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Kidney Congress 2018: Clinical and histopathological characteristics of patients with glomerulonephritis syndrome in Dar Es Saalam, Tanzania - Paschal Ruggajo, Amira Deng - Renal Research Group, MUHAS Academic Medical Centre, Dar ES Salaam, Tanzania

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The histologic pattern of specific glomerulopathies and their related clinical presentation and varies according to age, sex, race, socioeconomic status and geographic location. The underlying histopathological pattern of patients presenting with glomerulonephritis syndrome in Tanzania is virtually unknown. This study was set to determine different clinicolaboratory and histopathological patterns of glomerulonephritis syndrome in Dar es Salaam Tanzania. Hospital based descriptive cross sectional study, all adults from (18yrs and above) with proteinuria and hematuria who underwent renal biopsy from April 2017- December 2017 were consecutively recruited into this study. Patients infected with HIV, hepatitis B virus and hepatitis C virus were excluded due to resources constraints. 55 participants with glomerulopathies were enrolled for this study, but 40 were eligible for percutaneous renal biopsy. Two-thirds of participants were female (67.5%) with mean age (±SD) of 32.7 (9.8) years. The commonest lesions were Focal segmental glomerulosclerosis (32.2%), followed by minimal Change disease (20.0%) and membranous nephropathy (17.5%). Membranoproliferative glomerulonephritis, IgA nephropathy and Lupus nephritis were about (5.0%) respectively. Among others histologic findings were renal amyloidosis was about (5.0%), inconclusive findings (10.0%) and undetermined due to excessive fibrosis (1%). Primary glomerulopathies in Tanzania occur more commonly among young (< 40 year) female patients presenting with glomerulonephritis syndrome. Focal segmental glomerulosclerosis (FSGS) is the most common primary glomerulopathy. There is considerable heterogeneity in the histologic spectrum of glomerulopathies which is influenced by age and gender factors.

The treatment and renal histopathological characteristics of 24 patients with IgA nephropathy showing urinary excretion of over 3.0 g/day of protein were investigated retrospectively. Clinically, the incidence of hematuria, nephrotic syndrome, renal dysfunction and hypertension was 100%, 54%, 88% and 42% in the patients, respectively. Histopathologically, although various grades of mesangial proliferation and crescent formation were observed, subepithelial dense deposits and mem-branolysis of the glomerular basement membrane were characteristically recognized by electron microscopy in nearly half of the 24 patients. These patients were administrated several kinds of drugs, such as corticosteroids (C), immunosuppressive agents (I) dipyridamole (D), C+D, I+D or C+I+D+heparin. The treatment with C+D or I+D was particularly effective for reducing the urinary protein and preventing deterioration of renal functions. However, none of the treatments were effec-tive in many patients whose creatinine clearance (Ccr) was below 50 ml/min. In conclusion, these results suggest that treatment of patients with IgA nephropathy showing massive proteinuria and a Ccr of above 50 ml/min should be actively attempted by a combination of immunosuppression therapy (C and/or I) and D.

Mesangioproliferative glomerulonephritis is the most common type of chronic glomerulonephritis (CGN). However, the clinical characteristics and prognosis are not fully understood in patients without Immunoglobulin A (IgA) deposition. To explore the clinical and pathological characteristics of patients with mesangioproliferative glomerulonephritis without IgA deposition (N-IgAN), we performed dual retrospective analyses. A single-center study was performed in 60 patients with biopsy-proven N-IgAN. 98 age- and sex-matched IgA nephropathy (IgAN) patients were randomly selected as a control group. The clinical and histopathological data at the time of renal biopsy were compared between N-IgAN and IgAN. In a second study, the data for 477 patients who had undergone maintenance renal replacement therapy (RRT) was collected and examined for the causal primary diseases. Duration from onset of renal symptoms to renal biopsy in patients with N-IgAN (71.2±123.3 months) was significantly longer than that in patients with IgAN

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(65.9±74.9 months) (p=0.0328). Urinary protein excretion in N-IgAN patients (0.6±1.1g/gCr) was significantly lower than that in IgAN (1.0 ± 1.3 g/gCr) (p < 0.0001). Ratio of global sclerosis, segmental sclerosis, crescents, interstitial mononuclear cell infiltration, interstitial fibrosis, and tubular atrophy were significantly lower in N-IgAN patients. Of the 477 patients who had undergone maintenance RRT, 95 patients had CGN (19.9%). Among them, 37 patients had received a renal biopsy, only one patient was N-IgAN (1%). It appears that N-IgAN can be recognized as a benign disease entity in comparison with IgAN.

Membranoproliferative glomerulonephritis (MPGN) diagnosis occurred in 79 (4.2%) patients out of 1849 with glomerulonephritis enrolled in Paulista Registry of Glomerulopathies from 1999 to 2005.1 This glomerulopathy is more often associated with secondary causes, especially infection by hepatitis C virus (HCV) and autoimmune diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis.2 However Little et al.3 identified 34% of idiopathic forms in a group of predominantly male patients aged 24.9 years with nephrotic syndrome and low levels of complement C3 fraction in half of them. Histologically, MPGN is characterized by mesangial proliferation, matrix expansion and its interposition between the endothelium and the glomerular basement membrane, giving the capillary loops a double-contour appearance. In classical studies MPGN was categorized in three types, according to the location of the electron-dense deposit on electron microscopy: type I (subendothelial deposits); type II (dense homogeneous intramembranous deposits); type III (a variant of type I, with subepithelial and subendothelial deposits). Type I was associated with HCV infection, whereas type II affects younger individuals and was not related to systemic causes. Sethi and Fervenza5 proposed a new system of classifying MPGN based on immunofluorescence (IF), defining two groups: MPGN with immunoglobulin deposition on IF that could be associated to autoimmune diseases, infections, or monoclonal gammopathy, whereas MPGN without immunoglobulin deposition but with C3 deposition on IF that is classified as dense deposit disease (DDD) or C3 glomerulonephritis after electron microscopy examination. In other study, MPGN with none deposition seen on IF was introduced with other group and could be secondary to membrane reactions, as in thrombotic microangiopathy.6 In this new classification the use of electron microscopy is mandatory in cases of exclusive C3 deposition, in order to differentiate DDD and C3 glomerulonephritis. The new classification proposed by Sethi and Fervenza emphasizes the various mechanisms involved in the pathogenesis of MPGN, drawing distinctions between MPGN mediated by immune complexes, which had immunoglobulin deposition, and MPGN resulting from abnormalities of the alternative complement pathway with C3 deposition only. Some authors believe that idiopathic forms should be very rare emphasizing complement and genetic studies in these patients in order to clear their diagnosis. C3 glomerulopathy defined after Sethi and Fervenza5 as a MPGN with exclusive C3 deposition allows pathological discussion. Pickering et al. proposed a less restrictive definition with deposition of dominant C3 at least two orders of magnitude more intense than any immunoglobulin. In doing so most cases of immune complex diseases would be excluded. Because of recent proposed classification, we aimed to conduct a retrospective single center study of MPGN patients grouped by the new IF-based classification to compare clinical and biochemical characteristics, renal biopsy dates and follow up of patients, as well as the etiology in each group.

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