

Tamponade: To Be or Not to Be? Using Point-of-Care Ultrasound to Answer the Question



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INTRODUCTION

Hypotension in the postanesthesia care unit (PACU) is a commonly encountered occurrence with a broad differential. In the perioperative period, the etiology is frequently due to hypovolemia stemming from a combination of perioperative fasting, inadequate fluid resuscitation, and ongoing hemorrhage or fluid losses. While hypovolemia is the most common etiology, it is not the only etiology. It is critical to identify alternative causes of hypotension to appropriately manage the patient. We present a case in which point-of-care ultrasound (POCUS), specifically cardiac POCUS, was essential in diagnosing the cause of postoperative hypotension and guiding the appropriate treatment. In this case, cardiac POCUS refers to the focused cardiac exam performed bedside by an anesthesia provider to determine the etiology of hypotension. POCUS is being increasingly utilized across specialties to answer specific clinical questions, guide therapy, and direct consultations.

CASE PRESENTATION

The patient was a 51-year-old man with a history notable for esophageal squamous cell carcinoma status post-radiation and chemotherapy who underwent esophagogastroduodenoscopy for removal of esophageal stents. He had no other pertinent past medical history and no history of a pericardial effusion. He did not have a preoperative echocardiogram or chest imaging at our institution. After an uneventful intraoperative course, the patient was transferred to the PACU. Two hours after PACU admission, the patient was noted to be in atrial fibrillation on bedside monitor with mean arterial pressures of approximately 50 mm Hg. A 12-lead electrocardiogram was obtained that demonstrated atrial fibrillation with rapid ventricular response with a heart rate in the 120s. Subsequently, the PACU team was called to the bedside for evaluation of new-onset atrial fibrillation and hypotension.

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Keywords: Point-of-care ultrasound, Transthoracic echocardiography, Perioperative medicine, Cardiac tamponade

Conflicts of Interest: None.

The view herein are those of the authors and do not reflect the policy or position of the San Antonio Military Medical Center, the U.S. Air Force Medical Department, the U.S. Army Surgeon General, the Department of the Army, the Department of Defense, or the U.S. Government.

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2468-6441

<https://doi.org/10.1016/j.case.2022.04.015>

266

To evaluate possible etiologies, the PACU team utilized POCUS in addition to a clinical exam to evaluate cardiac contractility, preload, and afterload. A cardiac POCUS exam was performed by an anesthesiologist with experience in cardiac echocardiography. The left ventricular ejection fraction was visually estimated, and wall motion was assessed. The left ventricular function appeared normal, and no regional wall motion abnormalities were noted. Preload was assessed via visual estimations of ventricular size at end diastole to evaluate for adequate filling. Ventricular size was also evaluated at end systole to evaluate for the possibility of near complete emptying, which could be seen in a low-afterload state such as sepsis. The exam showed a large 19 mm pericardial effusion. As this exam was performed with the patient in atrial fibrillation, the right atrium was noncollapsible throughout the cardiac cycle. The right ventricle did not collapse in diastole, which would be strongly indicative of cardiac tamponade. These findings on cardiac POCUS lowered cardiac tamponade on the differential diagnosis, and management of atrial fibrillation was started.

While working up the cause of hypotension in this patient, small phenylephrine boluses were administered to treat hypotension. The patient's hypotension was responsive to these boluses, and thus an arterial line was inserted and a phenylephrine infusion started. Following the initiation of phenylephrine, the patient's blood pressure improved, with mean arterial pressures greater than 65 mm Hg. At this point the patient was still in atrial fibrillation, with heart rates greater than 100 beats per minute. The patient had a normal mean arterial pressure and an appropriate mental status. A crash cart with cardioversion capability was brought bedside but not used as the patient was stable. Treatment of stable atrial fibrillation was started with small doses of β -blockers.

Cardiology was consulted for a formal transthoracic echocardiogram as well as for management of new-onset atrial fibrillation. The patient was eventually transferred to the intensive care unit due to ongoing vasopressor requirements. His hypotension continued to improve overnight, and he was transferred out of the intensive care unit the following day.

Initial cardiac POCUS exam (Sonosite Edge II; Fujifilm) in the PACU demonstrated a large pericardial effusion without right ventricular or right atrial collapse. Although simultaneous electrocardiogram tracing was not obtained, tricuspid valve motion was evaluated to define the phase of the cardiac cycle (Figures 1 and 2, Video 1).

A cardiology consultation was requested, and a complete transthoracic echocardiogram was obtained (Philips Affiniti 70; Philips Healthcare), which confirmed the findings noted on POCUS and also revealed a nondilated inferior vena cava, making cardiac tamponade less likely (Videos 2 and 3).

DISCUSSION

Hypotension is a frequent occurrence in the intraoperative period and often continues postoperatively in the PACU. In this phase of care, hypotension has a multitude of etiologies, each with unique

VIDEO HIGHLIGHTS

Video 1: Subcostal 4-chamber view obtained with a Sonosite Edge II ultrasound machine. The video shows a large pericardial effusion.

Video 2: Subcostal 4-chamber view obtained with a Phillips Affiniti 70 ultrasound machine. The video shows a large pericardial effusion without right ventricular diastolic collapse or right atrial systolic collapse.

Video 3: Subcostal inferior vena cava view obtained using a Phillips Affiniti 70 ultrasound machine. It shows a nondilated inferior vena cava, which would be unlikely to be seen in the setting of cardiac tamponade.

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management strategies. With the advent of POCUS, differentials can be evaluated objectively in real time. Data extracted from POCUS exams can circumvent the need for additional testing, resulting in more expeditious treatment.

