associated with higher maternal complications because of unpredictable onset, variable symptoms and late recognition by healthcare providers. The aim of our review is to increase awareness about delayed onset Preeclampsia amongst clinicians and highlight the need for better understanding of the pathophysiology of this condition and guidance to reduce postpartum maternal morbidity and mortality.

Introduction:

Pre-eclampsia (PE), a hypertensive disorder in pregnancy, affects 6-8% of pregnancies globally [1]. Postpartum pre-eclampsia occurs in 25-30% of women with PE. Early presentation **(immediate postpartum PE**) is within the first 48 hours post-delivery, and this is usually a continuum of antepartum hypertension or preeclampsia. **Delayed Postpartum PE** occurs after 48 hours up to 6 weeks postpartum. It may be a continuum of hypertensive disorder of pregnancy or an entity with new onset. It is one of the most common causes of readmission to hospital in the post-partum period after discharge with financial and social implications [2]. Delayed presentation of postpartum PE can lead to severe complications due to its unpredictable onset and variable symptomatology [3]. Women with

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symptoms usually present in the first week postpartum to Emergency department [4], Primary health centers or family physicians. It may not be recognized early as other physicians are less aware about this entity and especially, if there was no admission for severe symptoms. Hence Prolonged postpartum vigilance after hospital discharge for blood pressure and neurological symptoms should be maintained to allow timely management and reduction of maternal morbidity and mortality.

Epidemiology and Risk Factors:
The incidence of delayed postpartum PE is reported to be 0.3% to 27.5% of all pregnancies [3] and is rising due to changing maternal demographics and medical interventions in prenatal care. There is wide variation in prevalence because mild disease is not recognized by other frontline healthcare providers in the early postpartum period.

60% of new onset Postpartum PE have no Antenatal diagnosis of hypertension and although majority present in 7 to 10 days postpartum, they can present up to 3 months postpartum [5]

Risk factors include advanced maternal age, obesity, black race, antenatal hypertension, gestational diabetes mellitus, cesarean delivery, use of IV fluids in labor, epidural analgesia, use of medications like vasopressors and ergot derivatives that raise blood pressure [2,6]. Unlike antepartum PE, Parity is not a risk factor for Postpartum PE as it can occur in both primiparous and multiparous women [3]

Ethnic disparities in the incidence and severity of PE highlight the need for tailored management strategies.

Pathophysiology:
PE is characterized by systemic endothelial dysfunction, leading to vascular damage and multiorgan involvement [3].

Postpartum PE may involve different pathophysiological mechanisms than antepartum PE, potentially related to placental debris or retained trophoblastic tissue. The placental vasculopathy is same in both antenatal and Post partum PE [3].

Immunological factors, such as abnormal maternal immune responses to fetal antigens, contribute to the development of PE [7]. Further research is needed in this area.

Clinical Presentation:
Symptoms include hypertension, proteinuria, edema, and symptoms of end-organ dysfunction such as headaches, visual disturbances, epigastric pain, pulmonary edema [1] Differential diagnoses should include Cerebrovascular thrombosis, Stroke, Reversible cerebral vasoconstriction syndrome, Peripartum cardiomyopathy, Lupus exacerbation, Acute fatty liver of pregnancy and Medication adverse effects [1].

Headache is the most common presenting symptom in 60-70% women and if associated with features like thunderclap, altered mental status, focal neurological deficits, seizures or visual disturbances, there is need for cerebral imaging studies and consultation by neurologist or neurosurgeon to rule out other causes of headache [8].

Delayed presentation often complicates the diagnosis, as symptoms may mimic normal postpartum changes and vary in severity [9]

Diagnostic Challenges:
BP measurement is usually not recommended in postpartum period after discharge if there was no antenatal hypertension, so women with postpartum hypertension who do not have severe features or do not present to any healthcare providers may be missed [1]

Diagnostic evaluation of new-onset postpartum hypertension should include a detailed history and physical examination [10], with close attention to clinical volume status, and cardiopulmonary and neurologic examination based on presenting signs and symptoms.

Diagnostic criteria for postpartum PE vary but generally include hypertension (systolic ≥ 140 mmHg or diastolic ≥ 90 mmHg) measured on at least 2 occasions 4 hours apart. Proteinuria (≥ 300 mg in a 24-hour urine collection) or Edema may be a presenting feature, but is no longer necessary for diagnosis of PE.

