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Acute Pulmonary Edema in an Eclamptic Pregnant Patient: A Rare Case of Takotsubo Syndrome

E **Kunal Karamchandani**
F **Brandon Bortz**
E **Sonia Vaida**

Department of Anesthesiology and Perioperative Medicine, Penn State Hershey Medical Center, Hershey, PA, U.S.A.

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Corresponding Author: Kunal Karamchandani, e-mail: kkaramchandani@hmc.psu.edu
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Patient: Female, 35
Final Diagnosis: Takotsubo cardiomyopathy
Symptoms: Seizures
Medication: —
Clinical Procedure: Cesarean section
Specialty: Critical Care Medicine

Objective: Rare co-existence of disease or pathology
Background: Acute pulmonary edema in a pregnant patient is associated with significant morbidity and mortality. Takotsubo syndrome, or stress-induced cardiomyopathy, is a rare cause of acute pulmonary edema in a pregnant patient, especially prior to delivery of the fetus.
Case Report: We describe a case of a pregnant patient who presented with acute pulmonary edema and eclampsia and was found to have Takotsubo syndrome. To the best of our knowledge, eclampsia as a precipitating factor for Takotsubo syndrome has not been described in literature.
Conclusions: Clinicians taking care of pregnant patients should be aware of the potential link between eclampsia and Takotsubo cardiomyopathy. Prompt correction of the precipitating cause along with supportive management as described is the key to a successful outcome.

MeSH Keywords: Eclampsia • Pulmonary Edema • Takotsubo Cardiomyopathy

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Background

Pulmonary edema in a pregnant patient can be a challenge to manage and can have various etiologies, including preeclampsia, tocolysis, sepsis, pre-existing cardiac disease, and pregnancy-associated cardiac disease. Takotsubo cardiomyopathy (TC), a form of stress-induced heart disease, has also been described as a cause, but its occurrence is rare. A recent review looked at the incidence of TC in pregnant women and found only 29 published cases, with the majority occurring in the post-partum period [1]. We describe a case of a 25-weeks-pregnant woman who presented with eclampsia and pulmonary edema and was found to have TC at presentation.

A written consent was obtained from the patient for publishing this case report.

Case Report

A 35-year-old G₂P₁ female, weighing 120 kg with a body mass index (BMI) of 47 kg/m², presented to the emergency department after having an episode of seizure at home and another episode in the ambulance. On admission, she was hypertensive with a blood pressure of 196/133 mm Hg and a heart rate of 154 bpm with occasional episodes of agitation. The husband denied any history of seizures and indicated that they had recently found out that the patient was pregnant, and they were scheduled for their first antenatal appointment in a few days. Her past medical history was insignificant, and her past surgical history was significant for an uneventful cholecystectomy. A computed tomography scan of the head was normal. Chest X-ray revealed pulmonary edema, and an echocardiogram (ECHO) revealed an ejection fraction (EF) of 25% with severe akinesia, ballooning of the apex, and excellent function in the inferolateral base (Figure 1). The patient's electrocardiogram (EKG) on admission revealed sinus tachycardia with non-specific ST-T changes with no ST elevations. Her laboratory work was significant for a positive urine pregnancy test, leukocytosis, and proteinuria detected on spot urine test. Her initial troponin I value on admission was 2.650 ng/mL (normal range 0–0.120 ng/mL). The fetus had an estimated gestational age of 25 weeks 5 days and a normal heart rate detected by Doppler. The patient received 4 g of IV magnesium sulphate along with a loading dose of IV phenytoin (20 mg/kg) for seizure control. Over the next hour, she became tachypneic with the respiratory rate in the 40s and hypoxemic, with a PaO₂ of 52 mm Hg on a Venturi mask with FiO₂ of 80%, and required an emergent intubation. The endotracheal intubation with a size 7.5 mm oral tube was uneventful and was followed by severe, persistent hypotension. Norepinephrine infusion was initiated and titrated to maintain the mean blood pressure (MAP) higher than 65 mm Hg.

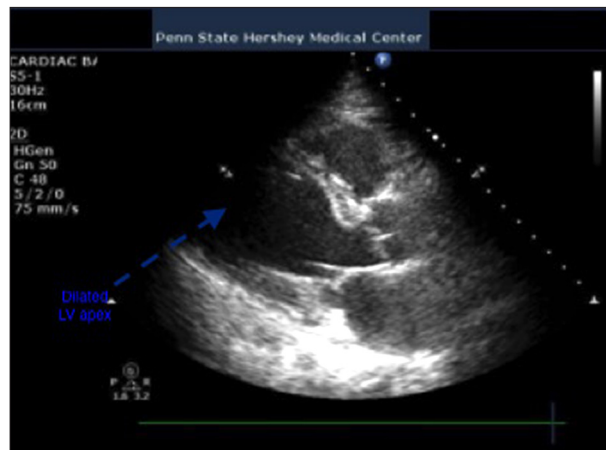


Figure 1. Echocardiography image on admission showing dilated left ventricular apex.

