Diagnose this

A 45-Year-Old Male with New Onset Shortness of Breath

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A 45 year-old male presented to the ED for the second time in one week, complaining of shortness of breath and low grade fever, taken at home with an oral thermometer. He had recently completed treatment for lymphoma. He denied chest pain, but admitted that he had noticed some new swelling in his legs. He was admitted with a diagnosis of presumed pneumonia and rapidly deteriorated, requiring intubation 12 hours following admission to the ICU. A portable anteroposterior (AP) chest x-ray was taken to check placement of this tubes and lines (Figure 1).

What is the most likely diagnosis?

A. Congestive heart failure
B. ARDS or shock lung
C. Lymphangitic carcinomatosis
D. Acute pulmonary hemorrhage
E. Bilateral pneumonia

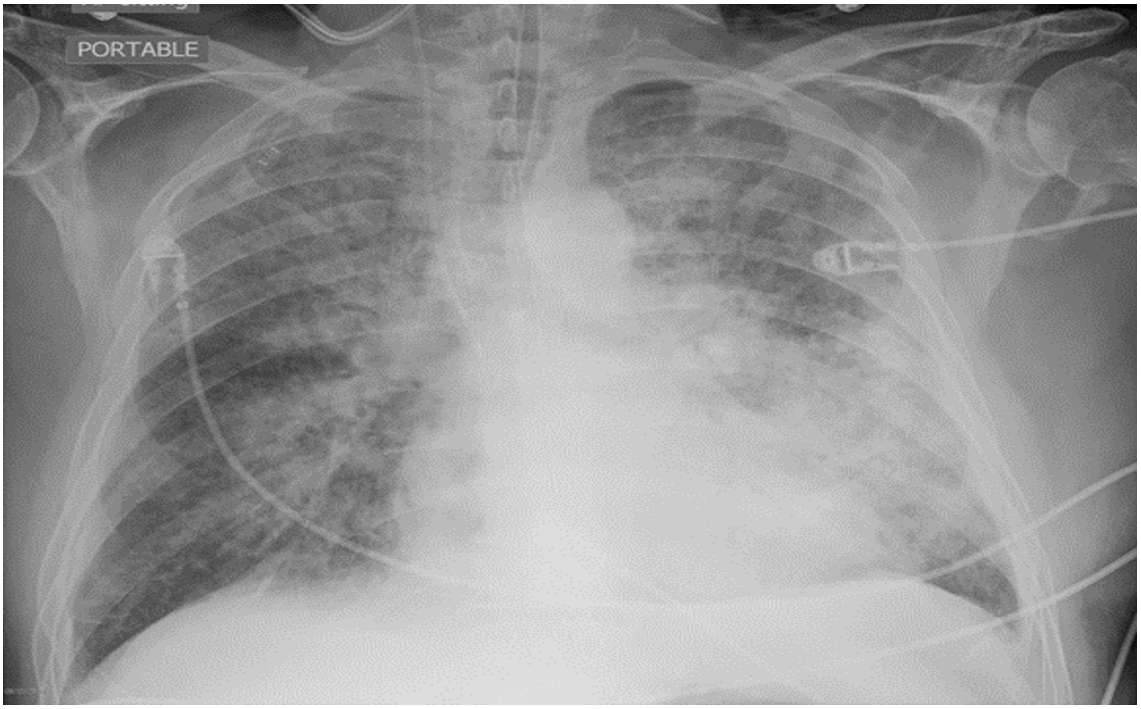

Figure 1. Anteriorposterior (AP) portable chest radiograph

Answer

The correct answer is "A".

ARDS would be incorrect as it classically presents as bilateral, peripheral airspace disease. It is not lymphangitic carcinomatosis as this disease classically causes thickened septal lines but airspace disease is absent. The answer is not an acute pulmonary hemorrhage as this may produce the airspace disease but typically lacks any interstitial findings. Finally, bilateral pneumonia is incorrect as the airspace disease is clearly unilateral and the interstitial findings are discordant.

