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"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 importantcause 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 typicalpresentation is macroscopic hematuria shortly after amucosal infection such as upper respiratory tract infectionand bronchitis. Patients rarely present with nephritic syndrome. It is now well-known that prognosis is highlyvariable with some patients showing a rapid progression,and IgAN has been an important cause of end stage renaldisease (ESRD). Factors including male gender, persistentmicroscopic hematuria, increased serum creatinine, proteinuria more than 1 g/d, and hypertension at presentationare 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 ofpatients 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 microcytic, hyprochromic anemia with lucocytosis (Hb 7.2 method photometry, Wbc 18.2 method electrical impedence), Derranged RFT (B.urea 225, method urease 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%) glomerulishow fibrocellular cresent formation.

III. Discussion

Although IgA N is primarily characterized by mesangialIgA deposition, light microscopic appearances andclinical features of patients can vary considerably. Proliferativeand crescentic forms of IgA are associated withnephrotic-range proteinuria. IgAN is a disease that maylead to ESRD. Approximately 25 to 30% of patients require renal replacement therapy within 20 to 25 years.Hypertension, severity of proteinuria , and the presenceof severe lesions on initial renal biopsy such as hyalinosis,and crescents are the most predictive factors forprogression to ESRD2.Dais et al retrospectively analyzed data from 144patients with IgAN. They concluded that crescents wereassociated with an increased initial serum creatinine, proteinuria, hypertension and progression to ESRD7. Reichet al revealed that the rate of GFR decline was significantlyslower in patients with

proteinuria <1 g/d than inthose with proteinuria >1 g/d, and proteinuria was themost important predictor of the rate of GFR decline8.Despite its prevalence and clinical importance, thereis no consensus for the treatment of patients with risk factorsfor a worse prognosis. The renoprotective effects ofangiotensin converting enzyme inhibitors (ACEI) and/orangiotensin receptor blockers (ARB) are well-known, butit has been recommended that these drugs should not beused alone in IgAN patients with poor prognostic factors6.In a study conducted by Hogg et al, it was found thatalternate day prednisone or omega-3 fatty acids was notsuperior to plasebo in slowing progression of renal disease9.In another study, a low dose of prednisolone had anantiproteinuric effect. However, it could not improve renalsurvival10. Nonetheless, there are a number of studiessuggesting 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 steroidsin IgAN. In that study, they found that the deteriorationin renal function was less in the treatment group than inthe control group ($P<0.048$), and that proteinuria wassignificantly decreased ($P<0.05$)11. The same authors alsoreported that ten years renal survival in patients treatedwith steroids for 6 months was better than in the controlgroups ($P=0.0003$)12.Tumlin et al investigated clinical and histological response to methylprednisolone and intravenous cyclophosphamide in patients with crescentic, proliferativeIgAN, and found significant decreases in serum creatinineand proteinuria. Furthermore, they established thatendocapillary proliferation, cellular crescents and karyorrhexiswere eliminated in all the patients. In that study,ESRD was developed only in one of 12 patients after 36months13.Ballardie et al showed that immunosupressive treatmentwith steroid and cyclophosphamide significantlypreserved renal function during the follow-up lasting 2-6years14.Our patient had many poor prognostic factors includingmale gender, nephrotic proteinuria, renal impairment,diagnosis.However, treatment with prednisolone and cyclophosphamidereduced proteinuria from 6.5 g/d to 2.2 g/dand decreased serum creatinine from 132 µmol/l, to 96.8µmol/l. We showed obvious regression of crescents onthe light microscopy. Furthermore, we did not observeany side effects associated with treatment.In conclusion, although prospective studies comparing immunosupressive treatment with supportive one arewarranted, we believe that immunosupressive treatmentis useful in IgAN patients with poor risk factors for progression.

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