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Refractory FSGS Associated with CRB2 Mutation

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Case Study : A 32-year-old man with a history of persistent proteinuria since middle school presented with ongoing nephrotic-range proteinuria. He had been initially diagnosed with focal segmental glomerulosclerosis (FSGS) at the age of 16 years. His younger brother had IgA nephropathy. At presentation, blood pressure was 164/67 mmHg. Serum creatinine was 0.71 mg/dL, and the urine protein-to-creatinine ratio (PCR) was approximately 6.1 g/g. A repeat kidney biopsy demonstrated enlarged glomeruli with mesangial hypercellularity and matrix expansion. No immune complex deposition was identified on immunofluorescence or electron microscopy. Electron microscopy showed focal moderate foot process effacement without electron-dense deposits, consistent with podocytopathy. High-dose prednisolone (1 mg/kg/day) was administered for 3 months but showed no response, and cyclosporine therapy was subsequently initiated. Despite prolonged treatment for 2 years, nephrotic-range proteinuria persisted without significant reduction. An SGLT2 inhibitor (dapagliflozin) was later added; however, no meaningful antiproteinuric response was observed. Throughout follow-up, urine PCR remained approximately 5–10 g/g. Serum creatinine gradually increased from 0.7 mg/dL to approximately 1.2 mg/dL over 4 years, indicating slowly progressive renal dysfunction. Given the persistent treatment-refractory disease course, whole exome sequencing was performed and revealed two heterozygous variants in CRB2 (p.Gly1205Ser and p.Val747Met), suggesting possible compound heterozygosity consistent with CRB2-associated genetic FSGS. This case highlights that CRB2-associated genetic FSGS may be refractory to conventional immunosuppressive and antiproteinuric therapies due to structural podocyte defects caused by genetic mutations.