

CASE OF THE MONTH

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An 86 year old AA woman was referred to University Hospital from the dialysis center where she had complained of worsening dyspnea over the past 2 weeks. She denied cough, fever, chills or rigors. Per her dialysis notes, bi-basilar crackles had been noted in recent weeks and an effort to increase dialysis volume was made; however, this led to episodes of hypotension (confirmed by dialysis records). At the time of admission, the patient also complained of bilateral chest pain which was constantly present but increased with deep inspiration. She also reported intermittent swelling of the right arm and right face over the past 8 weeks which became worse after lying down.

Her PMH was remarkable for adult polycystic kidney disease, aortic stenosis and CKD V requiring dialysis. Two prior admissions for symptoms similar to this presentation led to diagnoses of volume overload and secondary pleural effusion due to insufficient dialysis. She was also admitted 10 months ago for pneumonia and sepsis. Past surgical history was limited to placement of an A-V fistula, six months ago. She denied anorexia or weight loss. Family history was remarkable for a brother with polycystic kidney disease.

On exam, the patient was afebrile. Vitals: BP 106/75, P 84, O2 sat 92% on RA. Diminished breath sounds were noted on the right side. Cardiac exam revealed a systolic ejection murmur; JVD was noted. Abdominal exam was unremarkable. Edema of the right arm and right face were noted and there was an A-V fistula in her right arm; 2+ edema was also found in her lower extremities.

Admission labs revealed WBC 7.6, normal differential, Hgb 8.2, normal LFTs, serum albumin 3.6 and a serum creatinine of 5.1. Admission CXR revealed a large right pleural effusion and a minimal effusion on the left side. Old records (3 months ago) showed that moderate bilateral pleural effusions were present at that time; these records also contained an echocardiogram which demonstrated aortic stenosis and an ejection fraction of 45%.

On admission, our differential diagnosis included subclavian stenosis, Pancoast tumor (or other malignancy), subclavian venous thrombosis, congestive heart failure, constrictive pericarditis, pulmonary embolism and uremic pleuropericarditis. A repeat echocardiogram was unchanged and revealed class 1 diastolic dysfunction. In light of her recurrent pleural effusions, a diagnostic and therapeutic thoracentesis was performed (2L) which was transudative by Light's criteria; her symptoms improved significantly after the procedure. A RUE ultrasound was negative for thrombosis. Due to her ESRD, a chest CT and CT venogram were performed at the same time; no mass was found but the venogram revealed near total occlusion of the right subclavian vein. A percutaneous angioplasty was recommended but the patient refused; after experiencing recurrent symptoms one week later, she agreed to the angioplasty and, following this, her episodic hypotension, pleural effusions and edema resolved.

Final diagnosis: subclavian vein stenosis, presumably secondary to a subclavian line placed during her admission for pneumonia and sepsis (10 months ago).

Discussion: The five most common causes for pleural effusion are CHF, hypoalbuminemia, pulmonary embolism, infection and malignancy. While this woman had classic symptoms, subclavian vein thrombosis or stenosis should be considered in all cases of recurrent pleural effusion. Subclavian vein thrombosis complicates up to 5% of subclavian catheter placements and this condition may have contributed to both her dialysis hypotension and her relatively new onset of diastolic dysfunction; the latter may also be secondary to dialysis related pulmonary hypertension.