Laboratory studies demonstrating abnormal CBC (complete blood count), Platelet count, Serum Creatinine, Liver transaminases, Urine protein/creatinine ratio further support the diagnosis.

Imaging studies include Chest X ray, Echocardiogram, CT/MRI of Brain to identify pulmonary, Cardiovascular and neurological complications of postpartum PE [3,9].

Serum electrolytes and metabolic panel should be done to rule out other causes of Hypertension in postpartum period. Brain natriuretic peptide (BNP)levels should be done in patients with symptoms of volume overload.

If there is only Hypertension with BP less than 160/110 mmHg, it is labelled as Postpartum hypertension but If BP is increased to more than 160/110mmHg or associated with proteinuria, neurological, cardiovascular symptoms or laboratory abnormalities, then the entity is labelled as Postpartum Preeclampsia [3]. The presence or absence of proteinuria does not affect the frequency of adverse outcomes.

Challenges arise due to atypical presentations and the need for heightened clinical suspicion, particularly in the absence of traditional risk factors [9]

Emerging biomarkers [7] and imaging techniques may aid in earlier detection and risk stratification [9].

Management Strategies:
Delivery remains the definitive treatment for antenatal PE, but management of postpartum PE revolves around use of Antihypertensives, magnesium sulfate and diuretics.

Immediate management involves stabilizing the patient and initiating antihypertensive therapy to prevent severe complications. Antihypertensives are started if BP >150/100 mmHg with the goal to maintain BP at 140-150/90-100 mmHg (ACOG and the RCOG/NICE guidelines) and safety for Breastfeeding is considered when choosing any antihypertensive.

Antihypertensives for acute control of BP include IV Labetalol, IV Hydralazine, Oral Nifedipine and for maintenance include Oral Labetalol, or extended release Nifedipine [11].

Magnesium sulfate infusion is recommended in women with neurologic symptoms for seizure prophylaxis. [12]

Diuretics help to lower BP and intravascular volume and are guided by BNP levels. IV or oral Furosemide is recommended routinely in women with pulmonary edema or volume overload after clinical assessment of volume status [13,17].

Close monitoring of fluid balance, renal function, and neurological status is crucial, with a focus on individualized care plans.

There is conflicting evidence about role of uterine curettage in shortening the resolution time of Hypertension and laboratory abnormalities in women with antenatal PE, so routine postpartum curettage has no role in management of postpartum PE in the absence of retained products of conception [14].

Prognosis and Complications:
Delayed diagnosis increases the risk of maternal morbidity and mortality. Maternal complications are greater with postpartum PE as compared to antenatal PE.

Immediate complications include stroke and eclampsia, emphasizing the need for close postpartum follow up and the importance of timely intervention [9]. Postpartum readmissions account for higher health care costs and disruption of parent-infant bonding [2].

Long-term maternal complications include risk of chronic hypertension and cardiovascular diseases in later life [15] which require ongoing surveillance and management.

Follow up should include home BP monitoring or in a clinic within 5-7 days, then at 6 weeks postnatal visit and annually thereafter [16]. In our institution there is a protocol for evaluation of all delivered patients in the outpatient clinic at 1-week postpartum that includes routine weight and BP measurement.

Case reports highlight instances where delayed diagnosis led to severe outcomes despite postpartum follow-up [5].

These cases underscore the importance of education and awareness among non-obstetric-healthcare providers especially emergency room physicians and utilization of multidisciplinary approach [10,18] to enhance early recognition and management as most women present in Emergency department in first few weeks postpartum with many vague symptoms [4].

Management for future pregnancies and Recurrence risk:
Normalization of BP in interpregnancy period should be ensured and if needed, antihypertensives with a safety profile in pregnancy should be used. Baseline tests for Renal, Liver function and Proteinuria should be done and Low dose Aspirin started for PE prevention.

As there is risk of recurrence of postpartum PE in future pregnancies, fluid volume status should be assessed prior to discharge and BP should be measured at 1 week postpartum at the earliest in any subsequent pregnancy [3].

Conclusions:

Postpartum PE is associated with higher maternal morbidity and mortality as compared to antenatal PE because this entity is underrecognized and the etiology is unclear.

Improved awareness amongst clinicians, standardized protocols, and patient education are essential in reducing the impact of delayed postpartum PE on maternal health outcomes.

Home monitoring of BP in first week postpartum or early scheduling of first postpartum visit aids recognition of this condition and prompt management.

Further research is needed to enhance diagnostic accuracy, optimize management strategies, and explore novel therapies targeting the underlying pathophysiology.

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