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Beyond Volume Overload: Unmasking a Silent Pericardial Effusion in End-Stage Renal Disease

Carlee DeFrancesco[1]*, & Falguni Kalra[2]

[1]Touro College of Osteopathic Medicine – Middletown Graduation Year: 2026, USA.
[2]Sound Physicians / Montefiore Nyack Hospital, USA

***Corresponding author:** Carlee DeFrancesco, Touro College of Osteopathic Medicine – Middletown Graduation Year: 2026, USA.*

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Abstract

Pericardial effusion is a potentially fatal condition with varied presentations, often obscured in patients with complex medical histories. We report a patient with ESRD on dialysis, AML, and CHF who presented with hypoxia and dyspnea, initially presumed to be in CHF exacerbation. However, clear lung fields and lack of edema prompted further evaluation. An elevated D-dimer led to CT angiography, revealing a large pericardial effusion. After platelet transfusion, urgent pericardiocentesis drained 800 cc of hemorrhagic fluid. Despite intervention, the patient deteriorated and passed away. This case underscores the need for diagnostic vigilance in patients with multiple comorbidities.

Keywords: Pericardial Effusion, End-stage Renal Disease (ESRD), Cardiac Tamponade, Congestive Heart Failure (CHF), Diagnostic Anchoring Bias

Background

Pericardial effusion is an acute or chronic accumulation of extra fluid in the pericardial space. It is caused by a myriad of pathological conditions including but not limited to: infection, inflammation, or a defective myocardium leading to sanguineous filling caused by trauma or iatrogenic reasons. The presentation of a pericardial effusion is commonly asymptomatic initially in chronic effusions due to the ability of the pericardium to stretch and accommodate slightly more fluid. However, in an acute setting, the fluid quickly expands the pericardium, impairing the heart's ability to contract effectively resulting in tamponade. This presents very rapidly with hypotension, tachycardia, jugular venous congestion, and pulsus paradoxus. In cases like this, urgent decompression via pericardiocentesis is the preferred way to treat unstable patients. In stable patients, treatment focuses on diagnosing and resolving the underlying cause of the effusion.

Objective

To reflect upon anchoring bias and how it can delay recognition of pericardial effusion in patients with overlapping cardiac and renal comorbidities, and to highlight the need for thorough re-evaluation when clinical findings are not representative of the presumed diagnosis.

Case Presentation

Patient History and Presentation

An 83-year-old male with a history of acute myeloid leukemia (AML), immune thrombocytopenic purpura (ITP), end-stage renal disease (ESRD) on dialysis, congestive heart failure (CHF), hypertension (HTN), chronic obstructive pulmonary disease (COPD), gastroesophageal reflux disease (GERD), and benign prostatic hyperplasia (BPH) presented to the emergency department with progressive dyspnea and chest discomfort for two weeks with no aggravating or alleviating factors [1].

Vital Signs on Presentation

- BP: 123/64 mmHg
- HR: 81 bpm
- Temp: 98.8 °F (37.1 °C) Oral
- Respiratory rate: 16 breaths/min

● SpO_2: 97% on 2L nasal cannula

Physical Examination

● General: Well-developed, in no acute distress
● Pulmonary: Mild expiratory wheezes, no rales or rhonchi, breath sounds present bilaterally
● Cardiovascular: Regular rate and rhythm, no murmurs/gallop/rub
● Abdomen: Soft, nondistended, nontender
● Neurological: Alert and oriented ×3, cranial nerves intact, moves limbs, sensory intact
● Skin: Warm and well-perfused
● No peripheral edema or jugular venous distention observed

Laboratory Results

● Hemoglobin: 6.8 g/dL
● Platelets: 9 ×10⁹/L
● BNP: 5883 pg/mL
● Potassium: 6.7 mmol/L
● Sodium: 128 mmol/L
● D-dimer: 3.23 µg/mL
● Troponin: <0.3 ng/mL

Imaging Findings

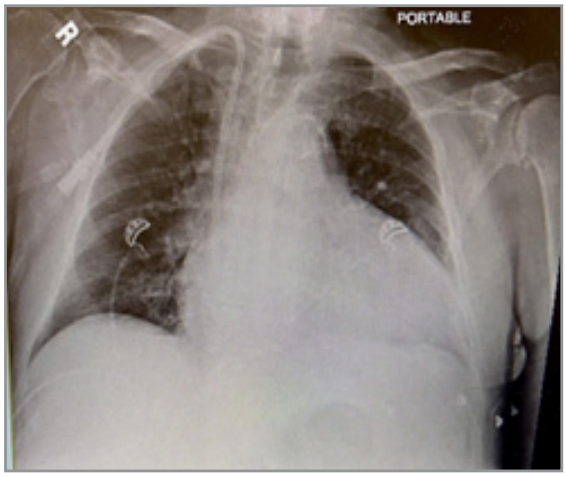

Figure 1: Initial Chest X-Ray: No pneumothorax or pleural effusion. No consolidation. Some pulmonary vascular congestion present.

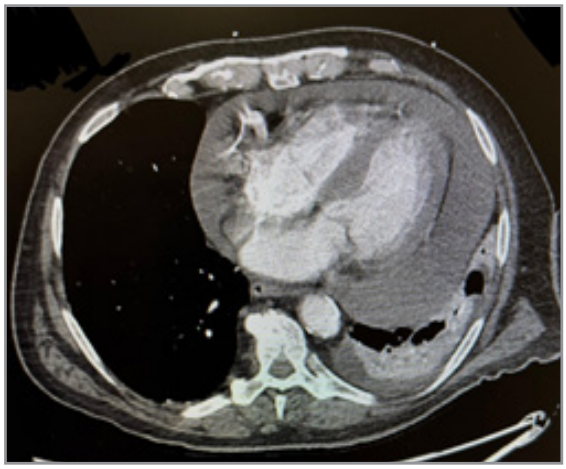

Figure 2: CT Angiogram Chest: No pulmonary emboli identified. Small left pleural effusion with no focal opacity. Large pericardial effusion.

