A case of paraneoplastic syndrome associated rapidly progressing glomerulonephritis in a patient with colon cancer

Sir,

Rapidly progressive crescenteric glomerulonephritis (RPGN) is often a consequence of systemic disease. We present a case of RPGN found in the setting of colo-rectal carcinoma, leaving paraneoplastic syndrome associated RPGN as the most likely etiology.

A 46-year-old African American female was admitted with a history of colon carcinoma managed with chemotherapy and partial colectomy. She had a normal creatinine 8 months prior to admission. Her creatinine was 3.1 mg/dl on the day of admission and her antinuclear antibodies, ANCA, antiglomerular basement membrane, antiproteinase-3 antibodies, and myeloperoxidase antibodies were negative. On the 3rd day of admission her creatinine was 4.5 mg/dl and urinalysis revealed active urine sediments. She underwent a renal biopsy, which revealed crescent-shaped mass of proliferating cells and leukocytes internal to bowman capsule along with thickening of periphery capillary walls, collapsed glomerular tufts and expanded mesangial areas with formation of mesangial nodules that stained periodic acid Schiff and silver positive on light microscopy. Electron microscopy showed thickening and segmental hyalinization of small size arteries and fibrosis in the interstitium. Immunofluorescence was negative for immune complexes [Figure 1]. She was managed with intravenous steroids and cyclophosphamide, and was discharged with a creatinine of 2.4 mg/dl.

The outcomes of malignancies are not limited only to tumor bulk or metastases. Malignancies can produce hormones, cytokines, or immunologic responses that have diffuse system impacts.[1] The development of unusual pathologies in cancer patients with no alternative etiology should raise suspicion towards a paraneoplastic syndrome. The glomerular damage in our patient is likely related to cytokine-mediated effects of malignancy, as evidenced by the lack of immune complexes on immunofluorescence.[2]

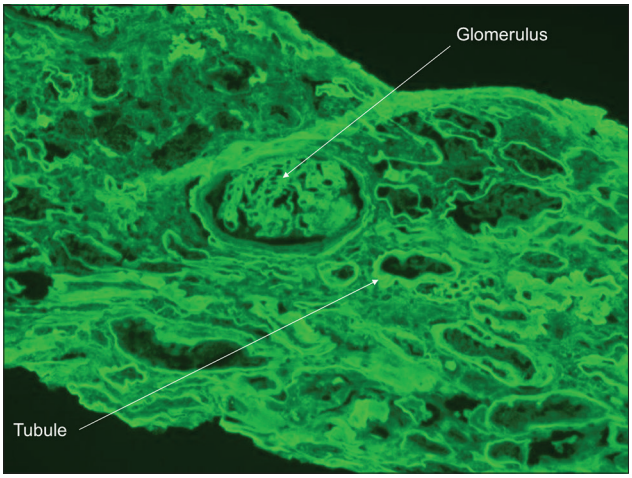

Figure 1: Immunofluorescence IgG 10X: Linear staining along glomerular peripheral capillary walls and tubular basement membranes (2+). No specific vascular staining

Under the influence of various cytokines, a pro inflammatory state is maintained.[3] Such an environment could be analogous to the labile cellular milieu encountered in systemic vasculitis. It is not surprising, then, that the end organ consequences of both pathologies be similar as indicated by the RPGN observed in our patient.

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