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Case Report

Anchoring Bias: A Cautionary Tale of Point-of-Care Ultrasound and Cardiac Tamponade

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INTRODUCTION

Cardiac tamponade is a hemodynamic condition where the accumulation of pericardial contents, typically a pericardial effusion, leads to significantly increased intrapericardial pressure. This pressure decreases diastolic filling and ultimately progresses to a life-threatening decrease in cardiac output.[1] Electrocardiographic abnormalities may include reduced voltage, sinus tachycardia, and electrical alternans (alternating QRS voltage). A chest x-ray may show an enlarged cardiac silhouette.[2] When there is high clinical suspicion for cardiac tamponade, echocardiography is the primary diagnostic modality for initial evaluation.[2,3]

Prompt diagnosis is crucial for improved outcomes in cardiac tamponade due to the rapid progression of hemodynamic compromise. Adding a bedside ultrasound protocol to standard care allows for a rapid evaluation that can enhance the assessment of differential diagnoses.[4] The bedside point-of-care ultrasound (POCUS) exam is a valuable adjunct for diagnosing tamponade. However, it is essential to recognize that while POCUS is quick and non-invasive, it also can leave clinicians vulnerable to cognitive bias and diagnostic errors.[5]

CASE REPORT

A 51-year-old female weighing 44 kg with a three-week history of congestion, cough, and dyspnea presented to the emergency department (ED) due to worsening symptoms and lethargy. Her past medical history included end-stage renal disease on hemodialysis (HD), cerebral vascular accident, deep vein thrombosis with subsequent pulmonary embolism and inferior vena cava filter placement, chronic pericardial effusions with pericardial windows, aortic insufficiency, regular tobacco use, and warfarin use. She had missed HD two days prior due to malaise but underwent HD the following day.

During transport to the ED via emergency medical services, she received a 1 L fluid bolus. On initial assessment in the ED, she was hypoxic, hypotensive, and drowsy but oriented. Fluid overload from missed dialysis was suspected as the cause of her presentation. Nephrology was consulted and arranged for HD and/or continuous renal replacement therapy. An electrocardiogram showed a low voltage QRS, prompting the ED physician to perform a POCUS, revealing a large pericardial effusion without tamponade. Cardiology was consulted and ordered a stat transthoracic echocardiogram, which confirmed no tamponade physiology. Cardiothoracic surgery was consulted, and a computed tomography (CT) thorax without contrast was ordered for operative planning. The hospitalist admitted the patient to the intensive care unit.

Sepsis was considered as a potential cause for her hypotension, so blood cultures were obtained, and she was started on broad-spectrum antibiotics and a norepinephrine drip. Another 1000 mL fluid bolus was given. A chest x-ray indicated right upper lobe consolidation, suggesting an infectious infiltrate or possibly a central obstructing mass. The subsequent CT thorax scan identified pneumonia as the likely cause, ruling out an endobronchial obstructing lesion, and noted cardiomegaly with a large pericardial effusion, recommending cardiothoracic surgery consultation for a pericardial window.

Shortly after the CT scan, upon returning to her ED room, the patient became cyanotic, unresponsive, apneic, and bradycardic. Cardiopulmonary resuscitation (CPR) was initiated, and the ED physician performed a blind pericardiocentesis during the first pulse check, obtaining 25 mL of fluid. CPR resumed, the patient was intubated, and return of spontaneous circulation was achieved after 10 minutes. An emergent pericardial window in the operating room was then planned.

The anesthesiology team, including two critical care-trained anesthesiologists, evaluated the patient and performed a POCUS exam, showing a persistent massive pericardial effusion without tamponade. To stabilize the patient for transport to the operating room, a pericardiocentesis under ultrasound guidance was performed, removing approximately 75 mL of fluid without significant hemodynamic improvement.

The patient underwent a pericardial window via a mini thoracotomy through the fifth intercostal space on the left. During the procedure, she experienced asystole, responsive to direct cardiac compression. Intraoperative transesophageal echocardiogram revealed left ventricular hypokinesis with an ejection fraction of <20%, severe tricuspid regurgitation, severe pulmonary hypertension, sustained bowing of the intra-atrial septum into the left atrium, and a concentric pericardial effusion. Upon opening the pericardium, 200 mL of fluid were removed.

Following surgery, the patient was transferred to the intensive care unit, intubated, and on high doses of multiple inotropes. Despite these efforts, her neurological status did not improve. Goals of care were discussed with her family, and the decision was made to designate her as "Do-Not-Resuscitate." She passed away 14 hours after initial presentation. Initial blood cultures taken in the ED later returned positive, indicating bacterial sepsis secondary to pneumonia, which may have contributed to her outcome.

DISCUSSION

Guidelines from the American Heart Association, American College of Cardiology, American Society of Echocardiography, and European Society of Cardiology label echocardiography as the first-level diagnostic tool in the evaluation of pericardial pathology.[6] It is essential for physicians to understand the physiologic and echocardiographic distinctions between a large pericardial effusion and cardiac tamponade, as each diagnoses will lead to vastly different paths of clinical management and acuity.