Overview

Heart failure is characterized by an inadequacy of the heart to deliver oxygenated blood to meet the demands of tissues and organs.[1,2] Generally, the heart is quite proficient at adapting to short-term changes in preload and afterload. However, sudden or prolonged changes in demand, preload or afterload can lead to progressive failure of the myocardial function.[1] The Canadian Cardiovascular Society (CCS) suggests that a precipitating factor for acute heart failure should always be sought.[3] The diagnosis is based on the clinical presentation, including symptoms (orthopnea, dyspnea) and signs (respiratory crackles and pedal edema).[3,4] A 12-lead ECG and the use of a validated measure of the severity of symptoms, such as the New York Heart Association (NYHA) classification scale to indicate functional capacity, should be a component of every patient's work-up.[3,4]

Imaging Findings

The CCS and American Heart Association (AHA) recommend that a chest x-ray be performed for all patients who have suspected, new onset, or acute decompensated heart failure.[2,3] It is used to assess for cardiomegaly and pulmonary congestion, although cardiomegaly may be absent in heart failure. Signs of pulmonary congestion on an x-ray include pulmonary venous redistribution, symmetrical perihilar peribronchial cuffing and Kerley B lines in the early stages. Peribronchial cuffing is the result of interstitial fluid accumulating around the bronchi, which causes apparent thickening of the bronchial wall. Adjacent vessel walls may appear indistinct for the same reason. Kerley B lines reflect interstitial fluid accumulating in subpleural interlobular septa. Pleural effusions may also be seen. These findings may progress to bilateral perihilar airspace edema in a classic "batwing" configuration. The chest x-ray can also demonstrate or rule out other potential causes of the patient's symptoms. Chest x-ray has a moderate specificity of about 76-83% but has a low sensitivity of 67-68%.[6]

The gold standard test for evaluating heart failure is a transthoracic echocardiogram (TTE). TTE should be performed within 72 hours of presentation and is recommended as the primary investigation for cardiac structure and function.[3,5] It is the most frequently used imaging modality for heart failure and has well-established diagnostic and prognostic value. It is also quite cost-effective and widely available, which is important given the widespread prevalence of this disease.[5] Echocardiography is used to assess left ventricular function, ventricular size, and valvular function. Specifically, it is used to estimate the left ventricular ejection fraction (LVEF), an important piece of information, as it assists in determining appropriate therapies, disease progression and prognostic information.

Clinical Features

Physical examination can provide information about the severity of illness and allows assessment of volume status. Clinical features of heart failure include orthopnea, elevated jugular venous pressure (JVP), cardiomegaly, third and fourth heart sounds, inspiratory crackles, pedal edema and hepatomegaly.[6] Decreased exercise tolerance can be a sign of heart failure and typically manifests as shortness of breath on exertion.

Biomarkers that should be measured to assess for heart failure include natriuretic peptides and troponins. Troponins are measured to detect a "silent" myocardial infarction as the underlying cause of a new onset CHF. Natriuretic peptide can be used to exclude or confirm heart failure if the test is accessible and there is uncertainty of the diagnosis.[3] Cardiac troponins may be slightly elevated in an episode of acute decompensation and do not necessarily indicate the presence of a myocardial infarction.[1]

Management

The 5-year mortality remains around 45-50% despite advancements in treatments.[5] Control of risk factors (e.g., hypertension, diabetes mellitus) and other conditions associated with heart failure (e.g., atrial fibrillation) can decrease the risk of future cardiovascular consequences.[2,3] Oral and intravenous diuretics are often used early in therapy to decrease congestion.[3,7] The

