Abstract Form

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Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction: Pulmonary-renal syndrome refers to an autoimmune-mediated process that causes rapidly progressive glomerulonephritis and diffuse alveolar hemorrhage. Early recognition is important to prevent multi-organ complications but can be difficult due to its nonspecific findings. Our patient had symptoms of fever, pulmonary masses, lymphadenopathy, hemoptysis, and rapidly worsening renal failure that overlapped with infectious and malignant processes.

Case Report: A 70-year-old male without significant past medical history presented with three weeks of whole-body itching and one week of cough productive of blood-streaked sputum. He endorsed unintentional weight loss of 9lbs over three months. His initial vitals were T 37.7, HR 102, BP 100/60, RR 18, and SpO_2 95% on room air. His exam was unremarkable other than chronic excoriations on his bilateral upper extremities. Labs revealed WBC 13.7, Hb 10.9, Na 127, K 4.3, CO2 18, BUN 30, Cr 1.7, and procalcitonin 0.48. UA showed 3+ blood, 2+ protein, WBCs, and RBCs. CXR revealed a right lower lobe consolidation and nodular opacities in the upper lobes. He was started on azithromycin and ceftriaxone for initial suspicion of community-acquired pneumonia. A follow up non-contrast CT chest showed multiple pulmonary masses and mediastinal lymphadenopathy. He continued to have fevers and episodes of hemoptysis more suggestive of malignant and infectious processes. Blood, sputum, and fungal serologies were negative; the QuantiFERON and sputum PCRs were negative for M Tb, but AFB cultures grew Mycobacterium terrae felt to be a contaminant. Interestingly, a Strongyloides IgG antibody was borderline positive. He was treated with ivermectin but a subsequent small bowel biopsy was negative for this. A CT-guided lung biopsy of the right lower lobe nodule showed atypical spindle cells in a background of inflammation; this was felt to be due to reactive stromal change and not malignancy. During the workup his renal function worsened daily, with creatinine increase from 1.7 to 3.1 by day 7 of admission. Concurrently, our autoimmune workup revealed a positive ANA (1:160), MPO-ANCA (85 AU/mL) and PR3-ANCA (62 AU/mL); complements, ACE, anti-GBM, ASO, RF, and cryoglobulins were all negative. Decision was made to pursue a renal biopsy which confirmed focal, pauci-immune (ANCA-associated) necrotizing and crescentic glomerulonephritis with focal necrotizing arteritis. Patient was cleared from an infectious standpoint to start immunosuppression on day 7 of admission with pulse-dose steroids and rituximab. His hemoptysis resolved and a repeat lung biopsy was recommended but the patient preferred a non-invasive CT Chest which showed improvement of the infiltrates, pulmonary nodules, and resolution of the mediastinal lymph nodes. The resolution of lung nodules from immunosuppression favored an inflammatory rather than a malignant process. He was discharged on maintenance rituximab and a 6-month prednisone taper. The lack of granulomas on biopsies favor a diagnosis of microscopic polyangiitis. Patient unfortunately required long term dialysis os discharge but showed gradual improvement in renal function.

Discussion: The differential diagnosis of pulmonary masses with mediastinal lymphadenopathy and hemoptysis typically centers on infection versus malignancy. However, when accompanied by rapidly worsening renal function clinicians should consider pulmonary-renal syndromes. These can be divided into 1.) anti-GBM disease, 2.) autoimmune connective tissue diseases, 3.) drug induced vasculitis, 4.) ANCA negative vasculitis, and 5.) ANCA associated vasculitis. The latter is usually caused by one of three entities: granulomatosis with polyangiitis, eosinophilic granulomatosis with polyangiitis, and microscopic polyangiitis differentiated largely by pathology. Early intervention of pulmonary-renal syndrome can significantly reduce mortality and morbidity. In our case, we were able to initiate immunosuppression for treatment of ANCA vasculitis with significant improvement in our patient's subsequent pulmonary imaging and clearance of his hemoptysis.