A Unique Case of Anti-Glomerular Basement Membrane Disease

Dear Editor,

Anti-glomerular basement membrane (anti-GBM) disease results from autoantibodies directed against the non-collagenous 1 domain (NC1) at the α3-chain C-terminus of type 4 collagen [α3(IV)NC1] with a predilection towards young men. Anti-GBM disease is rarely reported following urinary obstruction and extra corporeal shock wave lithotripsy.[1-3] A patient with metastatic prostate cancer developed kidney failure with a work-up showing anti-GBM disease.

An 86-year-old white male with a history of metastatic prostate cancer, treated with hormonal/radiation therapy with 654 ng/L PSA presented with kidney failure. He was found to be volume-overloaded with serum creatinine (14.7 mg/dL; baseline value 1.0) and 226 mg/dL BUN. Urinalysis revealed 3+ blood, 2+ protein, and numerous RBCs on microscopy. CT abdomen showed prostatic enlargement and bladder wall thickening consistent with chronic bladder outlet obstruction. An indwelling urinary catheter did not improve kidney function. Kidney biopsy showed necrotizing and crescentic glomerulonephritis and immunofluorescence showed linear IgG staining of GBM [Figure 1]. Anti-GBM antibodies were elevated to 182 units/mL (normal 0-20). ANA and anti-MPO/ anti-proteinase 3 antibodies were negative with normal complement levels. The patient was initiated on pulse methylprednisolone and plasmapheresis without further immunosuppressive therapy. He remained dialysis-dependent with no renal function recovery.

It is hypothesized that stabilized NC1 hexamer disruption and α3-chain cryptic antigen exposure to the host immune system are required for anti-GBM antibody development.[4]

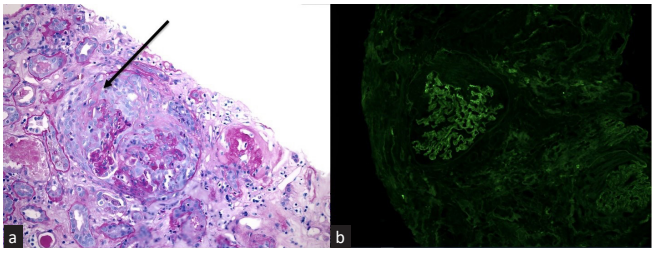

Figure 1: (a) Light microscopy showing hypercellular circumferential crescent (black arrow) compressing the glomerular tuft and closing the capillary loops (x100). (b) Immunofluorescence microscopy showing linear IgG along the glomerular basement membrane (x100).

Excreted NC1 may enter the renal interstitium during obstruction, dissociating in an acidic environment, triggering anti-GBM antibody production. Our case highlights a rare cause of kidney disease in a patient with cancer-associated outflow obstruction. Anti-GBM-associated RPGN development should be entertained when kidney failure persists despite relief of outflow obstruction.

Conflicts of interest: There are no conflicts of interest.

Kalathil K Sureshkumar[1], Ahmed Barazi[1], Khaled Nashar[1]

[1]Division of Nephrology and Hypertension, Medicine Institute, Allegheny General Hospital, Allegheny Health Network, Pittsburgh, PA, United States

Corresponding author: *Kalathil K Sureshkumar, Division of Nephrology and Hypertension, Medicine Institute, Allegheny General Hospital, Allegheny Health Network, Pittsburgh, PA, United States. E mail: kalathil.sureshkumar@ahn.org*

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