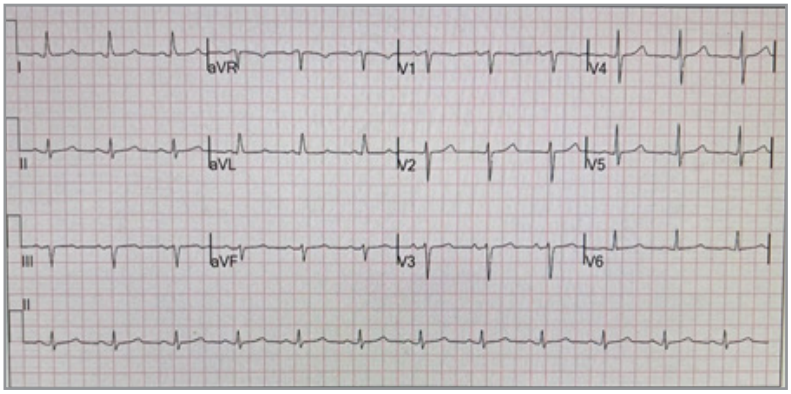

Figure 3: Initial EKG (ED): Sinus rhythm, prolonged QT interval 444. No evidence of ischemia.

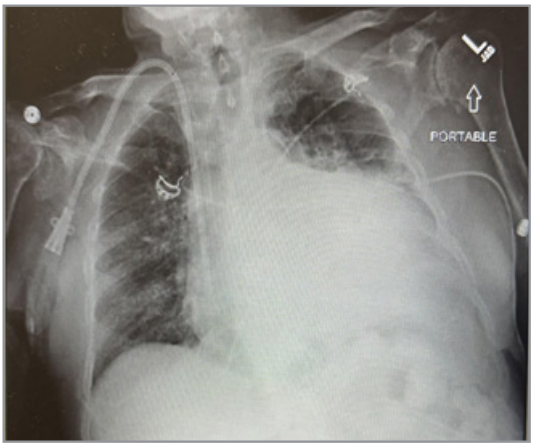

Figure 4: Chest X-Ray s/p Pericardial Drain: stable cardiomegaly with pericardial drain. Stable markings left suprahilar regions. No evidence of acute infiltrate.

Management and Outcome

Management of pericardial effusion hinges on the size and impact on cardiac function. While small, asymptomatic effusions may be monitored, large or symptomatic effusions—especially those causing hemodynamic compromise—require urgent pericardiocentesis.

In this case, due to the size of the effusion and the patient's symptoms, emergent drainage was performed in the catheterization lab. A total of 800cc of sanguineous fluid was removed, with continued drainage over the next 48 hours totaling an additional 300cc. Fluid was sent for cytological and microbiological analysis. The patient received multiple platelet and PRBC transfusions due to ongoing thrombocytopenia related to AML and ITP.

Despite aggressive management, the patient's condition worsened, and he was unable to maintain hemodynamic stability. After several days in the MICU, the patient succumbed to multisystem organ failure [2].

Discussion

This case illustrates a diagnostic pitfall that is common in clinical medicine: anchoring bias. The patient, with a complex history of ESRD on dialysis, CHF, and AML, presented with symptoms easily attributed to fluid overload—chiefly shortness of breath and hypoxia. In the setting of markedly elevated BNP and a known history of heart failure, the most probable clinical assumption was an acute CHF exacerbation. However, some key findings on physical examination, including clear lung fields, and absence of peripheral edema, suggested an alternative etiology and ultimately changed the diagnostic approach.

Pericardial effusion, though not uncommon in patients with ESRD or malignancy, is often overlooked in the acute setting due to its broad and sometimes subtle symptomatology. In ESRD, pericardial effusion may result from uremic pericarditis or volume overload, and is frequently chronic and asymptomatic. However, in this patient, the effusion was both large and symptomatic, most probably caused by hemorrhagic diathesis secondary to thrombocytopenia from AML and ITP. This combination was unique, and culminated in tamponade physiology, which was masked by the patient's baseline comorbidities.

The elevated D-dimer, although nonspecific, prompted a CTA to rule out pulmonary embolism, which furthermore revealed the large pericardial effusion. This finding was vital and underscores the value of maintaining a broad differential diagnosis. Anchoring bias may have delayed definitive diagnosis and management had the initial assumption of CHF not been questioned.

The absence of classic signs of tamponade, such as hypotension or overt jugular venous distention, further demonstrates how clinical presentation can be misleading, especially in elderly, medically complex patients.

This case serves as a crucial reminder of the limitations of pattern recognition when it is applied without critical appraisal. In patients with extensive medical histories, clinicians must be cautious not to default to the most "familiar" diagnosis without fully considering alternative possibilities. A comprehensive clinical examination, paired with careful attention to inconsistencies, can help avoid premature closure and ensure timely intervention in life- threatening conditions such as cardiac tamponade [3].

Conclusion

This case highlights the importance of avoiding diagnostic anchoring, particularly in patients with complex medical histories. While CHF was a reasonable initial assumption given the patient's history and symptoms, a careful physical exam and unexpected imaging findings led to the correct diagnosis. Clinicians must remain vigilant and maintain a broad differential to prevent delays in critical care interventions.

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