Late Onset Preeclampsia: A postpartum surprise with serious consequences: A case report

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INTRODUCTION

- Preeclampsia is a hypertensive disorder of pregnancy, it impacts about 2 to 8% of all pregnancies, and is a major cause of maternal morbidity and mortality.
- Late onset preeclampsia (LOP) is a new onset of hypertension and proteinuria after 34 weeks of gestational age.
- It is difficult to predict as biomarker predictors of preeclampsia are more successful for early than late onset.
- Also, early onset preeclampsia (EOP) is associated with growth restricted infants and it is not so much with late onset preeclampsia.

CASE

- A 23-year-old woman with a history of asthma, morbid obesity, and hypoventilation syndrome presented to the emergency room with shortness of breath.
- She is 6 weeks postpartum and has been treated for ARDS, influenza A, and bacterial pneumonia.
- In the ER, the patient's blood pressure was high, and she had CXR showed pulmonary edema.
- She was initially admitted for hypertensive emergency, started on nitroglycerin drip and was admitted to ICU.
- Upon further questioning her mother revealed that the patient had high BP during her pregnancy and was given dexamethasone for fetal lung maturation.
- She was treated with MgSO4. Her condition improved, she was tapered off of the nitroglycerin and transitioned to labetalol.
- She was also taken off of oxygen. She was discharged home to follow up with her obstetrician.
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DISCUSSION

- The cause of LOP is ‘intrinsic’ to the growing and ageing placenta, restricting intervillous perfusion.
- This case highlights the importance of asking about pregnancy and preeclampsia in all women of child-bearing age, especially in women with pre-existing medical conditions or complex medical histories, as symptoms may be attributed to their pre-existing conditions, further complicating the diagnosis.
- According to one study, maternal mortality is high in LOP compared to EOP. This also highlights the importance of follow-up care for patients, as timely intervention can lead to complete recovery.
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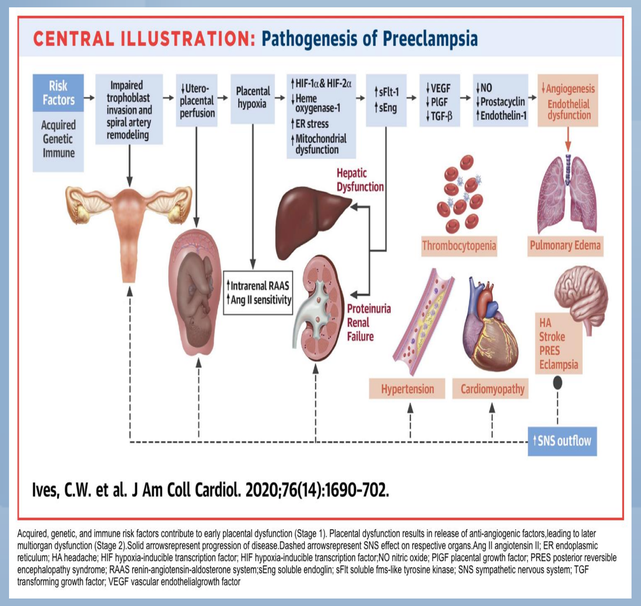
PATHOGENESIS



Acquired, genetic, and immune risk factors contribute to early placental dysfunction (Stage 1). Placental dysfunction results in release of anti-angiogenic factors,leading to later multiorgan dysfunction (Stage 2).Solid arrowsrepresent progression of disease.Dashed arrowsrepresent SNS effect on respective organs.Ang II angiotensin II; ER endoplasmic reticulum; HA headache; HIF hypoxia-inducible transcription factor; HIF hypoxia-inducible transcription factor;NO nitric oxide; PlGF placental growth factor; PRES posterior reversible encephalopathy syndrome; RAAS renin-angiotensin-aldosterone system;sEng soluble endoglin; sFlt soluble fms-like tyrosine kinase; SNS sympathetic nervous system; TGF transforming growth factor; VEGF vascular endothelialgrowth factor

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