The intrapericardial pressure (IPP) is proportional to the pericardial fluid volume and the stiffness of the pericardial sac – the latter otherwise can be described as inversely proportional to the compliance.[5] Normal IPP is lower than normal intracardial pressures; under normal circumstances, IPP does not exert influence on cardiac filling. Tamponade physiology will be seen when the IPP does impact cardiac filling. In other words, the detrimental hemodynamic effects of a pericardial effusion will be exerted due to increased IPP's, which depends on the rate of rise of the effusion and pericardial compliance. It is not necessarily impacted by the size of the effusion.[5,7] The pericardial sac can stretch to accommodate for an increase in intrapericardial fluid, but this increase in pericardial compliance will occur gradually.[3] The aforementioned physiology is the foundation for understanding the echocardiographic findings.

The earliest echocardiographic sign of tamponade is right atrial collapse during end-diastole and beginning of systole. It also is referred to as right atrial inversion or invagination (RAI).[3,5] Of all the chambers of the heart during the cardiac cycle, the lowest pressure is found in the right atrium during systole, approximately 3 - 5 mmHg.[6] The invagination of the right atrial wall is a passive response to the relative pressure on each side of the wall.[8] As IPP increases and exceeds right atrial pressure, the wall will collapse. Although echocardiographic sensitivity and specificity vary for RAI as a sign of tamponade, their values increase as the RAI increases in duration, especially when it is greater than one-third.[5,6,8] The optimal views for RAI visualization are the subxiphoid long-axis view and the apical four-chamber view.[5,6] In our case, the basic POCUS exam was looking for obvious pathology that might have been treatable via needle or medication; valvular pathology and gradients were formally assessed in the operating room with transesophageal echocardiography.

The echocardiographic sign of tamponade that carries the highest specificity is diastolic right ventricular collapse, also referred to as right ventricular inversion (RVI). The severity of tamponade correlates to the duration of RVI.[5] This finding follows the same physiological principle previously mentioned: chamber collapse simply reflects the relation of IPP to intracardiac pressures. The presence of RAI and RVI are dependent on intrinsic right heart pressures, and these signs may be absent in conditions such as pulmonary hypertension and tricuspid regurgitation.[7] The lack of any right-sided chamber collapse carries a 90% negative predictive value.[3]

The echocardiographic views for visualizing RVI are the apical four-chamber view, subxiphoid long-axis view, and the parasternal long-axis view[5]; in the parasternal long-axis view, RVI can specifically be appreciated with M-mode echocardiography, as it shows the most compliant right ventricle outflow tract.[7] Respirophasic variations in inferior vena cava diameter and mitral and tricuspid inflow velocities serve as surrogate measurements for assessing cardiac tamponade with the utilization of echocardiography. Although outside of the scope of this article, there is a review[6] that addresses this topic in-depth.

Septic shock and sepsis-related cardiogenic shock diagnoses also can be supported through ultrasound. Sepsis-related cardiogenic shock reports moderate depression in the left ventricular systolic function and normal left ventricular end-diastolic volume.[9] Using ultrasound for diagnosing septic shock allows for identifying characteristics of pleural effusion and the type of effusion based on the echogenicity pattern, or septa or empyema.[10] In our case, we could not rule out septic shock as a diagnosis because of the bacterial sepsis secondary to pneumonia that was identified after the patient expired. Additionally, the patient suffered cardiac arrest with return of spontaneous circulation after initial pericardiocentesis by the ED provider. Before a different diagnosis could be identified, and due to the emergent nature of the case, the patient was sent to the operating room for the pericardial winder via mini thoracotomy.

Just as clinical signs and symptoms do not serve as sole diagnostic indicators for cardiac tamponade, neither do ultrasonographic findings. As with all aspects of medical decision making, the physician incorporates clinical suspicion, maintains an evolving differential diagnosis, and incorporates all diagnostic measurements. While POCUS can serve as a powerful adjunct to the clinical examination, it should not be used as a substitute for or as equivalent to a comprehensive echocardiogram.[11] The cardiac POCUS exam has many protocols established, and it is intended to allow for rapid evaluation of reversible causes of shock, improve accuracy of diagnosis, and condense a differential diagnosis.[12]

One unique characteristic of this case report is that the patient underwent three variations of treatments for a pericardial effusion within a two-hour period. The patient underwent a blind pericardiocentesis, an ultrasound-guided pericardiocentesis, and a surgical pericardial window (a partial pericardiectomy) – none of which demonstrated a significant improvement in the patient's clinical status. While ultrasound-guided pericardiocentesis procedural methodology is beyond the scope of this article, Flint and Siegel[13] and Hatch et al.[14] go into a systematic, stepwise description.

This case report represents an example of anchoring bias – the tendency to place undue focus on a case's starting point, without adequate adjustment for new information. Anchoring bias occurs when there is not an adjustment to a differential diagnosis as new data emerges, possibly contradicting the initial presumptive diagnosis.[15] Despite the POCUS exams lacking evidence of tamponade physiology, and despite lack of improvement in hemodynamic stability post-procedures, the working diagnosis remained as shock secondary to cardiac tamponade. A large pericardial effusion in conjunction with a patient presenting in shock does warrant cardiac tamponade on a differential; however, lack of improvement following appropriate treatment should prompt re-evaluation of the diagnosis. Additionally, because the patient had a history of chronic pericardial effusions, anchoring bias may have played a large part in the treatment route. In this case, there were interdisciplinary discussions about whether the clinical picture and cardiac arrest were secondary to cardiac tamponade.

POCUS has gained wide acceptance among acute care physicians, as it facilitates the rapid diagnosis of several life-threatening conditions, potentially leading to changes in clinical decision-making.[11] In

this era of POCUS-driven care, it is imperative to remain cautious of anchoring bias to avoid incorrectly narrowing a differential diagnosis.

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