
A 65-year-old male was hospitalized for progressive shortness of breath, orthopnoea, and haemoptysis. He is known to have situs inversus totalis and was being treated for non-ischaemic dilated cardiomyopathy, HIV, and diabetes mellitus. On examination, he was tachypnic, tachycardic, afebrile with jugular venous distension, bilateral lower extremity oedema, left lung basal crackles, and a pansystolic murmur over the cardiac apex radiating to the right axilla and S3 gallop. B-natriuretic peptide (BNP) was 1800 pg/mL (normal ≤ 100) and white blood cell count was normal. Chest X-ray showed left lung opacity (Panel A) that occupied the upper and middle lobes of the trilobed left lung on CT scan (Panels B and C) which also ruled out pulmonary embolism. Echocardiogram showed reduced left ventricular systolic function and moderate mitral regurgitation. We challenged the emergency department diagnosis of pneumonia and intravenous diuresis was instated which rapidly resolved the patient’s symptoms with marked improvement in the left lung opacity 48 h after admission (Panel D), conforming to the diagnosis of unilateral pulmonary oedema (UPE). UPE is rarely encountered in congestive heart failure and is associated with higher mortality than bilateral pulmonary oedema due to the delay in diagnosis. It usually involves the right upper lobe (contralateral to the heart) with main explanation being a severe mitral regurgitation jet that predominantly affects the upper right pulmonary vein. Other explanations include the poorer lymphatic drainage of the right lung, decreased flow in the left pulmonary artery due to compression by the left-sided cardiac enlargement, prolonged rest on one side in patients with cardiac decompensation, or if there is pre-existing disease affecting the parenchyma or vasculature of a lung. In the presented case, UPE occurred in the left (contralateral to the heart) upper and middle lobes (notice the arrows at fissure sites in Panel C) as the patient had situs inversus with dextrocardia. Important clues that aid in achieving the diagnosis are the clinical evidence of congestive heart failure, absence of symptoms and signs of infection, elevated BNP, and normal procalcitonin. UPE can be confused with pneumonia leading to inappropriate initial therapy and hence the importance of maintaining a high index of suspicion for timely diagnosis and management.