

**CASE OF THE MONTH** Sumi Prakash MD, Rahul Shekhar MD, William Steinmann MD

A 67 year old Caucasian male presented to the Emergency Room with a 4 day history of increasing shortness of breath. He reported dyspnea with minimal exertion, dizziness on standing and chest pain with occasional radiation to his left arm; his companion noted that he had been making a rattling sound with respirations for the past few days. The patient had not been weighing himself but did notice swelling in his feet. He does not use home oxygen and denied any reflux symptoms, daytime or nocturnal cough, sputum production or history of asthma. He is a truck driver but denied any unusual travel; he also denied recent sick contacts, fever or chills. Six months ago, during a hospitalization for a burn, he was found to have a pleural effusion which, when tapped, was transudative; a workup for its cause, including an echocardiogram, was negative and the effusion resolved spontaneously.

The patient has a history of atrial fibrillation, diabetes mellitus type 2, arthritis, gout, hypothyroidism and hyperlipidemia. He underwent a gastric bypass procedure in 1976 and, despite having lost 200 pounds since that time, is still morbidly obese. Physicians have told him that he likely has obstructive sleep apnea but a sleep study has not been performed and he does not use CPAP. He denies tobacco, alcohol or illicit drug use. Allergy history is limited to Tylenol. Current medications included digoxin, aspirin, insulin, glyburide, furosemide, allopurinol, atorvastatin, tamadol, pregabalin, docusate and hydrocodone. His family history is negative for Cardiopulmonary disease.

Initial physical exam revealed T 36C, RR 18, P 56, BP 145/69 and O<sub>2</sub> Sat of 94% on 4L. He was alert but in mild respiratory distress, developing dyspnea with minimal exertion. Oral mucosa was dry and no JVD was noted. Auscultation revealed good air flow bilaterally with coarse rales at the bases. Cardiac exam revealed a regular bradycardia with a 1/6 systolic murmur heard best at the apex with radiation to the axilla; a loud P<sub>2</sub> was also noted. Abdominal exam was normal with no organomegaly. 3+ edema was noted in his lower extremities.

Admission lab data revealed: WBC 12.6 (80.5%G), Hgb 10.7, Platelets 144, INR 1.2, D dimer 1.3, Na 138, K4.2, Cl 99, CO<sub>2</sub> 25, BUN 104, Cr 1.7, FeNa 1.45 (on Lasix), Glucose 205, Ca 8.8, TP 7.9, Alb 2.6, TB 0.9, AP 91, AST 36, ALT 34; ionized CA and Mg were normal. His CK was 90, CK-MB 2.3, Troponin 0.01. Digoxin level was 0.5 and BNP was 840 (Cr 1.7). His UA was unremarkable and the TSH was mildly elevated at 6. ABGs revealed a pH of 7.37, pCO<sub>2</sub> 46, pO<sub>2</sub> 73, HCO<sub>3</sub> 26, O<sub>2</sub> sat 93% on 4L; his A-a gradient was 133.3 (nl 20.75).

Admission EKG revealed sinus/junctional bradycardia with a few PVCs but no evidence of acute ischemia. A CXR showed a right basal opacity (increased from previous studies), interpreted as possible basilar pneumonia; mild pulmonary vascular prominence was noted as well as mild, stable cardiomegaly. A VQ scan suggested a low probability of pulmonary emboli, with changes correlating with the right basilar infiltrate.

The patient was admitted with presumed RLL Pneumonia, acute renal failure and possible superimposed CHF. Due to his bradycardia, the digoxin was discontinued. He was started on renally-dosed Levaquin, Lasix was withheld and gentle IV hydration was initiated. An echocardiogram was read as demonstrating normal systolic and diastolic function. Oxygen requirement was 4-6 L/min and both insulin and statin therapy were continued. The patient remained afebrile and normotensive and his WBC fell to normal levels. Continued dyspnea prompted a followup CXR which revealed a right pleural effusion; a thoracentesis revealed transudative fluid and resulted in both improved symptoms and diminished O<sub>2</sub> requirement. The pleural fluid gram stain and culture were negative. (continued on page 5)

Despite the normal echocardiogram, management was shifted toward a probable diagnosis of CHF, with sodium restriction, diuretic therapy and graduated compression stockings. Cardiology was consulted due to the persistent bradycardia; they recommended augmentation of his thyroid supplementation which, unfortunately had no effect on his heart rate or symptoms. A repeat echocardiogram again revealed a normal LV ejection fraction but showed dilation of the RV and LA. With these findings and his lack of clinical improvement, Cardiology elected to proceed with electrophysiologic studies for evaluation of possible sick sinus syndrome; in the course of pacemaker evaluation, a right heart catheterization was performed which demonstrated marked elevation of the PA and PCW pressures; his cardiac output did not improve with pacing and he was thus not considered to be a pacemaker candidate. Following the procedure, the patient developed hypoxic respiratory failure and he was transferred to the CCU, where he was managed with dobutamine, diuresis, sildenafil, fluid restriction and biPAP for presumed OSA. A repeat right heart catheterization showed mildly depressed cardiac function with moderately elevated PCWP and a mean PA of 40 (down from 60 on the initial study) but with no significant response to oxygen.

**DISCUSSION:** We believe that this patient's most likely diagnosis is progressive right sided heart failure, which has gradually worsened over the past 6 months; the etiology is probably OSA/COPD with diastolic dysfunction, exacerbated by inadequate thyroid supplementation, inadequate diuresis, bradycardia and chronic anemia. An initial diagnosis of pneumonia, based on leukocytosis, CXR and echocardiogram reports, set the stage for inadequate management of his actual underlying condition, pointing toward the problem of physician bias in response to diagnostic reports; clearly, a series of echocardiogram reports were misleading. Worsening of his clinical condition during the hospitalization resulted from too much reliance on these studies and more definitive evaluation was delayed. The potential role of inadequate thyroid supplementation remains uncertain and the possibility of thiamine deficiency, secondary to his past gastric bypass, is under investigation; serum thiamine, RBC transketolase activity, lactate and pyruvate have been ordered to rule out Beriberi.

