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## CLINICAL VIGNETTE

# Evaluation of Dyspnea associated with Cardiomegaly

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### *Clinical Vignette*

A 23-year-old male presented to the hospital with new-onset dyspnea. The patient initially had noticed dyspnea with minimal exertion five days prior to admission which had progressed to severe dyspnea at rest associated with intermittent pleuritic chest pain on the day of admission. Review of systems revealed new-onset two-pillow orthopnea, without lower extremity edema, syncope, fever or recent illness.

The patient had a history of polysubstance abuse including heroine and opiates, anxiety and bipolar disorder. His last intravenous heroine use was about three week prior to admission. His medication included gabapentin, quetiapine, and divalproex sodium. There was no

family history of cardiac disease or sudden cardiac death.

Upon admission, the patient was significantly tachypnic with a heart rate of 123, blood pressure of 105/70, with oxygen saturation of 98% on nasal cannula. His neck veins were distended. Cardiac auscultation revealed distant hearth sounds without any murmurs or rubs. There was a pulsus paradoxus of 15 mm Hg. Lung exam was clear to auscultation without rales or wheezing. He had no lower extremity edema.

Laboratory values were significant for a white count of 26,000, hemoglobin of 10.6 g/dl, and D-dimer of greater than 10,000. Urine screen for drugs of abuse was negative.

