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Case Report

A Window in the Heart Is Sometimes a Good Start: It's Not Always Sepsis

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ABSTRACT

Cardiac tamponade is a life-threatening compression of the heart caused by abnormal accumulation of pericardial fluid. Important elements affecting its disposition and treatment are the rate of fluid accumulation relative to pericardial stretch and the effectiveness of compensatory mechanisms before critical hemodynamic compromise occurs. It is a clinical diagnosis and waiting for the threshold of steep rise in cardiac transmural pressure to critical levels may lead to catastrophic outcomes which is why early drainage has to be strongly considered in suspected cases.

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Case Report

A 75-year-old man with a medical history of coronary artery disease with previous percutaneous intervention, pulmonary embolism receiving rivaroxaban and rheumatoid arthritis on chronic corticosteroid suppression, presented with chest pain. He was hypoxemic, tachycardic and hypotensive on arrival. Pulsus paradoxus was not present.

His electrocardiogram showed diffuse ST segment elevations, PR interval depressions and low voltage QRS morphology, consistent with acute pericarditis. Tamponade was a concern at this point and a bedside echocardiogram was obtained which showed a hyperdynamic left ventricle (LV) with cavity obliteration, moderate circumferential pericardial effusion, and a decrease of >25% in mitral E wave velocities

during inspiration (Figure 1). He was initially evaluated by the Cardiac Care Unit service and deemed to have probable sepsis from unknown source, without urgent need for pericardiocentesis, due to the absence of right atrial (RA) and right ventricular (RV) collapse on imaging; he was admitted to the Medicine Intensive Care Unit (MICU). He was treated along the lines of sepsis and pericarditis with high dose aspirin, intravenous fluid resuscitation, empiric antibiotics and corticosteroids without major improvement. After 24 hours, he developed cardiogenic shock, acute kidney injury and hypoxemic respiratory failure caused by pulmonary edema. Further evaluation by cardiology showed that the effusion was bigger but still not causing RA/RV collapse. At this point, an urgent pericardial window was performed with intraoperative drainage of 300 mL of pericardial fluid, followed by rapid resolution of the shock and respiratory failure.

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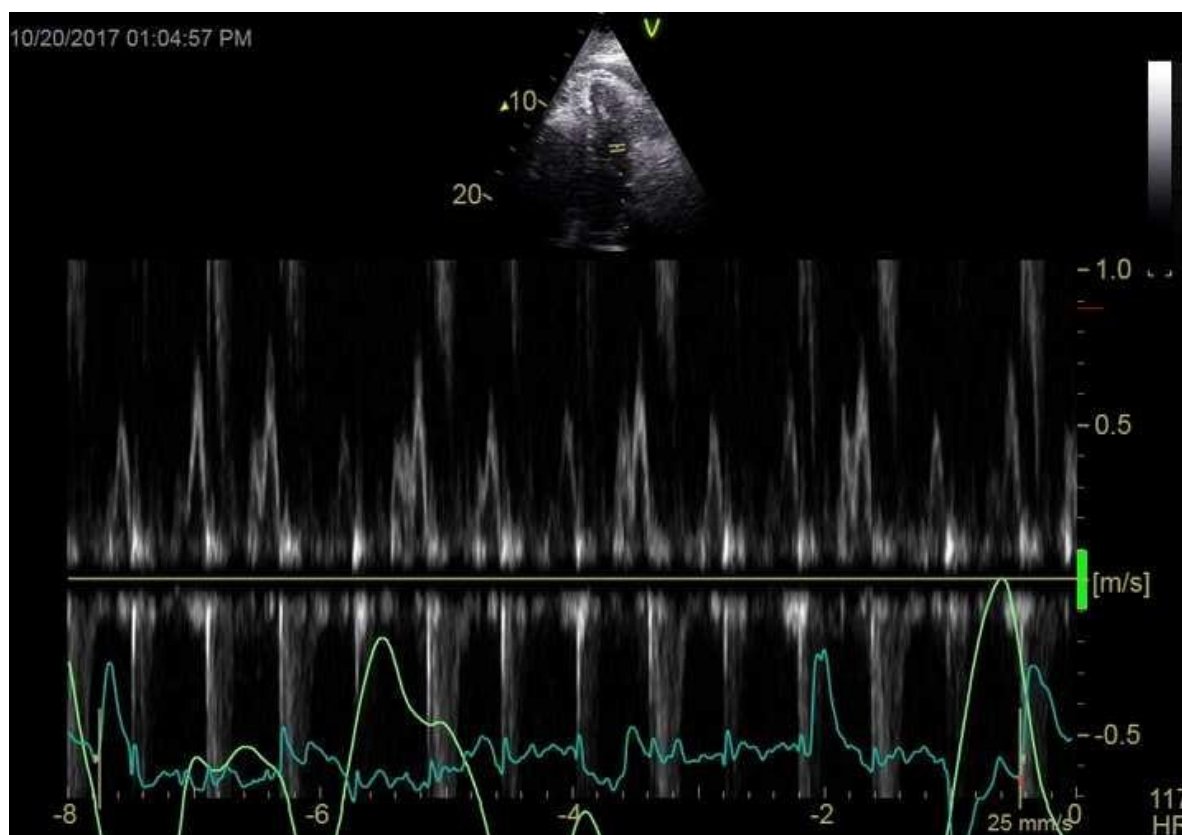


Figure 1: Echo with Doppler evaluation.

Discussion

The classic echocardiographic RA/RV collapse was not present in this case; however, the presence of LV obliteration and inspiratory variation of mitral flows is concerning for tamponade in the setting of hypotension despite fluid administration. A regional tamponade may have been present as well; the literature is nonspecific about LV obliteration representing this and is mostly limited to postoperative occurrences, which was not the case in our patient [1-4]. Pericardial tamponade is a clinical diagnosis and waiting for the threshold of steep rise in cardiac transmural pressure to critical levels may lead to catastrophic outcomes, as witnessed by the marked improvement in this patient's hemodynamics occurring after the surgical intervention. Early pericardial fluid drainage should be considered in cases similar to ours.

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