Fetal bradycardia ensued, which was persistent, and the patient was transferred to the operating room for an emergent cesarean section. A continuous propofol infusion at a rate of 25 µg/kg/min, which was started soon after intubation, was continued for providing anesthesia for the surgery. She received IV midazolam 2 mg at the start of the surgery. Neuromuscular blockade was achieved with IV rocuronium 50 mg. Norepinephrine infusion was continued throughout the surgery. A viable 25-week-old baby was delivered and sent to the neonatal intensive care unit (NICU) after being intubated for poor respiratory effort. The baby's APGAR scores at 1 minute, 5 minutes, and 10 minutes were 1, 5, and 6, respectively. The mother was transferred to the surgical intensive care unit (SICU) with the norepinephrine infusion maintained and mechanical ventilation with a FiO₂ of 100%. In the SICU, the norepinephrine was weaned off over the next 8 hours; she had a dramatic improvement in her cardiovascular and respiratory status over the next 24 hours. Her oxygen requirements decreased from a FiO₂ of 100% at admission to the SICU to 40% the next morning, and she was extubated after meeting the necessary criteria. Her EF improved to 45% on the next ECHO examination performed 48 hours later. Her troponin levels at 6 hours and 12 hours after admission were 2.550 ng/mL and 1.540 ng/mL (normal value 0–0.120 ng/mL), respectively. In view of significant improvement in the patient's clinical status and left ventricular function, it was decided not to pursue cardiac catheterization. She received around-the-clock diuresis with intravenous furosemide titrated for clinical response and blood pressure control with a combination of oral hydralazine and labetalol to target systolic blood pressures (SBPs) less than 160 mm Hg over the next few days. She was discharged home after six days of hospital stay on oral carvedilol 25 mg twice a day and lisinopril 10 mg once a day. A follow-up ECHO was performed six weeks after delivery, showing an ejection fraction of 65% (Figure 2), and her workup for viral myocarditis, including serological testing, was negative.

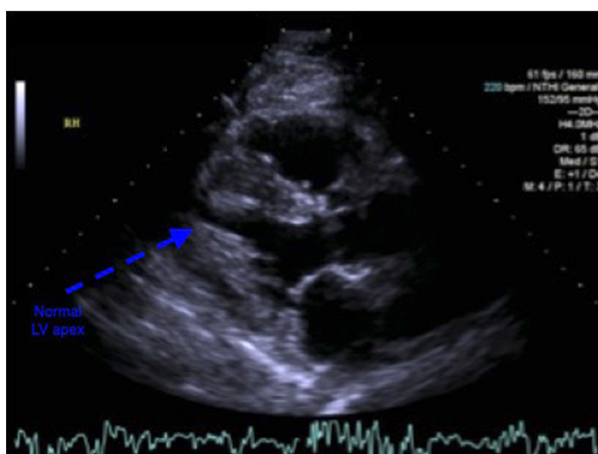


Figure 2. Echocardiography image taken at two-month follow-up showing normal left ventricular apex.

Discussion

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy and “broken-heart syndrome,” was first described in 1990 [2]. A marked hyper-adrenergic state appears to be the etiology in most of the cases [3]. The appearance of the heart on echocardiography is classically described as having “apical ballooning,” but it may present as apical akinesis or dyskinesis with relative sparing or hyperactivity of the base [4]. The condition mimics acute coronary syndrome and can be associated with most of the features associated with it, including EKG changes, elevation in the level of cardiac biomarkers, and similar clinical presentation. The most important distinguishing feature is the rapid recovery of left ventricular function [4]. Our patient had dilatation of the left ventricular apex with sparing of the base along with non-specific EKG changes and elevation in cardiac biomarkers. Her rapid clinical and echocardiographic improvements along with the absence of any risk factors for coronary artery disease made us not pursue cardiac catheterization.

The management of TC is essentially supportive, with measures to reduce the work of the left ventricle. During the acute episode of cardiogenic shock, the use of inotropic agents is controversial, considering that hyper-adrenergic activity is usually responsible for this condition [5]. Intra-aortic balloon pumps can be placed to help unload the left ventricle while augmenting the coronary perfusion. In the recovery phase, a combination of diuretics and antihypertensive agents appears to help reduce both volume and pressure overload on the ventricle. Appropriate management of the precipitating cause reverses the condition in majority of the cases [4], as happened in our patient after the delivery of the fetus.

While there are some data on TC presenting in the post-partum period, wherein the stress of cesarean section or vaginal delivery could trigger a hyper-adrenergic state, there are only two reported cases of this syndrome occurring prior to delivery of the fetus [6,7], and neither of these two patients was diagnosed with pre-eclampsia or eclampsia.

For our patient, we assume that eclampsia could have been a trigger for TC. Epileptic seizures have been reported to induce TC [8], and the possible mechanism is an excessive release of catecholamines. Although pre-eclampsia is associated with increased sensitivity to endogenous catecholamines [9] and development of peripartum cardiomyopathy (PPCM) [10], no association with TC has been described in the literature. It is very likely that the central nervous system excitation associated with eclampsia had a role in the development of TC in our patient.

Though our patient had significant recovery after the delivery of the fetus and supportive care in the SICU, management of acute pulmonary edema in a pregnant patient can be challenging. General anesthesia is the modality of choice for the delivery of the fetus, and invasive hemodynamic monitoring is essential. Postoperative mechanical ventilation and ICU care are needed in majority of the cases. Pulmonary edema can be caused by various conditions affecting the capillary hydrostatic pressure, colloid osmotic pressure, or the capillary permeability within the pulmonary interstitium [11]. Our patient presented with acute pulmonary edema at admission and did not have any history to suggest a pre-existing etiology. Acute cardiogenic shock associated with TC probably contributed to pulmonary edema in our patient.

Our patient’s presentation and management highlight the importance of recognizing TC as a cause of acute pulmonary edema in an eclamptic patient. Her dramatic recovery, after delivery of the fetus, points towards eclampsia as the most likely trigger for TC.

Conclusions

We describe a case of a pregnant woman, in her second trimester of pregnancy, who presented with eclampsia and acute pulmonary edema and was diagnosed with TC. It is rare for TC to present in young pregnant females, especially prior to delivery, and to the best of our knowledge, no cases have been described where eclampsia has been linked with the onset of TC.

Conflict of interest

None.

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