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Unusual Case of Acute Pulmonary Edema as a Manifestation of Postpartum Preeclampsia: A Case Report

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Abstract **Background:** Hypertensive disease of pregnancy remains a leading cause of maternal morbidity and mortality. Four subcategories of hypertensive disorders of pregnancy are recognized by the international society for the study of hypertension in pregnancy: chronic hypertension; gestational hypertension, pre-eclampsia, & white coat hypertension. Pre-eclampsia is defined as systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 on two occasions at least 4 hours apart after 20 weeks of gestation with accompanying signs of end organ damage in a woman with previously normal blood pressure. The majority of cases present ante-partum or immediately post-partum. I review the case of a woman diagnosed with pre-eclampsia at 2 weeks post-partum. **Case presentation:** A 38-year-old woman presented with increasing dyspnea, orthopnea, intermittent palpitations, and central chest pain 2 weeks after an uncomplicated vaginal delivery. On arrival, she was hypertensive at 168/117, spO2 of 100% on BiPAP. Exam was notable for diffuse rales, a regular rhythm, no JVD, and 1+ peripheral edema. Urine was positive for hemoglobin and protein and the liver panel revealed transaminitis (AST=50 ALT=53), Chest X-ray demonstrated increased pulmonary vasculature congestion, confirmed on CT angiography which was negative for venous thromboembolism. Echocardiography found a preserved ejection fraction without diastolic dysfunction or valvular abnormalities. She was managed with a nitroglycerin infusion and parenteral diuresis with resolution of her pulmonary edema. **Conclusion:** Pre-eclampsia affects 4-9% of all pregnancies and confers a high risk for both fetal and maternal morbidity and mortality. The definitive treatment for preeclampsia is delivery of fetus and placenta but there is limited information to guide the management of post-partum pre-eclampsia. It has been hypothesized that activation of maternal platelets in the placental bed play a role in preeclampsia through activation of inflammasomes such as sFlt-1. Yes, further research is needed to explore the benefit of suppressing these inflammasomes. Although peri-partum cardiomyopathy and venous thromboembolism usually come to mind in women with cardio-pulmonary complaints post-partum. In the appropriate setting, post-partum pre-eclampsia should also be entertained in the differential.

Keywords: *Pre-eclampsia, eclampsia, cardiomyopathy*

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1. Case Presentation

A 38-year-old African woman gravida 9 para 4 presented 2 weeks after an uncomplicated vaginal delivery. Blood pressure around time of delivery was 107/62. 1-week post-partum, she was evaluated by a Cardiologist for palpitations. Blood pressure at that visit was 124/82. Echocardiogram revealed normal systolic and diastolic function with estimated ejection fraction of 60-65%. She then developed progressive dyspnea, orthopnea, paroxysmal nocturnal dyspnea and central chest pain. On exam, vital signs revealed pulse 61, blood pressure 168/117, respiration rate 33, temperature 98.1F, oxygen saturation on room air 100%. There were diffuse rales heard over both lung fields, cardiac exams was unremarkable, no S3 gallop, no murmur, no friction rub or JVD. A 1+ peripheral edema was noted to be present at the lower extremities bilaterally. Pertinent laboratory: mild transaminase elevations (AST:50, ALT:53); Albumin: 30, BNP: 287.9, Hemoglobin/Hematocrit: 10.8/35.2, WBC: 8.8, Platelets: 351, Urine: positive for hemoglobin and protein. EKG revealed sinus tachycardia with no acute ischemic changes or arrythmia. Chest X-ray showed cardiomegaly, vascular congestion with no pulmonary infiltrate or effusion. CT Angiogram of chest was negative for pulmonary embolus though positive for cardiomegaly, bilateral alveolar edema and small bilateral pleural effusions. Initial impression was acute systolic heart failure versus ruling out post-partum cardiomyopathy. Parenteral diuretics with furosemide was subsequently

initiated. An echocardiogram however showed preserved left ventricular function with estimated ejection fraction 60-65%. Treatment with diuretics (Furosemide) was continued and a nitroglycerin infusion started, and she improved rapidly.

