

Epinephrine-Induced Tako Tsubo Cardiomyopathy: Point-of-Care Ultrasound Findings in Postoperative Care

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Abstract

Background: Takotsubo cardiomyopathy, also known as stress cardiomyopathy, apical ballooning syndrome, or “broken heart syndrome” (seen more often in women), is a condition with transient regional systolic dysfunction without angiographic evidence of obstructive coronary artery disease or acute plaque rupture. Point of Care Ultrasound (POCUS) can achieve images of cardiac function, which can often be used to interrogate possible causes of hypotension. Anesthesia teams are often required to obtain training in such modalities to be used in the perioperative setting. **Case Report:** The theme of the case is the unusual presentation of cardiomyopathy in the post-anesthesia care unit and the use of Point of Care Ultrasound to diagnose the cause of hypotension. **Conclusion:** This case illustrates why it is important to use diagnostic bedside modalities prior to generalized treatment of hypotension and how proficiency in POCUS is important.

Keywords

Tako Tsubo, Hypotension, Post-Anesthesia Care Unit

1. Introduction

The postoperative period is a crucial time in a patient’s recovery from surgery, and management during this time may have a significant impact on overall patient outcomes. Postoperative hypotension, in particular, has previously been shown to be significantly associated with mortality. [1] Though hypovolemia is frequently thought of as the most common cause of post-surgical hypotension, it is important for clinicians to be cognizant of other potential etiologies. [2] POCUS at the bed-

side has been introduced in the last decade as a quick modality to differentiate shock by acquiring and interpreting cardiovascular images.

Tako Tsubo cardiomyopathy, also known as an apical ballooning syndrome or 'broken heart syndrome', is a condition with transient regional systolic dysfunction without angiographic evidence of obstructive coronary artery disease or acute plaque rupture. [3] The prevalence is 5% - 6% in female patients and 2% - 3% in all patients, and it is a diagnosis of exclusion. [4] The exact pathophysiology is not well understood, but it is thought that the condition may be precipitated by exposure to supratherapeutic doses of catecholamines during times of physical or emotional stress. Epinephrine is a catecholamine that can induce arrhythmias, although the incidence is minimal when administered through the inhalational route.

While local and superficial administration of epinephrine is a common method utilized during bronchoscopies for hemostatic control, especially after bronchial biopsies, adverse cardiac events may develop even after a single dose. We present a case in which endobronchial epinephrine was administered intraoperatively to a 60-year-old female patient without any known cardiac history. Our emphasis is to appreciate unusual causes of hypotension and to use POCUS readily to differentiate shock, so that incorrect therapy (fluid boluses being the usual therapy) is not delivered blindly.

2. Case Report

A 60-year-old, 60 kg female patient presented for flexible and rigid bronchoscopy, endobronchial ultrasound, and transbronchial biopsy for a workup of possible interstitial lung disease of unknown etiology. Pulmonary function tests also revealed moderate restriction and severely reduced diffusion capacity for carbon monoxide (DLCO). Her past medical history was significant for hypertension, hyperlipidemia, and anxiety. Her social history was notable for light former smoking two cigarettes per day for ten years over 25 years ago. Since the patient was asymptomatic, active, and young, so no cardiac testing was done preoperatively.

On the day of the procedure and preoperative evaluation, the patient denied any previous cardiac disease and reported a history suggesting metabolic equivalents (METs) greater than four as she walked three miles daily. She endorsed continued intermittent cough and denied dyspnea or chest pain. Preoperative vital signs were within normal limits, including a blood pressure of 129/90.

In the operating room, general anesthesia was induced with intravenous lidocaine 10 mg, remifentanyl 20 mcg, propofol 150 mg, and rocuronium 60 mg. Intubation was successfully performed by the interventional pulmonology team with rigid bronchoscopy, and ventilation achieved with jet ventilation. Propofol and remifentanyl infusions were used for maintenance anesthesia, and anesthetic depth was monitored using SedLine® brain monitoring (processed electroencephalogram).

