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Short Note

C4d in IgA Nephropathy; A Personal Experience

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Keywords: IgA nephropathy; Immunoglobulin A; C4d; Tubulointerstitial fibrosis

IgA nephropathy (IgAN) is a kidney disease that is characterized by the deposition of IgA (immunoglobulin A) in the kidney tissue, leading to inflammation and damage. This disease is a common chronic glomerulopathy that affects people worldwide (1). Therefore, it is crucial to identify factors that worsen the disease, monitor its activity, and predict appropriate treatments. Complement component 4d (C4d) is a protein that is involved in the activation of the complement system, which is a part of the immune system. Likewise, C4d has been found to be present in the kidney tissue of patients with IgAN, particularly in areas where IgA deposits are present (2). This suggests that the complement system may be activated in response to IgA deposition and contribute to the inflammation and damage seen in IgAN. Studies have also shown that elevated levels of C4d in the blood or urine of patients with IgAN may be associated with more severe disease and poorer outcomes (3). Therefore, C4d may be a useful biomarker for monitoring disease activity and predicting prognosis in patients with IgAN. Additionally, targeting the complement system, including C4d, may be a potential therapeutic strategy for treating IgAN (4). To determine whether C4d staining at the time of kidney biopsy had any correlation with the demographic, clinical and biochemical variables in IgAN, we previously conducted a a preliminary study on 29 renal biopsy-proven IgAN patients. The definition of IgAN necessitates the existence of widespread mesangial deposits of IgA across with C1q deposition. In our study, samples retrospectively and by a random selection were subjected to C4d immunohistochemical staining. In this study, out of the 29 patients, 68% were male. Serum creatinine had a mean of 1.72±1.2 mg/dl and proteinuria had a magnitude of 1582±1214 mg/day. In our study significant correlations were observed between percent C4d positivity and serum creatinine (r=0.61, p=0.0005), magnitude of proteinuria (r=0.72, p=0.0001), proportion of globally sclerotic glomeruli (r=0.43, p=0.02) and proportion of tubulointerstitial fibrosis (r=0.54, p=0.0023). In conclusion, our study provides further evidence for the role of complement activation in the pathogenesis of IgAN. However, larger studies are needed to confirm our findings and determine the clinical significance of C4d deposition in IgAN (5).

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