A 73-Year-Old Woman With Pulseless Electrical Activity Arrest

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A 73-year-old woman with a history of heart failure with preserved ejection fraction, severe pulmonary hypertension (World Health Organization class 2), and chronic kidney disease stage 3 presented to the ED after 2 days of weakness and shortness of breath. Her BP was 70/40 mm Hg; oxygen saturation was 84% on room air; respiratory rate was 30 breaths per minute, and heart rate was 73 beats per minute. On examination, she was mentating well but was in mild distress. She had rales at both lung bases and significant pitting edema in the lower extremities. Laboratory results were remarkable for BUN 56 mg/dL, creatinine 2.41 mg/dL (from baseline creatinine 1.8 mg/dL,) and pro-brain natriuretic peptide >15,000 pg/mL; chest radiography revealed bilateral pleural effusions. She was admitted to the ICU and found to have new right ventricular (RV) failure for which she underwent aggressive diuresis, transiently requiring inotropic support. Her ICU course was complicated by new onset atrial fibrillation for which she was started on apixaban 5 mg bid and rate control. With diuresis, her hemodynamics improved as did her renal function, and she was transferred to the hospital ward. Repeat echocardiogram demonstrated chronic diastolic dysfunction, RV failure, pulmonary hypertension (right ventricular systolic pressure, 78.7 mm Hg), and no pericardial effusion. While maintaining negative fluid balance with oral furosemide, she experienced an acute kidney injury that was attributed to prerenal azotemia from over-diuresis. Diuretics were held, but renal function worsened to a BUN of 116 mg/dL and creatine of 4.65 mg/dL over 1 week. She experienced accompanying symptoms of nausea and vomiting; plans were made for initiation of hemodialysis to treat uremia. The morning that was planned for elective placement of hemodialysis catheter, she experienced ventricular tachycardia followed by pulseless electrical activity (PEA) arrest. Chest compressions were started; advanced cardiac life support was initiated, and the patient was intubated. A point-of-care ultrasound scan (POCUS) of her heart was performed (Videos 1, 2).

Question A: What can be determined from the ultrasound image, and what is the next step?

Question B: Would you give any medications at this point?
Answer A: The patient experienced a large pericardial effusion with cardiac tamponade resulting in PEA arrest. Typical 2-dimensional echocardiographic findings of tamponade include large quantity of pericardial effusion, right atrial systolic collapse, RV diastolic collapse, and inferior vena cava (IVC) plethora. In the POCUS clips (Videos 1, 2), a large circumferential pericardial effusion is seen with 3 cm of echo-free space anterior to the RV. The chest compressions simulate systole, and the time in between compressions simulates diastole. There is evidence of right atrial collapse during compressions, likely corresponding to late diastolic and early systolic collapse. Video 2 shows cardiac standstill during a “pulse check.” Small bubbles that are seen in the right atrial and RV in both Video 1 and 2 are from IV administration of advanced cardiac life support medications.

At this point, we established the diagnosis of cardiac tamponade and proceeded with urgent ultrasound-guided pericardiocentesis using a 6-F catheter drainage system that was available readily on the hospital ward. This kit is more frequently used for paracentesis and thoracentesis. Manual aspiration of bloody-appearing pericardial fluid resulted in return of spontaneous circulation (Video 3); 500 mL of pericardial fluid was drained immediately. A saline solution flush test was performed to confirm pericardial placement of this pigtail catheter (Video 4). The pigtail drain was left in place to gravity and drained another 600 mL of fluid with resolution of pericardial effusion (Video 5). Fluid cell count eventually resulted as 1,594,000 RBCs and 3,690 WBCs.

Answer B: The patient had been receiving apixaban 5 mg bid for anticoagulation to prevent the risk of stroke secondary to atrial fibrillation. Given the grossly bloody appearance of pericardial fluid, it is appropriate to consider reversal with adexanet alfa. The patient was given a 400 mg IV bolus followed by a 480 mg infusion of adexanet alfa.

Discussion
With the increasing availability of POCUS, any clinical suspicion of cardiac tamponade should be evaluated quickly. In fact, pericardial effusion leading to tamponade has been described as a “never event” for hospitalized patients. POCUS is particularly advantageous, given that the clinical signs and symptoms or tamponade lack sensitivity and specificity.