2. Images

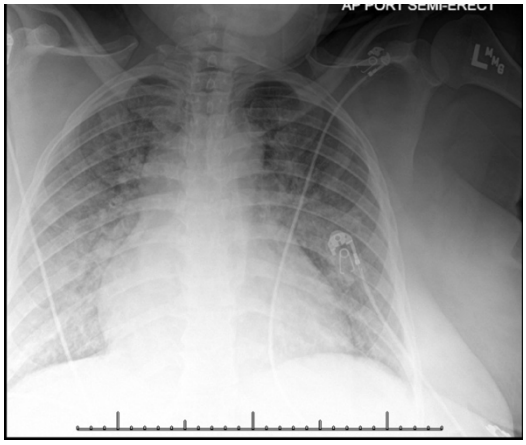

Figure 1. CXR reveals cardiomegaly and increased pulmonary vasculature markings

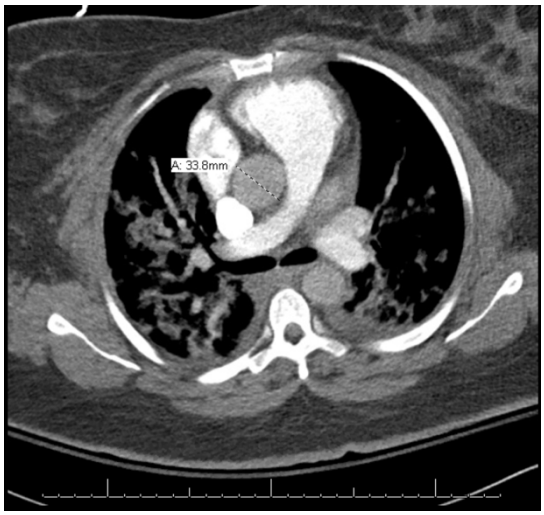

Figure 2. CT Angiography chest reveals cardiomegaly with pulmonary vascular congestion

3. Discussion

Hypertensive disorders of pregnancy remain a concern as a major cause of maternal and fetal morbidity and mortality. Pre-eclampsia/eclampsia carries the highest risk. The majority of cases present ante-partum but it is estimated that up to 5% of cases can have a late presentation [1]. Late presentations can pose a diagnostic challenge with respect to differentiating them from more common diagnoses that may have similar symptoms including pulmonary emboli and post-partum cardiomyopathy as was the case with this patient. This patient presented with signs and symptoms typical of acute systolic heart failure, but this was at odds with the echocardiogram findings. The risk of post-partum pre-eclampsia was higher when there was a 10-mmHg increase in mean arterial blood pressure between delivery and the early post-partum period [2]. One study suggested improved outcomes in post-partum pre-eclampsia with the addition of a calcium channel blocker such as nifedipine to diuretic therapy [3]. There are few distinguishing clinical parameters to guide diagnosis and treatment and at present, treatment is symptom directed. The pathophysiology of pre-eclampsia/eclampsia remains poorly understood. When it occurs ante-partum, the mainstay of treatment is prompt delivery of the fetus and placenta. It is hypothesized that activation of maternal platelets in the placental bed plays a role in promoting pre-eclampsia through activation of inflammasomes such as soluble Flt-1. Research is in progress to see if inhibition of molecules like sFlt-1 can suppress the clinical manifestations of pre-eclampsia.

In conclusion, it is important to recognize that late presentations of pre-eclampsia occur so that appropriate diagnostic studies and management can be instituted.

References

[1] Al-Safi, Zain, et al. “Delayed Postpartum Preeclampsia and Eclampsia.” *Obstetrics & Gynecology*, vol. 118, no. 5, 2011, pp. 1102-1107.

[2] Cohen, Jonathan, et al. “Blood Pressure Changes during the First Stage of Labor and for the Prediction of Early Postpartum Preeclampsia: a Prospective Study.” *European Journal of Obstetrics & Gynecology and Reproductive Biology*, vol. 184, 2015, pp. 103-107.

[3] Veena, P., et al. “Furosemide in Postpartum Management of Severe Preeclampsia: A Randomized Controlled Trial.” *Hypertension in Pregnancy*, vol. 36, no. 1, Nov. 2016, pp. 84-89.

[4] Kohli, S., et al. “Maternal Extracellular Vesicles and Platelets Promote Preeclampsia via Inflammasome Activation in Trophoblasts.” *Blood*, vol. 128, no. 17, Feb. 2016, pp. 2153-2164.

[5] Robertson, Sarah A. “Preventing Preeclampsia by Silencing Soluble Flt-1?” *New England Journal of Medicine*, vol. 380, no. 11, 2019, pp. 1080-1082.