The patient was stable during the case, with the pulse ranging from 60 s to 70 beats per minute and mean arterial pressures ranging from 70 to 100 mmHg. Approximately fifty minutes after the procedure started, a routine dose of intrabron-

chial epinephrine was administered by interventional pulmonology according to routine steps to ensure hemostasis following instrumentation and biopsy. Immediately following intrabronchial epinephrine installation, intraoperative ECG showed ventricular tachycardia (VT), which was treated and aborted by 40 mg of intravenous esmolol. The abnormal rhythm lasted for fifteen seconds. Following the incident, the procedures undertaken included transbronchial biopsy and hemostasis. Approximately 10 minutes after this event, the procedure ended, the airway was transitioned from rigid bronchoscopy to supraglottic airway (laryngeal mask airway) by the anesthesia team, and the total intravenous anesthetic was stopped. Concomitantly, significant hypotension with blood pressures ranging from 68 - 80 systolic over 48 - 65 diastolic was noted. This was treated with multiple boluses of intravenous phenylephrine, and a phenylephrine infusion was started with normalization of blood pressure. The patient was successfully extubated and transported to the post-anesthesia care unit (PACU) on a phenylephrine infusion at 0.5 mg/kg/min.

In the PACU, a 12-lead electrocardiogram (ECG) showed normal sinus rhythm without evidence of ischemia, CXR showed known bi-basilar opacities, pulmonary edema, and no pneumothorax. CBC, CMP, and high-sensitivity troponins were within normal limits. Interventions included administering 500 ml intravenous fluid bolus in addition to the 600 ml given intraoperatively and weaning phenylephrine infusion as tolerated with a mean arterial pressure (MAP) goal greater than 65 mmHg. The patient reported feeling tired; however, they denied any other symptoms, including chest pain or dyspnea. After a liter of NS was given over 60 minutes without improvement in MAP, a bedside POCUS was performed by the physician team as the patient continued to require a vasopressor. The POCUS exam was concerning for apical hypokinesis and a significantly dilated left ventricle. The visual EF was 25% - 30% with a reduced VTI (volume time integral) < 14.

A cardiology consult was placed, and a formal transthoracic echocardiogram was obtained, which confirmed beside POCUS exam findings showing “extensive areas of severe LV systolic dysfunction with akinesis of mid to distal segments and hypokinesis of the apex and estimated ejection fraction of 25% - 30%, all suggestive of Tako Tsubo stress vs obstructive cardiomyopathy”. (**Figure 1**)

She was switched to norepinephrine infusion for cardiogenic shock and admitted to the coronary care unit (CCU) for further management. Dobutamine was not chosen due to the history of recent ventricular arrhythmias and ongoing hypotension. She was weaned off the vasopressor and started on diuresis shortly after admission to the CCU. A left heart catheterization was also done, which showed no coronary artery disease. Further workup, including Iron studies, Lipids, Hb A1C (diabetes), TSH (thyroid), and HIV (AIDS), was unremarkable. She was transferred to the floor on postoperative day two, where she was started on metoprolol for heart failure goal-directed medical therapy. During her CCU stay, she was in heart failure with pulmonary edema, requiring supplemental oxygen and daily diuresis. She was discharged home on postoperative day three on home oxygen with a plan

for cardiology follow-up and repeat transthoracic echo to assess ejection fraction recovery.