The visceral pericardium covers the entire external surface of the heart and can be visualized as a thin echogenic layer on ultrasound scanning. A pericardial effusion typically appears as an anechoic (black) area between the visceral pericardium and parietal pericardium. Complex or loculated effusions might have internal echoes and a more heterogenous appearance.

The likelihood of the development of tamponade is not determined strictly by the size of a pericardial effusion, but instead by compliance of the pericardium and acuity of fluid accumulation. In the patient described here, a cardiology-performed echocardiogram 1 week before her cardiac arrest did not have an effusion, which highlights that rapid fluid accumulation can lead to the development of tamponade physiologic condition.

After the establishment of the presence of a pericardial effusion with the use of POCUS, additional findings can raise suspicion for tamponade physiologic condition, which include right atrial systolic collapse, RV diastolic collapse, and a dilated IVC without respiratory variation. However, right atrial collapse can occur at any point throughout the cardiac cycle. If the duration of right atrial collapse exceeds one-third of the cardiac cycle, the specificity increases. RV collapse typically occurs in diastole and has a lower sensitivity (48% to 60%) but higher specificity (75% to 90%) compared with right atrial collapse. In tamponade physiologic condition, the RV pressure is lowest in early diastole when the intrapericardial pressure exceeds the RV pressure, which leads to diastolic collapse. The duration of the RV collapse is also correlated with the severity of tamponade. If RV filling pressures are elevated at baseline, diastolic collapse is less likely to occur, such as in the case of acute or chronic pulmonary hypertension, severe left ventricular failure or any other cause of RV hypertrophy. Conversely, diastolic RV collapse may occur earlier if the RV filling pressure is lower, such as with hypovolemia. Advanced POCUS users who are trained in pulsed wave Doppler may also be able to assess mitral and tricuspid
valve inflow velocity variation with the respiratory cycle, a Doppler surrogate of pulsus paradoxus. The finding of a pericardial effusion with the use of POCUS should prompt urgent cardiology-performed echocardiogram to determine whether echocardiographic features of tamponade are present, including respiratory variation in mitral and tricuspid inflow velocities with the use of pulse wave Doppler. In patients in stable condition, the decision to pursue elective drainage of a pericardial effusion and timing of drainage should involve expert consultation that takes into account clinical features that include the likely cause of pericardial effusion; some causes are more likely to progress to tamponade (eg, malignant effusions and those due to renal failure). However, in the instance of a pericardial effusion causing tamponade as the likely explanation of a PEA arrest, pericardiocentesis should be performed emergently with a needle with syringe or any available drainage equipment.

In the case described earlier, emergency pericardiocentesis proved to be a lifesaving intervention. Urgent hemodialysis was initiated, and pericardial effusion did not recur after adexanet alfa infusion. The patient was extubated and later discharged from the hospital neurologically intact with plans for long-term outpatient hemodialysis. Spontaneous hemorrhagic pericardial effusion has been reported previously after the initiation of factor Xa inhibitors. In this case, hemorrhagic conversion of uremic pericarditis while undergoing anticoagulation is also a possibility. Although the efficacy of adexanet alfa in the pericardial bleeding subgroup was not specifically reported in the ANNEXA-4 study, subjects with pericardial bleeding were included in this study. Novel reversal agents should be considered in hemorrhagic pericardial effusion that occurs while receiving factor Xa inhibitor therapy, especially if tamponade physiologic condition or cardiac arrest is present.

Reverberations
1. POCUS can be used to identify the presence of a pericardial effusion rapidly. Users should learn to identify findings that are concerning for tamponade physiologic condition in B-mode, such as right-sided chamber collapse and a dilated IVC without significant respiratory variation.
2. Urgent ultrasound-guided pericardiocentesis should be performed for patients with pericardial effusion who experience hemodynamic instability or cardiac arrest, with the use of any immediately available drainage equipment.
3. Patients who receive factor Xa inhibitors who experience the development of a hemorrhagic pericardial effusion with tamponade might benefit from new reversal agents.

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Additional information: To analyze this case with the videos, see the online version of this article.

References