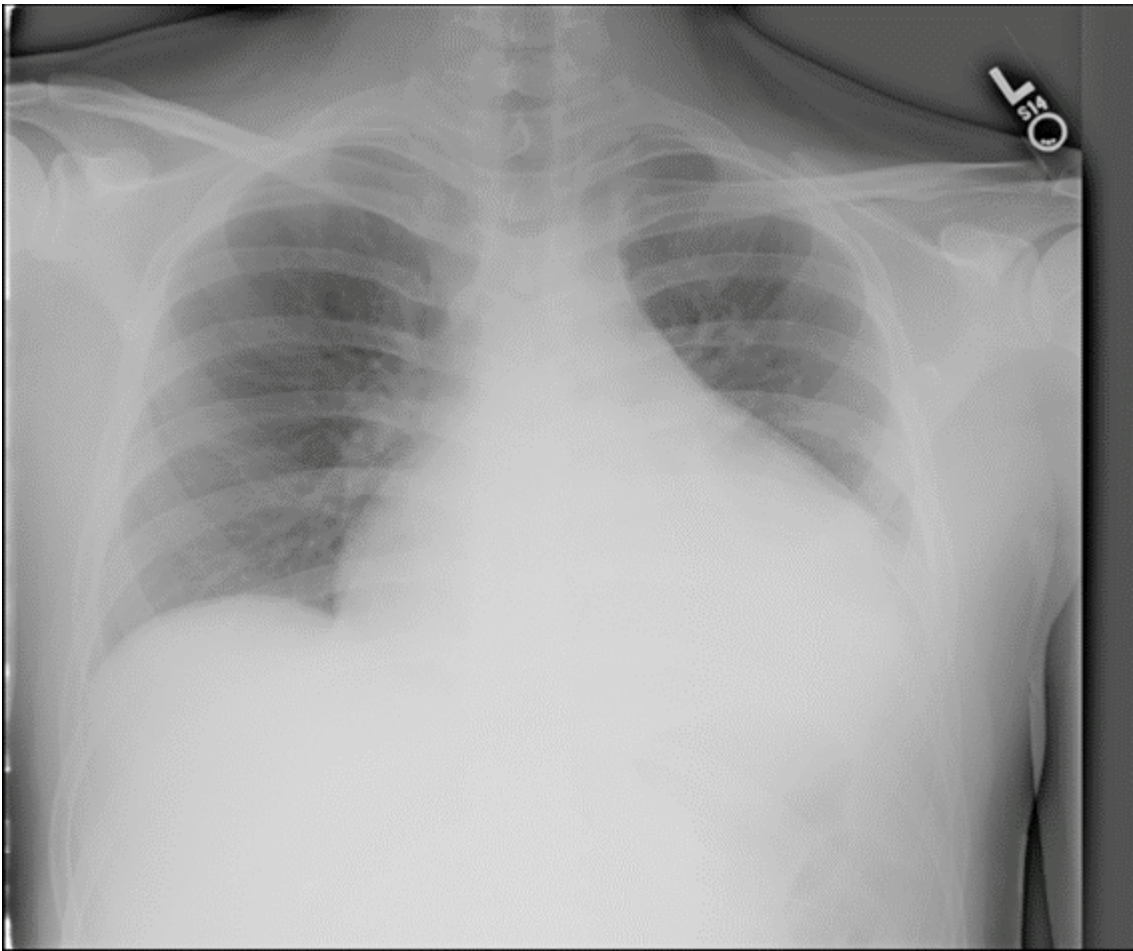


Figure 1

EKG revealed sinus tachycardia at the rate of 120 bpm and lateral T-wave inversions. Chest X-ray showed cardiomegaly with lingular atelectasis and trace pleural effusion without interstitial pulmonary edema or consolidation (figure 1). CT angiogram of chest showed no pulmonary embolism but evidence of a large pericardial effusion.

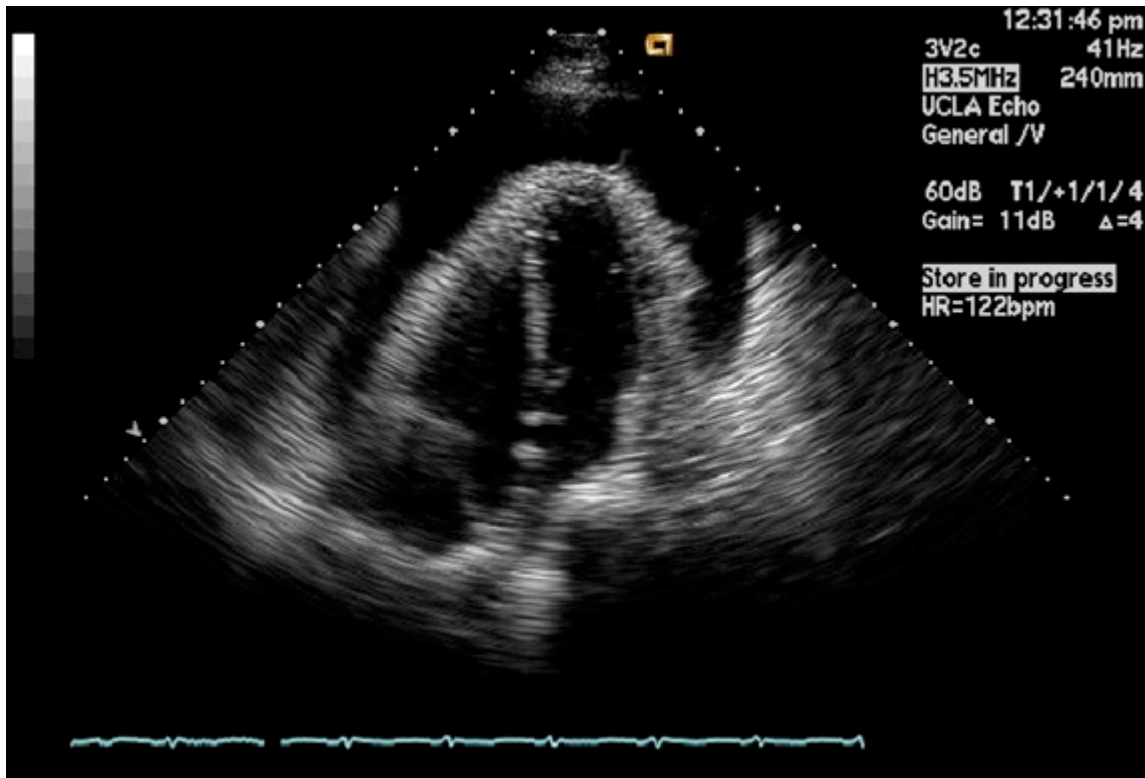


Figure 2

An Echocardiogram (figure 2) done in the ED showed a large antero-apical pericardial effusion. Transmitral flow showed 25% variation with respiration. The IVC measured 1.7 cm with poor respiratory variation. The patient underwent urgent pericardiocentesis and 850 ml of bloody fluid was removed with significant improvement of respiratory symptoms. His chest pain improved with administration of indomethacin. Infectious, rheumatologic and malignancy workup were unrevealing at the time of discharge.