The case described above illustrates the power of POCUS for evaluating hypotension in a complex patient in the PACU. The patient had a complicated medical history, resulting in a myriad of possible etiologies to explain his hypotension, each with conflicting treatment goals. Hypotension from a new pericardial effusion concerning for cardiac tamponade would lead to treatment with fluids, avoidance of reductions in contractility, and pericardiocentesis. Hypotension from atrial fibrillation with rapid ventricular response would lead to treatment with cardioversion or β -blockade. Hypotension from hypovolemia in an acutely ill, fasting patient would be corrected with volume administration.

Cardiac tamponade is a clinical diagnosis describing the hemodynamic compromise that results from cardiac compression due to fluid in the pericardial space. It is often stressed that cardiac tamponade is a clinical diagnosis and that the confirmatory test for cardiac tamponade is hemodynamic improvement following pericardiocentesis. Physical exam findings consistent with tamponade include dyspnea, tachycardia, pulsus paradoxus, and elevated jugular venous pressure.¹ The sensitivity of these physical exam findings is high, but specificity is lacking.¹ These clinical findings can be found in many of the possible causes for hypotension in this patient as described above. Of note, pulsus paradoxus (a decrease in systolic blood pressure >10 mm Hg with inspiration) is often used clinically to detect hemodynamically significant pericardial effusions. This can be readily assessed with an intra-arterial catheter, as sphygmomanometric pressures may be insufficiently precise in this setting.²

In our patient without invasive arterial access whose clinical presentation was undifferentiated, the use of POCUS was informative. While tamponade is a clinical diagnosis, echocardiography was especially useful in the setting of the patient's conflicted clinical picture. When fluid accumulates in the pericardial space, it first affects the low-pressure chambers. Thus right-sided chambers are more affected compared with left-sided chambers. Effects are first seen in the phase of the cardiac cycle in which the filling pressures are the lowest, thus during systole for the atria and during diastole for the ventricle. Due to changes in atrial pressures seen in atrial fibrillation, atrial fibrillation may complicate the ability to distinguish the presence of atrial



Figure 1 Subcostal 4-chamber view obtained with a Sonosite Edge II ultrasound machine. Diastolic still frame shows lack of right ventricular collapse. Diastolic frame was identified by motion of the tricuspid valve.



Figure 2 Subcostal 4-chamber view obtained with a Sonosite Edge II ultrasound machine. Systolic still frame shows lack of right atrial collapse. Systolic frame was identified by motion of the tricuspid valve.

compression in systole due to tamponade but should not affect the duration of right atrial inversion in relationship to the duration of the cardiac cycle.³ Echocardiographic evidence of right ventricular diastolic collapse has been shown to occur early in the clinical course of cardiac tamponade and has a higher sensitivity and specificity than pulsus paradoxus alone.⁴

The presence or absence of pulsus paradoxus in the setting of hypovolemia or atrial fibrillation with rapid ventricular response may be misleading when evaluating for cardiac tamponade. Pulsus paradoxus is based on the concept that heart-lung interactions cause variations in the loading conditions of the right and left ventricle in the presence of cardiac tamponade are exaggerated. In situations of hypovolemia, there is also an exaggerated decrease in left ventricular filling during inspiration, which can meet the definition for pulsus paradoxus in the absence of cardiac tamponade.⁵ Additionally, pulse pressure variation has been studied as an indicator for hypovolemia and volume responsiveness. The studies of pulse pressure variation and volume responsiveness have very specific parameters, which were not met by the patient in this case (mechanically ventilated, paralyzed, tidal volume >7 mL/kg, no arrhythmias).⁶ As both pulse pressure variation and pulsus paradoxus are based on heart-lung interactions, both could be exaggerated in either cardiac tamponade or hypovolemia. Thus, the presence or absence of either would not narrow the differential in this case. Additionally, in this case the

presence of atrial fibrillation precludes the ability to accurately use pulsus paradoxus or pulse pressure variation to assist in the determination of hypovolemia or cardiac tamponade. In atrial fibrillation, there is beat-to-beat variation in stroke volume independent of respiration. Furthermore, it has been shown that this stroke volume variation increases with increasing heart rate in atrial fibrillation.⁷ The presence or absence of pulsus paradoxus, unless extremely abnormal, would not have assisted with the determination of cardiac tamponade in this case because hypovolemia as well as atrial fibrillation with rapid ventricular response remained on our differential diagnosis.

The bedside cardiac POCUS exam showed a large pericardial effusion without right atrial systolic or right ventricular diastolic collapse. The absence of these echocardiographic features made cardiac tamponade a less likely culprit for the patient's hypotension. The use of cardiac POCUS allowed us to determine that while the patient had a large pericardial effusion, it did not have the echocardiographic features consistent with cardiac tamponade. This lowered cardiac tamponade on our differential diagnosis. Due to the endoscopic nature of the procedure and minimal blood loss, we thought that hypovolemia was also unlikely to be the cause of the hypotension. Because there was an acute rhythm change noted prior to the hypotension and there was no echocardiographic evidence of tamponade, we determined that atrial fibrillation with rapid ventricular response was the most likely explanation. The hypotension was temporized with phenylephrine boluses, and metoprolol was given to slow the heart rate. Soon after the administration of metoprolol, the heart rate decreased, and the patient eventually converted to normal sinus rhythm. An immediate improvement in blood pressure was noted.

CONCLUSION

POCUS is an invaluable bedside tool that spans medical specialties and phases of care. As equipment availability and expertise increase so will the applicability of POCUS. This case demonstrates the utility

of POCUS for PACU care by an anesthesia provider searching for the primary etiology of postoperative hypotension. Application of POCUS directly improved this critically ill patient's care by narrowing the differential for hypotension and allowing for rapid and appropriate treatment selection.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.case.2022.04.015>.

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