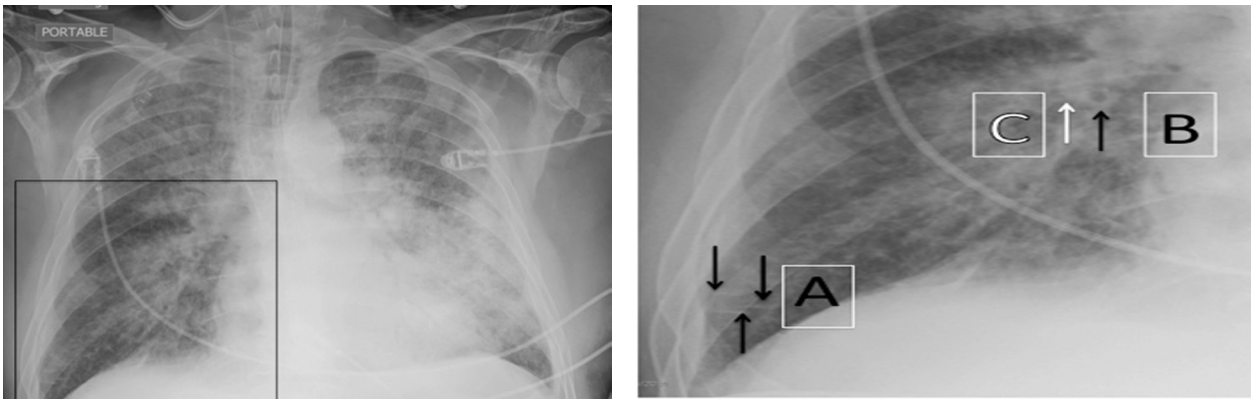

Figure 2. AP radiograph and zoomed-in periphery of the right lower lobe. (A) Kerley "B" lines indicating fluid in the interlobular septa. (B) Bronchial wall thickening indicating fluid in the interstitial space. (C) Vascular wall indistinct indicating fluid in the interstitial space.

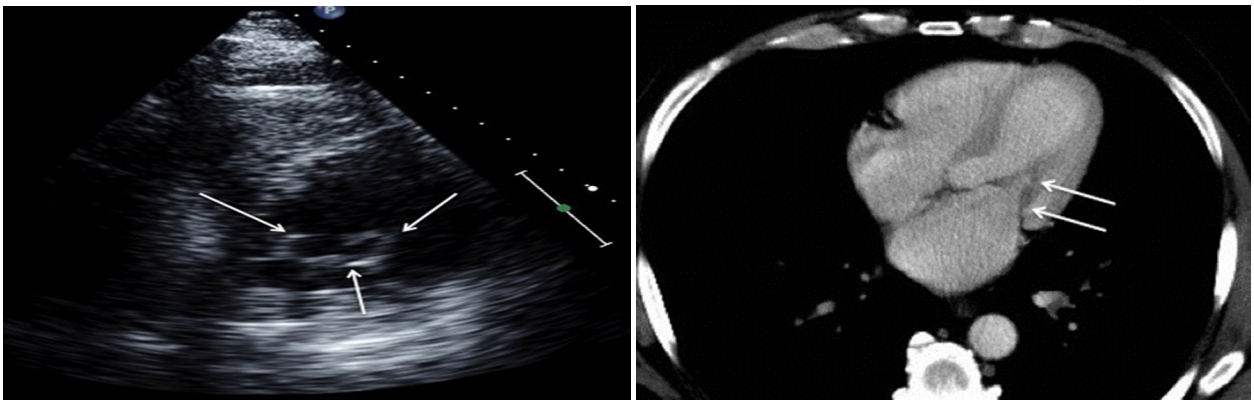

Figure 3. Short-axis echocardiogram/cardiac ultrasound (left) in the same patient shows thickened mitral valves resulting in a "fish mouth" appearance (arrows). An axial CT scan (right) shows thickened mitral leaflets and sub-mitral apparatus (arrows).

goal is to decrease cardiac filling pressures, peripheral congestion and pulmonary edema.[3] Oxygen may be used as part of treatment in hypoxic patients. It should be used with caution in normoxic patients as it may increase systemic vascular resistance and effect cardiac output.[3] The use of angiotension converting enzyme (ACE) inhibitors have been shown to improve survival, however, they should not be started in the setting of an acute episode. Angiotensin receptor blockade (ARBs) agents may be used if an ACE inhibitor is not tolerated. Beta-blockers are recommended for many patients to control symptoms and improve cardiac performance unless contraindicated (e.g., hypotension, bradycardia).[3,7] Mineralocorticoid receptor antagonists may be needed in those patients with more severe functional impairment (NYHA II-IV) if symptoms still persist despite optimized initial therapy.[2,3] Most importantly, any underlying condition (e.g., coronary artery disease) should be treated to improve quality of life and reduce the risk of sudden death.

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