Anti-Glomerular Basement Membrane Disease Following COVID-19 Infection

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Introduction

- The medical community continues to uncover the intricate ways in which COVID-19 can influence and exacerbate pre-existing conditions, especially autoimmune diseases.
- Here, we present a case of a 44-year-old female who developed anti-glomerular basement membrane (anti-GBM) disease.

Case Report

- 44-year-old African American female with a history of hypertension presented to our hospital with one week of hematuria, and shortness of breath.
- Labs revealed a creatinine of 3.4mg/dL, eGFR of 16mL/min, proteinuria of 1288mg/day, and 3+ blood on urinalysis. She tested positive for COVID-19 and started antiviral therapy, yet her creatinine continued to rise to 10mg/dL. Common autoimmune etiologies such as lupus were ruled out.
- CH50 remained >60u/mL, suggesting ongoing immune activity and inflammation, raising the possibility of COVID-19 as the most likely etiology for the AKI.
- Renal biopsy findings with positive anti-GBM antibody titer was consistent with anti-GBM disease (see figure 1). She underwent dialysis, plasmapheresis, and steroid therapy, reducing antibody levels from 120.6AI to 13.9AI.
- Immunosuppressive therapy was deferred due to active COVID-19. She remains dialysis-dependent, with plans for renal transplantation.

Discussion

- Anti-GBM disease is characterized by autoantibodies targeting the glomerular basement membrane, often leading to rapidly progressive renal failure.
- Patients with anti-GBM disease often present with hematuria, proteinuria, and rapidly declining kidney function, and in cases involving the lungs, symptoms can sometimes include hemoptysis and shortness of breath. Diagnosis is via renal biopsy, which will usually demonstrate linear IgG deposits along the glomerular basement membrane, as was found in our patient.
- Plasmapheresis is particularly important as it helps to remove circulating anti-GBM antibodies in the blood, thereby reducing further autoimmune sequela on the kidneys and lungs. This approach has been shown to improve renal outcomes, especially when initiated early in the disease course
- Emerging evidence suggests COVID-19-associated endothelial injury to type IV collagen may initiate aberrant autoimmune responses. While anti-GBM disease is rare, its association with COVID-19 highlights novel associations with autoimmune diseases.
- This case is unusual in that our patient lacked pulmonary involvement, which is often seen in anti-GBM disease. Our patient is also of African American descent, raising questions regarding COVAN, anti-GBM, and APOL1 variants.
- These genetic factors remain an area of active investigation, as they may influence susceptibility to severe kidney disease. Further research is warranted regarding COVID-19's role in triggering autoimmune diseases and optimizing management strategies.

Conclusion

- The COVID-19 pandemic has reshaped our understanding of viral illnesses, revealing unexpected complications and interactions with underlying medical conditions.
- The incidence of anti-GBM disease as it relates to COVID-19 remains low, with less than 15 cases documented since the onset of the pandemic. While there is an association between COVID-19 and anti-GBM disease, it is a relatively rare occurrence.
- The careful balancing of risks and benefits is crucial in determining the appropriate course of treatment, and further research is needed to better understand the interaction between COVID-19 and autoimmune conditions, especially as it relates to patients presenting with new onset renal failure.

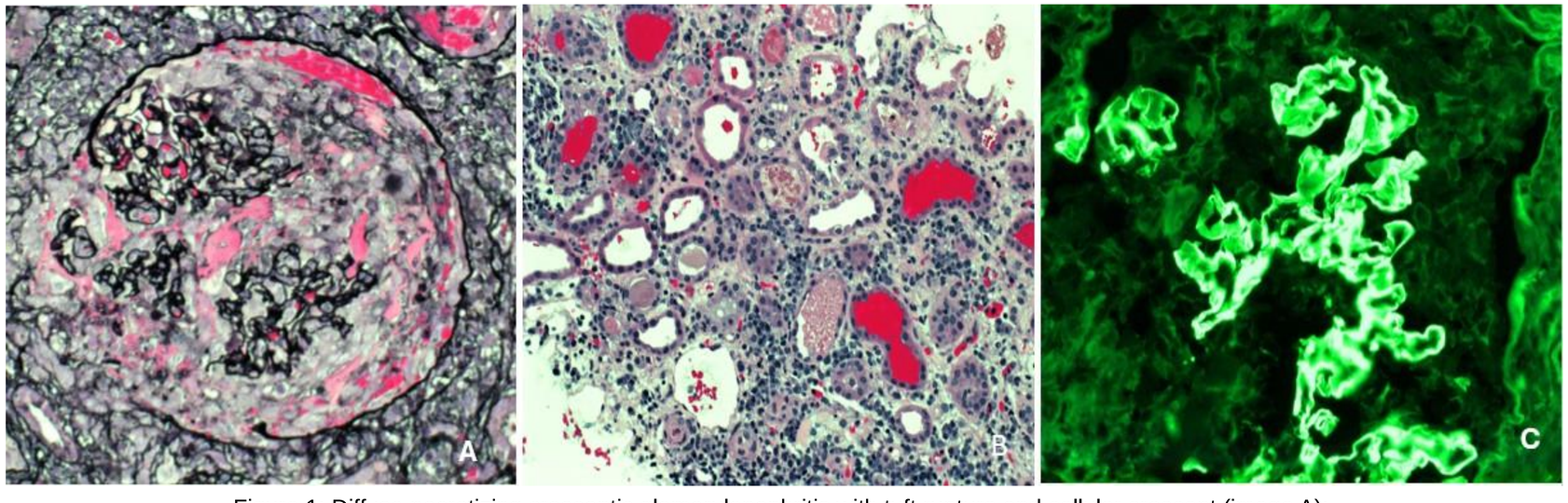

Figure 1: Diffuse necrotizing crescentic glomerulonephritis with tuft rupture and cellular crescent (image A); the tubulointerstitium shows acute tubular injury and numerous red cell casts (image B). Immunofluorescence for IgG shows bright linear staining of the glomerulus (image C)