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Case Report
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Rare Case of Late Post-partum Seizure Preceding Peri-partum Cardiomyopathy Presentation

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Abstract

Hypertensive disorders of pregnancy such as pre-eclampsia and eclampsia have been associated with the development of peri-partum cardiomyopathy. Pre-eclampsia and eclampsia can also present in the late post-partum period and may be overlooked by clinicians. We report a rare case of late post-partum eclampsia heralding the onset of peri-partum cardiomyopathy.

Keywords: Peri-partum cardiomyopathy; Pre-eclampsia; Cardiac index; IABP

Introduction

We describe a rare case of 40 years old with uncomplicated full-term delivery via elective Lower Segment Caesarian Section (LSCS), who later developed post-partum generalized seizures Day 7 post-partum, requiring hospitalization for observation. She was discharged but subsequently was re-admitted Day 12 post-partum, for acute pulmonary edema with acute heart failure requiring ventilatory support, Intra-Aortic Balloon Pump (IABP) and inotropic support. To our knowledge, this case is the first known report of late post-partum seizure, heralding subsequent onset of peri-partum cardiomyopathy.

Case Presentation

Our patient is a 40 years old South Asian lady with no prior medical history. She was Gravida 3 Para 2, with a prior successful delivery in 2009, an early miscarriage in 2017 and with the most current pregnancy being uneventful, delivering at full term via elective LSCS in December 2018. Her Blood Pressure (BP) at Day 3 post-partum was 124/81 mmHg prior to her discharge.

Admission for post-partum seizure

The patient started having frontal headache in the evening at Day 6 post-partum. In the morning at Day 7 post-partum, the patient was witness by her husband to be having seizures. She was unconscious with generalized jerks and mouth foaming. The episode lasted 5 min to 10 min. She had second episode lasting under 1 min in the ambulance and a third episode in the Emergency Department (ED). Postictal she was initially confused, restless and uncooperative. She was given IV Midazolam 5 mg as well as IV phenytoin 1 g loading dose in the ED. Initial BP in the ED triage was 197/113 mmHg with a HR of 146 bpm but subsequent BP in ED was 147 to 160/100 to 109 mmHg. CT head scan showed no intracranial hemorrhage or established territorial infarct.

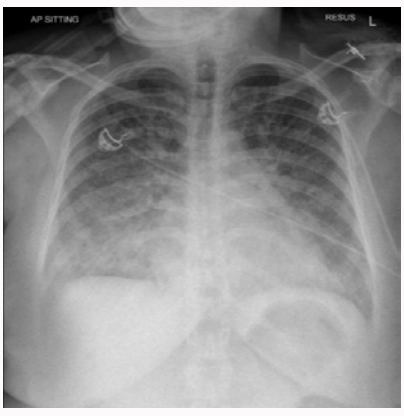

Figure 1: CXR at showing cardiomegaly and extensive bilateral alveolar edema.

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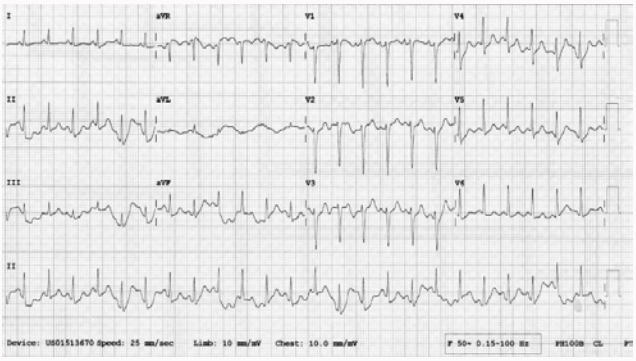

Figure 2: Initial ECG showing sinus tachycardia and non-specific mild ST depressions.

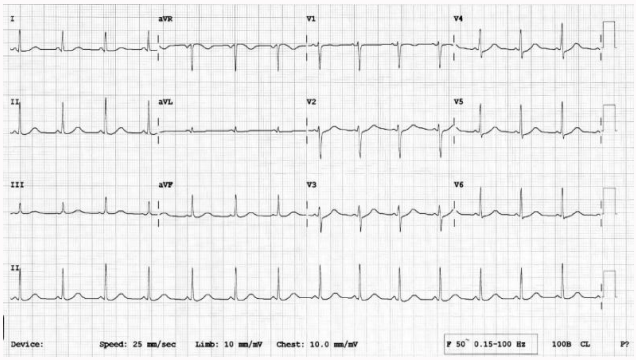

Figure 3: ECG Day 2 admission.

