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CLINICAL VIGNETTE

Treatment of Cryoglobulinemic Glomerulonephritis Secondary to Hepatitis C Virus-Associated Cryoglobulinemia with Plasmapheresis

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Case Report

Patient is a 71-year-old female with history of hepatitis C virus, genotype 2, and hypertension. She was admitted to the hospital for refractory hypertension and newly diagnosed proteinuria and hematuria. The patient was seen in renal clinic about 3 weeks prior to her hospitalization with difficulty in controlling her previously well-controlled hypertension despite unchanged medications. Patient also complained of edema in lower extremities. A work up for secondary causes hypertension including hyperaldosteronism, pheochromocytoma, and renal artery stenosis were negative. Urine tests showed hematuria, large blood on urine dipstick, and proteinuria with urine protein to creatinine ratio of 2.78 and 2.67 in two separate urine specimens. She had an elevated serum creatinine of 1.1 mg/dL; compared to 0.7 mg/dL in the past, and had no prior history of hematuria or proteinuria. Additional laboratory tests included negative antinuclear antibodies (ANA) and antineutrophil cytoplasmic antibodies (ANCA). She had low complement C4 level of 7mg/dL and C3 level of 45 mg/dL, as well as positive serum cryoglobulin.

A clinical diagnosis of glomerulonephritis secondary to cryoglobulinemia associated with hepatitis C virus was considered. She underwent kidney biopsy, which showed cryoglobulinemic glomerulonephritis in a membranoproliferative pattern. The patient underwent plasmapheresis during her hospitalization and repeat serum cryoglobulins after plasmapheresis were negative. Her hematuria and proteinuria also improved after plasmapheresis. The patient then started antiviral therapy for hepatitis C virus. She had complete resolution of her proteinuria and hematuria as well as her cryoglobulinemia after successful treatment of her hepatitis C. Follow-up tests showed no hematuria, and a urine protein/creatinine ratio of 0.07 one year after kidney biopsy.

Discussion

Chronic hepatitis C virus (HCV) infection is frequently associated with a variety of autoimmune manifestations. Mixed cryoglobulinemia (MC) appears in up to 50% of chronic HCV-infected patients. MC results in the deposition of immune complexes in small or medium-sized blood vessels causing vasculitis and leading to clinical manifestations including palpable purpura and skin ulcers, arthralgias, renal

disease, and peripheral neuropathy.² Hepatitis C virus (HCV) infection has been associated with a large spectrum of glomerular lesions in both native and transplanted kidneys. They include membranoproliferative glomerulonephritis in the context of type II MC, the most common type,³ membranoproliferative glomerulonephritis without MC, membranous glomerulonephritis and polyarteritis nodosa. In addition to antiviral and immunosuppressive therapies, plasmapheresis therapy for cryoglobulinemic glomerulonephritis secondary to hepatitis C virus-associated cryoglobulinemia. Both regular plasmapheresis⁴ and double filtration plasmapheresis^{5,6} have resulted in improvement in renal function in this group of patients. Plasmapheresis has also been a successful therapy in patients with non-renal^{7,8} and pulmonary renal⁹ cases of hepatitis C virus-associated cryoglobulinemic diseases.

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