“A Case Report of a Patient presented With Upper Respiratory Tract Infection and Diagnosed with IgA Nephropathy”

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I. Introduction

Berger first described the glomerulonephritis now termed IgA nephropathy. IgA nephropathy (IgAN) is the most common form of glomerulonephritis in the world, and currently is known to be an important cause of end stage renal disease (ESRD). It is classically characterized by episodic hematuria associated with the deposition of IgA in the mesangium. There is a male preponderance, a peak incidence in the second and third decades of life, and rare familial clustering. The most typical presentation is macroscopic hematuria shortly after an mucosal infection such as upper respiratory tract infection and bronchitis. Patients rarely present with nephritic syndrome. It is now well-known that prognosis is highly variable with some patients showing a rapid progression, and IgAN has been an important cause of end stage renal disease (ESRD). Factors including male gender, persistent microscopic hematuria, increased serum creatinine, proteinuria more than 1 g/d, and hypertension at presentation are associated with a worse outcome. On biopsy, crescents, global or segmental sclerosis, tubular atrophy, interstitial fibrosis, interstitial cellular infiltrate, and peripheral capillary wall alterations such as deposits or endocapillary proliferation also indicate a poor prognosis. There is no consensus among authors for treatment of patients at risk for progression.

II. Case Report

A 18 year old male patient was admitted with complain of fever, cough with sputum and breathlessness, generalized body ache.

On Examination :- His general condition was unsatisfactory his initial blood pressure in left arm supine position was 180/110mmhg and pulse rate of 96/min. Pallor & edema was present, Ictreus, cyanosis, Lymphnode absent.

CNS:-WNL Patient was conscious oriented to time, place, person CVS :- S1S2+, No added sound, no murmur was heard

R/S:- B/L AE+, B/L coarse crepts lower zones

P/A:- Soft, Non-tender, No-palpable organomegaly

His initial investigation revealed microcyclic, hyprochromic anemia with lucocytosis (Hb 7.2 method photometry, Wbc 18.2 method electrical impedance), Derranged RFT (B.urea 225, method urose with Indicator dye, S.cret 15.1 method enzymatic, Na140 method Direct ISE, K 7.0 method Direct ISE) LFT WNL.

Urine routine/Microscopy revealed pulse as 2-3/HPF, RBC 14-15/HPF, Epithelial Cell 1-2/HPL, Albumin +++, method Dipstic Reflectance Spectrophotometry/Microscopy. Viral markers were negative.

USG (W/A) was done which was suggestive of renal medical disease.

Right Kidney –10.7x3.7 cm altered echotexture

Left Kidney – 10.7x4.8cm

Kidney biopsy was done which was s/o IgA nephropathy associated with global tuft sclerosis in 9/19(47.3%) glomeruli, secondary segmental sclerosis in 10/19(52.6) capillary tufts and mild increase in mesangial matrix/cellularity in viable glomerular areas6/19(31.5%) glomerulushow fibrocellular cresent formation.

III. Discussion

Although IgA N is primarily characterized by mesangial IgA deposition, light microscopic appearances and clinical features of patients can vary considerably. Proliferative and crescentic forms of IgA are associated with nephrotic-range proteinuria. IgAN is a disease that may lead to ESRD. Approximately 25 to 30% of patients require renal replacement therapy within 20 to 25 years. Hypertension, severity of proteinuria, and the presence of severe lesions on initial renal biopsy such as hyalinosis, and crescents are the most predictive factors for progression to ESRD. Dais et al retrospectively analyzed data from 144 patients with IgAN. They concluded that crescents were associated with an increased initial serum creatinine, proteinuria, hypertension and progression to ESRD. Reichter al revealed that the rate of GFR decline was significantly slower in patients with...
proteinuria <1 g/d than int hose with proteinuria >1 g/d, and proteinuria was the most important predictor of the rate of GFR decline. Despite its prevalence and clinical importance, there is no consensus for the treatment of patients with risk factors for a worse prognosis. The renoprotective effects of angiotensin converting enzyme inhibitors (ACEI) and/or angiotensin receptor blockers (ARB) are well-known, but it has been recommended that these drugs should not be used alone in IgAN patients with poor prognostic factors. In a study conducted by Hogg et al., it was found that alternate day prednisone or omega-3 fatty acids was not superior to placebo in slowing progression of renal disease. In another study, a low dose of prednisolone had an antiproteinuric effect. However, it could not improve renal survival. Nonetheless, there are a number of studies suggesting that steroids and/or cyclophosphamide reduce proteinuria and preserve renal function. Pozzi et al. assessed the efficacy and safety of a 6-month course of steroids in IgAN. In that study, they found that the deterioration in renal function was less in the treatment group than in the control group (P<0.048), and that proteinuria was significantly decreased (P<0.05). The same authors also reported that ten years renal survival in patients treated with steroids for 6 months was better than in the control group (P=0.003). Tumlin et al. investigated clinical and histological response to methylprednisolone and intravenous cyclophosphamide in patients with crescentic, proliferative IgAN, and found significant decreases in serum creatinine and proteinuria. Furthermore, they established that endocapillary proliferation, cellular crescents and karyorrhexis were eliminated in all the patients. In that study, ESRD was developed only in one of 12 patients after 36 months. Ballardie et al. showed that immunosuppressive treatment with steroid and cyclophosphamide significantly preserved renal function during the follow-up lasting 2–6 years. Our patient had many poor prognostic factors including male gender, nephrotic proteinuria, renal impairment, diagnosis. However, treatment with prednisolone and cyclophosphamide reduced proteinuria from 6.5 g/d to 2.2 g/d and decreased serum creatinine from 132 μmol/l to 96.8 μmol/l. We showed obvious regression of crescents on light microscopy. Furthermore, we did not observe any side effects associated with treatment. In conclusion, although prospective studies comparing immunosuppressive treatment with supportive one are warranted, we believe that immunosuppressive treatment is useful in IgAN patients with poor risk factors for progression.

References