Original Article

Histological spectrum of renal disease in HIV/AIDS patients with significant proteinuria: An Indian perspective

Bhupendra Verma[1], Amrita Singh[2]

Departments of [1]Cardiology and [2]Nephrology, Ujala Hospital, Kashipur, Uttarakhand, India

Abstract

Background: Chronic kidney disease (CKD) has become epidemic in HIV/AIDS patients across Western and Eastern countries. HIV-associated nephropathy (HIVAN) has been consistently reported in studies from North America, Europe and African countries. However, studies from Asian countries are very sparse and differ strikingly in histological spectrum of renal disease, particularly in presence of HIVAN. **Objectives:** The study was carried out to in a teaching hospital from India to delineate the histological spectrum of renal disease and detect presence HIVAN in those with significant proteinuria (≥1gm/day). **Patients and Methods:** Urine analysis was done in 510 consecutive hospitalised HIV/AIDS patients after screening 640 such patients with age >18years. Patients with dipstick proteinuria ≥1+ were subjected to 24-hour urinary protein estimation. Renal biopsy was done in 10 patients with proteinuria ≥1gm/day. **Results:** Dipstick proteinuria ≥1+ was present in 29% patients. In patients undergoing kidney biopsy, the most frequent glomerular lesion was mesangial proliferative glomerulonephritis (30%) followed by HIVAN (20%). Tubulo-interstitial lesions were seen in 60% of biopsies. Pooled analysis of all the available kidney biopsy series from India revealed prevalence of HIVAN to be 16.5%. **Conclusion:** Contrary to the popular belief, HIVAN appears to be a common entity in this part of world too. High degree of clinical suspicion is required as diagnosis of HIVAN caries higher morbidity and mortality. Moreover, an early diagnosis and timely management can improve prognosis in such patients.

Keywords: Glomerular lesion, histopathology, HIVAN, kidney biopsy, renal biopsy

Introduction

Chronic kidney disease has become epidemic in HIV/AIDS patients worldwide, especially being common in Black population.[1] Kidney pathology is broadly divided into glomerular disease, tubulointerstitial, and vascular diseases.[2] Glomerular disease directly related to HIV infection, HIV-associated nephropathy (HIVAN), is well established in North America, Africa and Western Europe.[3] In Western and African studies up to 60% of all renal biopsies performed for HIV positive patients reveal HIVAN. HIVAN is a collapsing form of focal glomerulosclerosis (FSGS) associated with microcystic dilatation and tubulointerstitial injury. It usually presents with rapidly progressing renal dysfunction and high-grade proteinuria, often associated with high mortality.[4] The incidence of HIVAN has declined since the introduction of cART (combination antiretroviral therapy), whereas incidence of non-HIV-associated chronic kidney diseases (CKD) has increased.[5]

In view of high burden of renal diseases in HIV-infected patients, the Infectious Diseases Society of America (IDSA) recommends screening for kidney disease using urinalysis and estimation of renal function.[6] These guidelines further recommend proteinuria assessment using quantitative methods to allow

Address for correspondence: Dr. Bhupendra Verma, General Physician and Consultant Cardiologist, Department of Cardiology, Ujala Hospital, Kashipur - 244713, Udham Singh Nagar, Uttarakhand, India.
E-mail: bhupendra.269@gmail.com

Access this article online

Quick Response Code:

Website: www.jfmpc.com

DOI: 10.4103/jfmpc.jfmpc_104_19

How to cite this article: Verma B, Singh A. Histological spectrum of renal disease in HIV/AIDS patients with significant proteinuria: An Indian perspective. J Family Med Prim Care 2019;8:860-5.

earlier identification of potential kidney disease. Renal biopsy is advocated wherever feasible, because the treatment options and prognosis are influenced by the actual histological diagnosis. While HIVAN has been reported consistently in Western studies, the prevalence in studies from Asia has been found to low/absent.[7,8] The available studies from India differ strikingly, with some studies showing presence of HIVAN,[9-11] whereas others reporting complete absence of this entity.[8,12,13] With this perspective, the present study was carried out to elucidate the histological spectrum of renal disease in HIV patients from a tertiary care center from India. Kidney biopsies were performed in HIV patients with proteinuria ≥1 g/day, to delineate the glomerular lesions including presence of possible HIVAN.

Materials and Methods

Study population and protocol

This was a single center observational study done at the Institute of Medical Sciences (IMS), Banaras Hindu University (BHU), Varanasi, India. All consecutive HIV positive patients of ≥18 years of age, admitted in general medicine or nephrology departments, were included in the study. The participants were subjected to detailed history, physical examination, and laboratory investigation. The urine was tested using standard dipstick method for screening of proteinuria. The patients having a dipstick proteinuria of 1+ or more were subjected to quantitative 24-hour urinary protein estimation. Percutaneous ultrasound-guided kidney biopsy was done in patients with proteinuria of ≥1 g/day. The study was conducted according to the Helsinki Declaration and the Good Clinical Practice Guidelines. Written informed consent was obtained from all patients. Ethical approval for the study protocol was obtained from Ethics Committee of IMS, BHU.

