



CODEN [USA]: IAJPBB

ISSN: 2349-7750

**INDO AMERICAN JOURNAL OF  
PHARMACEUTICAL SCIENCES**<http://doi.org/10.5281/zenodo.1495174>Available online at: <http://www.iajps.com>

Research Article

**A COMPREHENSION STUDY ON ASSOCIATION OF KIDNEY  
DISEASE WITH HIV INFECTION****<sup>1</sup>Dr. Ahsan Shahzad, <sup>2</sup>Dr. Mehwish Manzoor, <sup>3</sup>Dr. Maemoona Ahmad**<sup>1</sup>Medical Officer at BHU Patwali, Chakwal<sup>2</sup>Women Medical Officer at BHU Burj Attari, Sheikhpura<sup>3</sup>Shahbaz Sharif Mother & Children Complex, DHQ Hospital, Sheikhpura**Abstract:**

*HIV-positive individuals are at increased risk for a variety of renal disorders, including acute kidney injury (AKI), HIV-associated nephropathy (HIVAN), comorbid chronic kidney disease (CKD), and treatment-related kidney toxicity. HIVAN, the classic kidney disease of HIV infection, has become less common with widespread use of antiretroviral therapy (ART); simultaneously, however, the prevalence of other kidney diseases has increased. The basic aim of the study is to find an association of kidney disease with HIV infection. This study was conducted at Shahbaz Sharif Mother & Children Complex, DHQ Hospital, Sheikhpura during 2018. In this study we find an association of CKD with HIV infection. For this purpose we select the 100 patients of CKD who attend the hospital regularly. We develop a questionnaire for finding the knowledge of people about an association of CKD with HIV. It is concluded that the presence of kidney disease should be anticipated, and screening and proper interpretation of the relationship between serum creatinine level and GFR are recommended. Just as optimal control of HIV replication is achievable for most patients, so too is control of hypertension and diabetes.*

**Corresponding author:**

**Dr. Ahsan Shahzad,**  
Medical Officer at BHU Patwali,  
Chakwal

QR code



Please cite this article in press Ahsan Shahzad et al., *A Comprehension Study on Association of Kidney Disease with HIV Infection.*, Indo Am. J. P. Sci., 2018; 05(11).

**INTRODUCTION:**

HIV-positive individuals are at increased risk for a variety of renal disorders, including acute kidney injury (AKI), HIV-associated nephropathy (HIVAN), comorbid chronic kidney disease (CKD), and treatment-related kidney toxicity. HIVAN, the classic kidney disease of HIV infection, has become less common with widespread use of antiretroviral therapy (ART); simultaneously, however, the prevalence of other kidney diseases has increased. Before effective antiviral therapy became available, HIVAN was so frequent and its clinical features were so dramatic heavy proteinuria and rapid progression to end-stage renal disease (ESRD) in immunosuppressed black persons that HIVAN became almost synonymous with HIV-associated chronic kidney disease (CKD) [1]. As HIV spread through the black community, the ESRD incidence increased substantially, and by the early 1990s, HIVAN became the third leading cause of ESRD in black persons aged 20–64 years [2].

CKD is defined as kidney damage or reduced kidney function that persists for >3 months. A useful indicator of kidney damage is elevated urinary protein excretion, measured qualitatively with use of a urine dipstick or measured quantitatively with use of a spot urine protein-to-creatinine ratio (or 24-h urine collection). Kidney function can be reliably estimated from the serum creatinine by calculating the creatinine clearance or glomerular filtration rate (GFR) through use of the Cockcroft-Gault or Modification of Diet in Renal Disease (MDRD) equations, respectively [3]. A GFR <60 mL/min meets criteria for CKD, a cutoff supported by epidemiologic data linking lower GFR to an increased frequency of hospitalization, cardiovascular events, or death. Neither the Cockcroft-Gault nor the MDRD equations has been specifically validated in the HIV-infected population. The MDRD equation was derived from patients with low GFR and therefore can yield variable results in persons with normal renal function. Nonetheless, these estimates remain the most highly validated formulas available, and both equations are more sensitive than measurement of serum creatinine alone [4].

Race is an important risk factor for CKD. Black persons comprise 10% of the general population in the United States but account for >30% of patients with ESRD [3]. Young, male blacks have an 11-fold increased risk of CKD, compared with their white counterparts. Five new cases of ESRD develop for every 100 cases of CKD in black persons, whereas only 1 new ESRD case develops for every 100 cases of CKD in whites. The burden of kidney disease in black persons with HIV infection, compared with

their HIV-infected white counterparts, is similarly disproportionate. Among persons with HIV infection who receive dialysis, 91% are black [5].

**Aims and objectives**

The basic aim of the study is to find an association of kidney disease with HIV infection.

**MATERIAL AND METHODS:**

This study was conducted at Shahbaz Sharif Mother & Children Complex, DHQ Hospital, Sheikhpura during 2018. In this study we find an association of CKD with HIV infection. For this purpose we select the 100 patients of CKD who attend the hospital regularly. We develop a questionnaire for finding the knowledge of people about an association of CKD with HIV.

**Data collection**

A specific questionnaire was developed to determine viral hepatitis perception. This instrument was composed of two topics: demographic characteristics and viral hepatitis perception. Sociodemographic data included gender, age, education, and monthly family income.

**Ethical consideration**

This research project was approved by “Departmental Ethics and Research committee” of the hospital. The purpose of the study was explained to the study participants accordingly. Permission was obtained from hospitals research center and nephrology clinic.

