

CLINICAL VIGNETTE

A Case of Dyspnea and Distant Heart Sounds

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Case Report

A 74-year-old male presented for an outpatient cardiology consultation for evaluation of chest pain, shortness of breath and leg swelling. His past medical history includes hypertension and squamous cell cancer of the tongue status post resection and radiation therapy in the mid 1990s. Two weeks prior, he noticed an episode of chest tightness and dyspnea walking uphill on his usual 3-mile route, which resolved quickly with level walking. He also noted increasing lower extremity edema, which worsened over the day, and resolved overnight. He noted only one other episode of dyspnea while walking up a hill during a golf game. He denied orthopnea, paroxysmal nocturnal dyspnea, palpitations, lightheadedness or syncope. He also denied nausea, vomiting, diaphoresis, fevers, chills, night sweats, cough or recent travel.

The patient reported a mechanical fall three months before when he tripped in the bathroom and hit his chest against the sink before falling to the ground. He did not suffer any fractures or head trauma but recalled significant bruising on his chest and abdominal walls following the fall.

His outpatient medications include irbesartan 150mg daily, furosemide 20mg daily, and finasteride 5mg daily. His regular physician, who had stopped his amlodipine 1-week prior, saw him. He has no allergies. He has no significant family history. He has a 30-40-pack year history of smoking but quit in 1982. He is married and works as a court reporter.

Physical Examination

On physical exam he was afebrile with a blood pressure of 109/78, heart rate of 86, respiratory rate of 12, and oxygen saturation of 98% on room air. JVP was 12 cm with Pulsus of 6. Heart sounds were faint with a soft 1/6 early peaking crescendo-decrescendo murmur heard best at the right upper sternal border without radiation to the carotids. There were no rubs or gallops. Decreased breath sounds were noted with dullness to percussion at the left base

below the scapula. Lung sounds were otherwise clear. The liver edge was palpable 1cm below the costal margin, but nonpulsatile. Extremities were warm and well perfused with 2+ pitting edema to just below the knees.

An EKG revealed normal sinus rhythm @78 and low voltage when compared to prior EKG several weeks earlier. An echocardiogram revealed a large circumferential pericardial effusion with right atrial and ventricular invagination, evidence of respiratory variation of mitral inflow pattern, and dilated inferior vena cava. LV ejection fraction was normal at 60-65% and he had evidence of mild aortic stenosis with a calculated valve area of 1.9 cm^2 . He was admitted to the hospital for pericardiocentesis and 860 cc's of bloody fluid was drawn from the pericardial space. Studies on the fluid revealed: 2,305,000 RBC, 1500 WBC with a normal differential. Adenosine deaminase, Gram stain and culture were negative. Cytology was negative for malignant cells and revealed only a few lymphocytes, reactive mesothelial cells, and histiocytes. CT scans of head, neck, chest, abdomen, and pelvis did not reveal any source of neoplasm.

Is this cardiac tamponade? Tamponade is mainly a clinical diagnosis classically consisting of the triad of hypotension, soft or absent heart sounds, and jugular venous distention with a prominent x descent and blunted y descent (a trio of findings known as Beck's triad). Our patient has no signs of hemodynamic compromise (no tachycardia, hypotension, or pulsus paradoxus) but did have distant heart sounds and elevated jugular venous pressures. Echocardiographic signs pointing to tamponade include diastolic RA and RV collapse (when the intrapericardial pressure exceeds that inside the lower pressure right-sided chambers), respiratory variation in mitral and tricuspid inflow patterns (an exaggeration of normal respiratory variations signifying interventricular dependence), and IVC plethora (a dilated IVC with reduced collapse with inspiration). His echocardiogram raised concern for tamponade, but clinically he had more of a subacute picture of an evolving large pericardial effusion without tamponade.

The major causes of pericardial effusion depend on the type of effusion (hemorrhagic versus serous) and also vary by patient demographics and geographic region. In this case the effusion was clearly hemorrhagic based on the results of the pericardiocentesis, and the most common causes would be iatrogenic (secondary to invasive cardiac procedures), malignancy (most commonly lung cancer followed by breast, esophageal, melanoma, leukemia, and lymphoma), complication after an acute myocardial infarction, post-cardiac surgery, tuberculous pericarditis and other infections, uremia, dissection and trauma¹⁻⁴. In our case, the patient's remote history of malignancy raises that to the top of the list, especially since he is not from a TB endemic area, but his fluid cytology was negative and CT scans did not suggest any other malignancy. In larger effusions such as in this case, consideration should be given to connective tissue disease and thyroid disease, although these generally cause serous effusions rather than hemorrhagic. Furthermore, the TSH and connective tissue disease markers returned normal. Radiation induced pericardial effusions are possible, but those, too, are usually serous and require radiation to the chest, whereas his radiation was to the face and neck. He had no recent viral illnesses or other signs of infection and an adenosine deaminase level in the pericardial fluid was normal, ruling out TB. He did not have any evidence of an MI, nor any recent cardiac procedures ruling out those as possible etiologies as well.

So, what are we left with as the potential cause for his effusion? His prior chest wall blunt trauma is interesting, but occurred about 3 months prior to his presentation. In the literature, pericarditis and hemorrhagic pericardial effusions usually occur within weeks after the trauma^{5,6}. However, there have been reports of patients with blunt chest wall trauma presenting with pericardial effusions up to 105 days out from the inciting traumatic event^{7,8}. Our patient is clearly within that window raising the possibility of delayed presentation of hemorrhagic pericardial effusion from his prior blunt trauma sustained during his fall 3 months prior.

While this patient did not have a definitive diagnosis, the case provided a great review of the critical clinical and echocardiographic features in tamponade in addition to the differential diagnosis for hemorrhagic pericardial effusion.

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