Differential diagnosis of Acute dyspnea with cardiomegaly

*Congestive heart failure exacerbation*

Numerous conditions can lead to congestive heart failure with associated cardiomegaly.

Dilated cardiomyopathies (DCMP) are characterized by dilation and impaired contraction of one or both ventricles. Etiologies of DCMP are varied and include idiopathic, ischemic, familial, valvular, toxic, infiltrative and autoimmune causes<sup>1,2</sup>. Chest pain on exertion is seen in up to one-third of these patients. The patient's history of substance abuse raises the possibility of alcohol or cocaine-related DCMP though he denied cocaine or excessive alcohol use. In the absence of any murmurs valvular causes are unlikely. Infectious, autoimmune and familial etiologies are less likely in the absence of any other constitutional symptoms or family history.

Hypertrophic cardiomyopathy (HCM) is a genetic disease of the cardiac sarcomere and is characterized by hypertrophy of the left ventricle disproportionate to the hemodynamic load. Its clinical manifestations in symptomatic patients include dyspnea and angina with cardiomegaly on CXR<sup>3</sup>. The absence of a systolic murmur and evidence of LVH on EKG makes the diagnosis less likely.

Additionally, lack of interstitial pulmonary edema or pulmonary venous congestion on CXR suggests another cause for the patient's presentation.

#### *Pericardial Effusion and Cardiac tamponade*

Pericarditis and associated pericardial effusion can be caused by a number of disease processes including infectious, neoplastic, autoimmune, uremic, and idiopathic causes<sup>4</sup>. Effusion without pericarditis can be seen in nephrotic syndrome, cirrhosis and hypothyroidism. Cardiac tamponade is a form of cardiogenic shock, and the differential diagnosis may initially be elusive. Tachypnea and dyspnea on exertion that progresses to dyspnea at rest are the key symptoms. Most physical findings are nonspecific. Tachycardia (a heart rate of more than 100 beats per minute) is the rule. Exceptions include patients with bradycardia during uremia and patients with hypothyroidism. Heart sounds may be attenuated owing to the insulating effects of the pericardial fluid and to reduced cardiac function. A pericardial rub may be present especially in patients with inflammatory effusion<sup>5</sup>.

Clinically significant tamponade usually produces absolute or relative hypotension and jugular venous distention. A key diagnostic finding, pulsus paradoxus — conventionally defined as an inspiratory systolic fall in arterial pressure of 10 mm Hg or more during normal breathing — is the most specific physical finding<sup>6</sup>. Other conditions causing pulsus paradoxus include massive pulmonary embolism, profound hemorrhagic shock, other forms of severe hypotension, and obstructive lung disease.

An electrocardiogram may show signs of pericarditis, but the only quasispecific sign of tamponade is electrical alternans, which is alternation in electrical signals that may affect any or all electrocardiographic waves or only the QRS<sup>7</sup>.

Doppler echocardiography is the principal tool for diagnosing pericardial effusion and cardiac tamponade. Doppler study discloses marked respiratory variations in transvalvular flows. The inferior vena cava is dilated, with little or no change on respiration. Among echocardiographic signs, the most characteristic are chamber collapses, which are nearly always of the right atrium and ventricle<sup>8</sup>.

The treatment of cardiac tamponade is drainage of the pericardial contents, preferably by needle pericardiocentesis. Recurrences, especially in patients with malignant tamponade, may require balloon pericardiotomy or pericardial window, to allow the fluid to drain from the pericardium and the absorbing surface of the pleura or peritoneum<sup>9</sup>. Death in patients with tamponade is usually manifested by pulseless electrical activity.

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