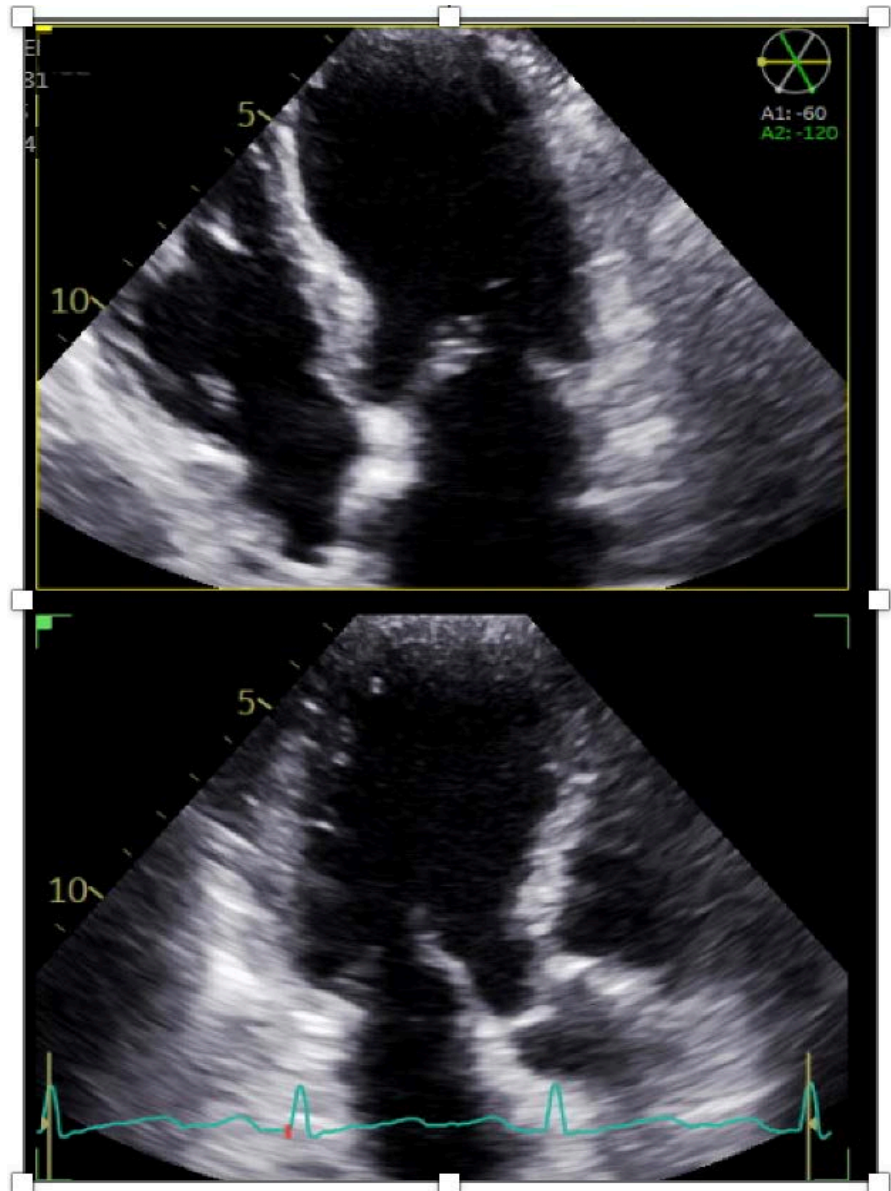


Figure 1. 4 and 3 chamber POCUS apical view.

3. Discussion

Takot Tsubo's cardiomyopathy is a diagnosis of exclusion requiring clinical and diagnostic assessment. [5] This case shows how bedside POCUS can be diagnostic for unusual presentations as causes of hypotension in the PACU and should be routinely performed prior to fluid challenges or vasopressor therapy after general anesthesia. Although several causes can contribute to stress cardiomyopathy, such as ischemia, spasm, and stress of the biopsy itself, the temporal relationship to the epinephrine nebulization seems the most likely culprit, and ischemia was later ruled

out. Often, perioperative causes of hypotension are blindly attributed to residual vasoplegia from the anesthetic. [6] However, an US-driven assessment of undifferentiated causes of hypotension or shock can be very useful as demonstrated in this case, saving time and inappropriate fluid administration and end-organ damage. [7] It is likely that if POCUS had been performed upon admission to the PACU while the patient was receiving a continuous infusion of phenylephrine, additional fluid loading might have been avoided. Providers and trainees should continue to practice and gain skills in quick bedside lung and heart POCUS. [8]

The POCUS was done after the usual course of treatment, which was a fluid bolus. The proportion of patients vasoplegic from general anesthesia in the PACU is much higher than the chance of Tako Tsubo cardiomyopathy, especially as the patient was asymptomatic and had no EKG changes. Also, although POCUS in the PACU is not the usual standard of care yet, it is definitely available, and more providers should become comfortable with its use in all perioperative scenarios.

The occurrence of VT during the case, which temporally followed the intratracheal administration of epinephrine, appears to be the most apparent cause of cardiomyopathy. This is particularly true as the typical causes of such a condition, like extreme catecholamine surges or stress, were absent in the PACU. The POCUS conducted in the PACU was a basic point-of-care assessment but revealed a significantly reduced EF (approximately 20% - 30%), enabling us to treat her with inotropes and confirm the findings with a formal TTE. We were also able to avoid administering additional fluids, which would typically be the standard approach in the PACU, and instead send her to the CCU. The POCUS facilitated the appropriate management of the case.

4. Conclusion

This case illustrates how POCUS can help differentiate hypotension in the recovery room or PACU. *There is no prior case report of such an occurrence, making this a novel case report.* Often, the treatment given, such as fluids, may be counterproductive, as seen in this case. The use of intraoperative catecholamines can lead to cardiac stress, and all postoperative hypotension should not be treated the same. Handover in the PACU from OR anesthesiologists and surgeons should mention any major events and agents used. The take-home message in this case is that all hypotension is not the same, that the use of POCUS should be made available, and that all providers must be comfortable with its use. Trainees must be trained in this modality to obtain simple cardiac POCUS views.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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