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Abstract Topic : Glomerular and Tubulointerstitial Disorders

A Case of eldery patent with anti-glomerular basement disease manifestinating rapid progressive glomrulonephritis without pulmonary involvement

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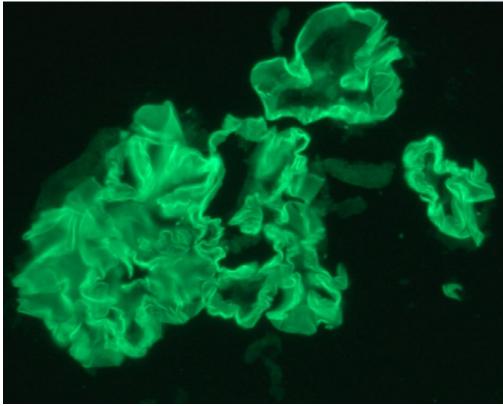
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Case Study: Anti-glomerular basement membrane (anti-GBM) disease is a rare autoimmune disorder that typically presents with rapidly progressive glomerulonephritis (RPGN) and is often accompanied by pulmonary hemorrhage. However, in certain cases, the pulmonary manifestations may be absent, which can delay the diagnosis. An 82-year-old woman with a history of diabetes, hypertension, and chronic kidney disease (CKD) was admitted to the hospital due to poor oral intake and gross hematuria. She had experienced a cough and myalgia one week prior and had taken NSAIDs and antibiotics. Upon admission, her blood urea nitrogen was 66.1 mg/dL, creatinine(Cr) was 4.43 mg/dL, and her fractional excretion of sodium was 6.5%. A chest X-ray revealed no abnormalities, and she had no cough, sputum production, or dyspnea. By the third day of hospitalization, her Cr had increased to 5.26 mg/dL, prompting serological testing and a kidney biopsy. Despite conservative management, her renal function continued to worsen, and she required dialysis on the sixth day. A kidney biopsy revealed findings consistent with anti-GBM glomerulonephritis, and her anti-GBM antibody titer was markedly elevated at 113.15 U/mL. Although plasmapheresis was recommended, patient declined it. Immunosuppressive therapy alone failed to restore her renal function. Throughout her stay, she remained free of pulmonary symptoms, and repeated chest X-rays remained normal. This case illustrates how the absence of respiratory manifestations and the history of nephrotoxic drug use led to delayed diagnosis and treatment. The initial suspicion was acute tubular necrosis, potentially due to nephrotoxic agents, but the kidney biopsy ultimately confirmed anti-GBM disease. Early testing for anti-GBM antibodies in patients with unexplained acute renal failure, even in the absence of respiratory symptoms, is crucial for timely and accurate treatment. In conclusion, clinicians should consider anti-GBM disease as an atypical cause of acute kidney injury, recognizing that pulmonary involvement may not always be present.

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