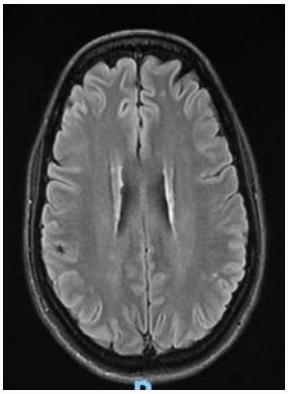

Figure 4: MRI Brain (T2, FLAIR) showing hyper intense foci in the periventricular and deep white matter.

CT head venogram shows no dural sinus thrombosis. Our patient gradually became more alert in the ED, neurological examination, when patient was orientated and cooperative, was normal. She was subsequently admitted for monitoring. There were no further seizure episodes inpatient and Phenytoin was discontinued. Her BP in the ward ranged from 126 to 147/81 to 98 mmHg. No antihypertensives were started. She was unable to provide a clear urine specimen to assess for proteinuria during the admission. EEG had shown continuous generalized slow activity suggestive of moderate diffuse encephalopathy, no epileptiform activity. Patient was subsequently discharged at Day 3 of admission.

Re-admission for acute heart failure

Two days later, at Day 12 post-partum, patient had a sudden episode of breathlessness and was sent to the ED. She was in acute pulmonary edema and required endotracheal intubation and ventilation (Figure 1).

Initial BP was 201/126 mmHg and heart rate was 166 bpm at triage (Figure 2). Initial treatment was given with IV Frusemide 120 mg total and IV GTN 100 mcg/min infusion. IV GTN was reduced and stopped as blood pressure continued to decrease reaching the lowest point of 68/31 mmHg. Inotropic support with IV dopamine was commenced. Bedside 2DE showed LV ejection fraction of 30% with global hypokinesia. Initial laboratory investigations showed hemoglobin of 19.5 g/dl, total white blood cell count of 22×10^9/L and platelet count 537×10^9/L. Liver function test showed elevated ALP at 227 U/L and only mildly elevated AST at 50 U/L. Urine dipstick showed 4+ proteinuria. Serum creatinine, prothrombin time and partial thromboplastin time were normal. NT-pro BNP was elevated at 6242 pg/ml and troponin T peaked at 99 ng/L.

Intra-Aortic Balloon Pump (IABP) support was initiated. Coronary angiogram showed normal coronary arteries. A diagnosis of peripartum cardiomyopathy was confirmed by exclusion and patient was continued on acute heart failure therapy.

Further management

She was continued on ventilatory support, hemodynamic support with dopamine at 8 mcg/kg/min, IABP and IV diuretics. A swan ganz catheter was inserted and showed Pulmonary Capillary Wedge Pressure (PCWP) of 7 mmHg and cardiac output of 4.1 L/min by thermodilution (cardiac index was 2.36). She was also started on oral Bromocriptine 2.5 mg twice daily for 1 week then 2.5 mg daily for 6 weeks in accordance to peripartum cardiomyopathy guidelines [1]. Our patient achieved excellent diuresis and repeat CXR the next day should significant improvement in pulmonary edema. She remained on IABP support and low dose dopamine at 5 mcg/kg/min. A formal 2D Echocardiogram showed LV ejection fraction of 35% to 40%. Pulmonary capillary wedge pressure was 5 mmHg. As her ventilatory support requirements were low, being on pressure support ventilation with positive end expiratory pressure of 5 mmHg, pressure support of 8 mmHg and FiO_2 30%, she was successfully extubated at Day 2 post-endotracheal intubation (Figure 3).

Our patient was gradually weaned off dopamine and IV diuretics were switched to oral medications. Cardiac output study at day 4 without inotropes and with IABP on standby showed cardiac output of 3.47 l/min (cardiac index 2.1) and pulmonary capillary wedge pressure of 13 mmHg. IABP was removed and patient was gradually started on medical therapy for heart failure. An MRI/MRA brain was performed and did not show any acute infarct, hemorrhage, enhancing lesions or dural sinus thrombosis (Figure 4). There were however nonspecific hyper intense foci possibly due to chronic micro-vascular ischemic changes.

