Spitting blood and casts with mud: A rare and unusual presentation of C3 glomerulonephritis

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Introduction
C3 glomerulonephritis (C3GN) is a rare disease that falls under the umbrella of C3 glomerulopathy. It is manifested by abnormal activation of the alternate pathway resulting in complement deposition in the glomeruli. Kidney biopsy with characteristic findings of electron-dense deposits in the mesangial and capillary wall confirms the diagnosis. Until recently, C3GN presenting with primarily lung involvement had not been reported. We present a unique case of initial pulmonary manifestation C3GN.

Case Report
This is a 21-year-old caucasian male with no past medical history presented to the hospital with a chief complaint of acute hemoptysis for 1 day. One week prior to admission, he visited urgent care with symptoms of cough, rhinorrhea, and congestion. He was diagnosed with acute bronchitis. He was treated with steroids, bronchodilators, and a 3-day course of Cefdinir. Per patient, symptoms initially improved after finishing the antibiotic course but the day prior to arrival, he experienced fevers, chills, night sweats, and hemoptysis. On admission, he had an acute kidney injury with elevated creatinine of 1.73 mg/dL, anemia with Hgb of 11.9 g/dL. Urinalysis revealed proteinuria (> 500 mg/dL) and hemoglobinuria. Inflammatory markers ESR and CRP were elevated, 18 and 13.1, respectively. Computed Tomography Angiography showed extensive bilateral parenchymal infiltrates with associated pleural fluid suspicious for diffuse alveolar hemorrhage. Infectious and autoimmune workup was negative. Complement levels showed normal C4 and low C3 of 51 mg/dL. Antistreptolysin O, ANCA and anti-GBM titers were within normal limits. Due to continued worsening hemoptysis, the patient was taken for a bronchoscopy. Results were consistent with diffuse alveolar hemorrhage. Renal biopsy was consistent with C3 glomerulonephritis.

On admission, he was started on steroids and broad-spectrum antibiotics. Despite steroid therapy, his respiratory status worsened, he was transferred to the ICU and intubated. Once kidney biopsy confirmed C3GN, he received several rounds of plasmapheresis and was started on mycophenolate mofetil. His renal function returned to baseline and his pulmonary symptoms subsided post-therapy.

Discussion
Pulmonary renal syndrome (PRS) is a rare condition that includes diffuse alveolar hemorrhage (DAH) and glomerulonephritis. Oftentimes, the rapid deterioration can lead to death; thus, a rapid diagnosis of the underlying disease improves survival. Commonly, PRS is associated with autoimmune etiology such as systemic vasculitis, Goodpasture's syndrome, or Systemic Erythematous Lupus. This patient, uncommonly, had PRS secondary to C3GN; even more unusual, the initial presentation of C3GN was hemoptysis. Classic manifestations of C3GN
include acute renal failure, proteinuria, and hematuria. Management of PRS depends on treating the underlying cause as therapies may differ. Most often, the treatment modalities will involve pulse dose steroids, plasmapheresis, and immunosuppressive agents (eculizumab for C3GN).

References
5. Pulmonary renal syndrome: A 4-year, single-center experience