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reviewers and the Colleges. You are encouraged to keep sending in your articles for review and publication in this distinguished and vastly informative journal.

PROF. G. E. ERHABOR

Editor-in-Chief

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HIV ASSOCIATED NEPHROPATHY

Human immunodeficiency virus infection can lead to progressive deterioration in renal function known as HIV-associated nephropathy (HIVAN). Importantly, individuals of African ancestry are more at risk than their European descent counterparts.

There is a wide geographical variation in the prevalence of HIV-associated nephropathy ranging from 38% to 48.5% worldwide. Few publications are available about prevalence in African countries. Despite the global recommendation and use of combine active antiretroviral therapy in treating HIV-infected patients, HIVAN is a leading cause of chronic renal disease in HIV-1 positive individuals.

HIVAN was described in persons living with AIDS in 1984 but earlier named AIDS-associated nephropathy. HIV-positive cases showed similar clinic pathological features. The nephropathy is more common amongst people of African descent, largely due to polymorphism in APOL1 gene. Two APOL1 risk alleles, G1 (containing two missense mutations, rs73885319 and rs60910145) and G2 (a frameshift deletion rs71785313), at the serum resistance-associated interacting-

domain-encoding region of APOL1 are associated with an increased susceptibility to developing HIVAN. Although individuals with HIVAN are predominantly males, studies have failed to show a direct correlation between gender or age and HIVAN.

Presentation is usually with low CD4 count, high viral load and massive proteinuria and renal insufficiency. Manifestations include hypoalbuminemia, with almost little or no peripheral oedema and hypertension, a very rapid progression to end-stage renal disease with normal or enlarged kidney sizes. Renal histology shows global or focal segmental glomerulosclerosis, degenerative and hypertrophic changes in visceral epithelial cells, mesangial deposits of C3, IgM and at times IgG, microcystic tubular dilatation containing plasma proteins, interstitial oedema and tubuloreticular inclusions in glomerular and peritubular endothelial cells. Significantly, *collapsing glomerulopathy* was not included in earlier reports until 1986. The histological lesions are however very similar to heroin-associated nephropathy or idiopathic FSGS.

Typically, there is glomerular

basement membranes collapse, with hypertrophy and hyperplasia of glomerular epithelial cells, and active tubulointerstitial disease indicated by microcystic tubular dilatation, interstitial inflammation and tubular injury. Microcystic changes are the most consistent renal findings in HIVAN and these lesions were responsible for the renal enlargement.

The estimated prevalence of HIVAN in Nigeria was estimated to be 77% and 76.7% in the age group of 21 – 40 years. Surprisingly, children with HIV are not protected against HIVAN. The prevalence of HIVAN in children in Nigeria was estimated to be 31.6% associated with high mortality. Risk factors in children include proteinuria, advanced disease, low CD4 count and the use of the combined highly active antiretroviral therapy (HAART).

The finding of HIVAN amongst children, who acquired HIV-1 through vertical transmission in the late 1980s, indicated the presence of HIV-related glomerulopathy that could evolve independently of intravenous drug use. As documented by the authors of '*HIV associated Nephropathy among Children with Renal Disease in Port*