**Statistical analysis**

Student’s t-test was performed to evaluate the differences in roughness between group P and S. Two-way ANOVA was performed to study the contributions. A chi-square test was used to examine the difference in the distribution of the fracture modes (SPSS 19.0 for Windows, SPSS Inc., USA).

**RESULTS AND DISCUSSION:**

HIVAN has a distinct histology, representing a collapsing form of focal segmental glomerulosclerosis (FSGS). The pathogenesis of HIVAN requires local HIV infection of the kidney, with the virus infecting tubular and glomerular epithelial cells. Although FSGS is the predominant glomerular lesion in HIVAN, other reported glomerular lesions in patients with HIV include IgA nephropathy, cryoglobulinemia, amyloidosis, and a lupuslike immune complex glomerulopathy.

In addition, as patients with HIV infection age, comorbid kidney diseases such as diabetic nephropathy and arterionephrosclerosis have become

increasingly common. When secondary FSGS develops in these patients, the potential overlap with HIVAN can be diagnostically challenging. Kidney biopsy is required to distinguish between these lesions.

Indinavir causes nephrolithiasis and chronic interstitial nephritis in as many as 12% of patients who receive it. The mainstay of prevention of this condition is adequate hydration, with intake of at least 1.5 L of noncaffeinated fluid. One report described 4 cases of renal colic and nephrolithiasis in patients who received lopinavir-ritonavir treatment, but a causal effect was not established [7]. Three cases of kidney stones containing atazanavir have been reported, and a review of the US Food and Drug Administration adverse event reporting system detected 12 additional confirmed cases [8]. Most cases required hospitalization for pain relief and stent insertion, percutaneous nephrostomy, lithotripsy, or endoscopic surgical extraction. Predisposing factors are unknown, and the drug was discontinued in most but not all cases.

The association of tenofovir with kidney disease has been an area of interest since the drug underwent preclinical testing, because of its structural similarity to adefovir and cidovir. These acyclic nucleotide analogues are excreted by renal tubule cell uptake and secretion [34]. Cidofovir at therapeutic doses can cause ARF, and a high incidence of ARF was noted when adefovir was tested for treatment of HIV-1 infection at dosages of 120 mg per day, which is 10-fold higher than the dosage for treating hepatitis B virus infection.

In fact, tenofovir-associated renal dysfunction is a rare event in prospective clinical trials, particularly among ART-naive patients. No significant change in GFR was demonstrated in a comparison of tenofovir versus stavudine in combination with lamivudine-efavirenz [9] or in a comparison of tenofovir-emtricitabine versus fixed-dose zidovudine-lamivudine with efavirenz in ART-naive patients during 144 weeks of treatment. The overall incidence of severe reduction in GFR was low (1.6%), and no difference in renal safety endpoints was detected between the regimens. [10]

### CONCLUSION:

It is concluded that the presence of kidney disease should be anticipated, and screening and proper interpretation of the relationship between serum creatinine level and GFR are recommended. Just as

optimal control of HIV replication is achievable for most patients, so too is control of hypertension and diabetes. The future holds enormous opportunities for research in new markers for early detection of kidney disease, prevention strategies, novel therapeutics, and a better understanding of the interaction between black race and kidney disease.

### REFERENCES:

1. Choi AI, Li Y, Parikh C, et al. Long-term clinical consequences of acute kidney injury in the HIV-infected. *Kidney Int.* 2010;78(5):478–85. Acute kidney injury in HIV-infected individuals was associated with progression of renal disease and death but not CVD.
2. Levey AS, Tangri N, Stevens LA. Classification of chronic kidney disease: a step forward. *Ann Intern Med.* 2011;154(1):65–7.
3. Khatua AK, Taylor HE, Hildreth JE, et al. Non-productive HIV-1 infection of human glomerular and urinary podocytes. *Virology.* 2010;408(1):119–27.
4. Sharma M, Callen S, Zhang D, et al. Activation of Notch signaling pathway in HIV-associated nephropathy. *AIDS.* 2010;24(14):2161–70.
5. Izzedine H, Acharya V, Wirden M, et al. Role of HIV-1 DNA levels as clinical marker of HIV-1-associated nephropathies. *Nephrol Dial Transplant.* 2011;26(2):580–3. The authors note that HIV-1 DNA determines the occurrence of HIVAN.
6. Izzedine H, Sene D, Cacoub P, et al. Kidney diseases in HIV/HCV-co-infected patients. *AIDS.* 2009;23(10):1219–26.
7. Nourse PJ, Cotton MF, Bates WD. Renal manifestations in children co-infected with HIV and disseminated tuberculosis. *Pediatr Nephrol.* 2010;25(9):1759–63.
8. Masia M, Enriquez R, Sirvent A, Gutierrez F, et al. Severe acute renal failure associated with rhabdomyolysis during treatment with raltegravir. A call for caution. *J Infect.* 2010;61(2):189–90.
9. Arendse CG, Wearne N, Okpechi IG, Swanepoel CR. The acute, the chronic and the news of HIV-related renal disease in Africa. *Kidney Int.* 2010;78(3):239–45.
10. Reese PP, Tehrani T, Lim MA, et al. Determinants of the decision to accept a kidney from a donor at increased risk for blood-borne viral infection. *Clin J Am Soc Nephrol.* 2010;5(5):917–23.