She was started on low dose ACE inhibitor (Enalapril 2.5 mg daily), Mineralocorticoid receptor antagonist (Spironolactone 12.5 mg daily), Frusemide 20 mg daily, and Ivabradine 2.5 mg twice daily. She underwent inpatient rehabilitation and was discharged at Day 10 post admission. As her 2D echocardiogram ejection fraction was 35% and no ventricular arrhythmias were detected while inpatient, Implantable Cardio Defibrillator (ICD) was not required.

Our patient was subsequent reviewed in the outpatient clinics and was well. A follow-up 2DE four months post discharge showed further recovery with LVEF of 50%. Frusemide and Spironolactone

were stopped, and patient was continued on low dose Enalapril and Bisoprolol.

Discussion

Hypertensive disorders of pregnancy such as pre-eclampsia and eclampsia are estimated to occur in 4.6% and 1.4% of pregnancies respectively [2] and represent a major cause of maternal morbidity and mortality. Preeclampsia is the development of hypertension (SBP ≥ 140, DBP ≥ 90) after 20 weeks gestation, with either proteinuria or the presence of either thrombocytopenia impaired renal function, transaminitis or pulmonary edema. HELLP (Hemolysis, elevated liver enzymes and low platelet count) syndrome is a severe form of pre-eclampsia with higher morbidity and is diagnosed by LDH levels over 600 U/L, AST and ALT elevation of twice the upper limit, and platelet count of less than 100×10^9/L.

Eclampsia is the development of new onset seizures, in the absence of other causes such as cerebral ischemia, infarction, hemorrhage or thrombosis [3]. While predominantly a disease of during pregnancy and the immediate postpartum (48 h), late postpartum eclampsia, occurring 48 h to 4 weeks is well recognized with 16% of eclampsia occurrences in the late postpartum period [4-7]. Contrary to the nomenclature, pre-eclampsia and eclampsia are not linear progression of a disease and over a third of patient presenting with eclampsia may not have previously been diagnosed with pre-eclampsia.

Peripartum cardiomyopathy on the other hand, is less frequent, occurring in 1 out of 1000 to 3000 pregnancies, but represent an equally, if not more severe development with up to 13% of patients having persistent severe left ventricular dysfunction (LVEF <30%) or major events (death, LVAD, transplant) at 1 year follow-up [8]. The association of pre-eclampsia and peri-partum cardiomyopathy is well known with 22% of patients with peri-partum cardiomyopathy have preexisting pre-eclampsia. However over 90% of patients with pre-eclampsia do not develop peripartum cardiomyopathy [9]. Although they might share some similar substrates in their pathophysiology, clinically, they are separate entities with different management strategies.

The two hit hypothesis for peri-partum cardiomyopathy suggest that patients with genetic predisposition to cardiomyopathy have an underlying mutation in the TTN gene which codes titin, the large protein molecule in myocytes. This constitutes the "first hit". During late pregnancy and delivery, prolactin levels peaks. Oxidative stress peripartum results in cardiomyocytes expressing cathepsin D, an enzyme that cleaves prolactin into a 16 kDa fragment which causes endothelial apoptosis. This is the "second hit" that then results in cardiac dysfunction. Other factors such as soluble fms-like tyrosine kinase receptor 1 (sFlt-1) a protein released at higher amounts by the placenta towards the end of pregnancy, and believed to be responsible for the endothelial dysfunction and development of hypertension in pre-eclampsia, is also postulated to contribute to the peri-partum cardiomyopathy [10]. In addition to standard heart failure management (beta-blockers, ACE inhibitors, mineral corticoid receptor antagonist and diuretics), bromocriptine, through its effect on reducing prolactin secretion has been shown to improve LVEF recovery and maternal morbidity in severe peripartum cardiomyopathy [11,12]. Due to the prothrombotic effect of bromocriptine prophylactic anticoagulation should be given while on bromocriptine.

Conclusion

We have described a rare case of late post-partum eclampsia which precede the presentation of acute heart failure from peri-partum cardiomyopathy. Late post-partum pre-eclampsia and late post-partum eclampsia are uncommon but potentially serious conditions that may lead to patient admission to general medical or neurology units. An awareness of their management and their association with peripartum cardiomyopathy would allow for close monitoring and management to reduce maternal morbidity. Prompt management of peri-partum cardiomyopathy with guideline-based therapy and hemodynamic support till recovery allows for the most favorable outcomes.

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