Kidney biopsy and histopathology

Kidney biopsy was done in HIV positive patients with proteinuria of ≥1 g/day, after proper counseling and taking written informed consent. Biopsy was contraindicated in patients with small/contracted kidneys, single kidney, polycystic kidney disease, hydronephrosis, and presence of urinary tract infection. Blood pressure was adequately controlled in hypertensive patients before biopsy. Biopsy was performed under ultrasound guidance with a biopsy gun (BARD 16/18 G, 22 mm, cutting edge) with the use of local anesthesia. The patients were discharged after observation for 6 hours, if the urine was clear. Two cores were taken by biopsy and the tissue was placed in 10% formalin for light microscopic (LM) examination. The tissue was studied using H and E (hematoxylin and eosin), PAS (periodic acid Schiff), and AFOG (acid fuschin orange green) stains. Immunofluorescence (IF) and electron microscopy (EM) was not done as per the institute's policy.

Definitions

Diagnosis of HIV and AIDS were based on 1993 revised classification system by CDC (Centers for Disease Control).[14] CKD was defined as kidney damage (structural or functional abnormalities of the kidney) or estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2 for 3 months or more, irrespective of cause.[15] Significant proteinuria for the purpose of kidney biopsy was defined as ≥1 g/day. Nephrotic range proteinuria was defined as a 24-hour urine protein ≥3.5 g/day.

Results

Study population

A total of 510 consecutive hospitalized HIV/AIDS patients were included in the study after screening 640 such patients [Figure 1]. Dipstick proteinuria of grade ≥1+ was seen in 147 patients (29% of total). 24-hour urinary protein estimation was done in all these patients. Only 15 patients (10.2%) had proteinuria >1 g/day, and they were eligible for kidney biopsy. However, renal biopsy could not be done in five patients, as two patients didn't consent and urinary tract infection, contracted kidney, and single kidney was observed in one each patient.

Renal histopathology

Renal biopsy was studied by light microscopy in 10 patients [Table 1]. The classical HIV associated nephropathy (HIVAN) was seen in two patients (20%). Figure 2 shows a classic case of HIVAN, with features of collapsing FSGS (focal segmental glomerulosclerosis), microcystic tubular dilatation, tubules filled with cast and interstitial infiltrates. Most common glomerular lesion was mesangial proliferative glomerulonephritis (MesPGN), found in 3 cases (30%). Diffuse proliferative glomerulonephritis (DPGN), membranoproliferative glomerulonephritis (MPGN) and diabetic nephropathy were seen in one patient each. Figure 3 shows the histopathological features in the patient with MPGN, also known

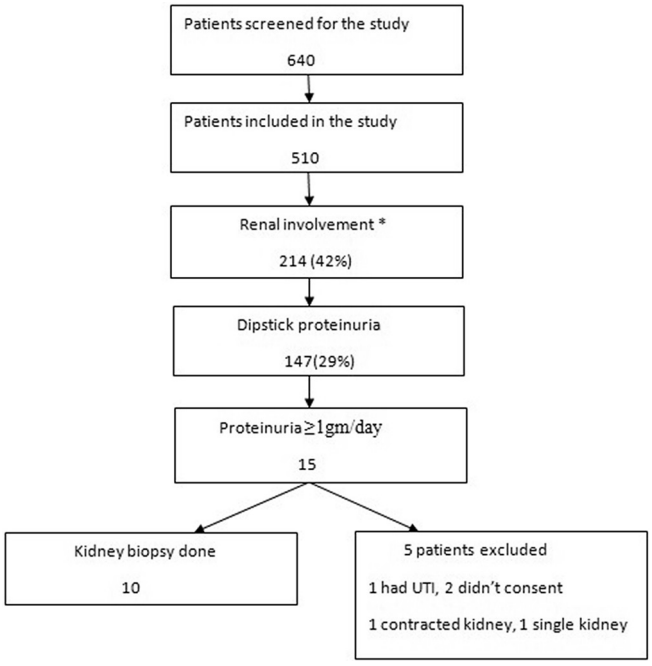

Figure 1: Flow chart showing the study plan. *defined as dipstick proteinuria ≥1 + and/or serum creatinine ≥1.5 mg/dl

Table 1: Renal histopathology in HIV patients with proteinuria ≥1 gm/day (10 patients)

Age/sex	Proteinuria	Creatinine	CD4 count	c-ART	Histopathology (predominant lesions)
34/M	3.8	2.3	108	no	HIVAN with CIN
28/M	4.2	1.8	38	no	HIVAN
36/M	2.5	1.6	230	yes	Diabetic nephropathy with IN
26/F	1.4	0.9	178	yes	Focal MesPGN with IN
41/M	3.9	1.2	310	no	Focal MesPGN
32/F	2.1	2.5	325	yes	Diffuse MesPGN
27/F	1.1	0.8	140	yes	MPGN with CIN
33/M	2.3	1.3	258	no	DPGN
22/M	1.7	3.4	48	no	CIN
29/F	2.3	2.6	122	yes	CIN

cART: Combination anti-retroviral therapy; Mes PGN: Mesangioproliferative glomerulonephritis; MPGN: Membranoproliferative glomerulonephritis; DPGN: Diffuse proliferative glomerulonephritis; MN: Membranous nephropathy; IN: Interstitial nephritis; CIN: Chronic interstitial nephritis; HIVAN: Human immunodeficiency virus associated nephropathy

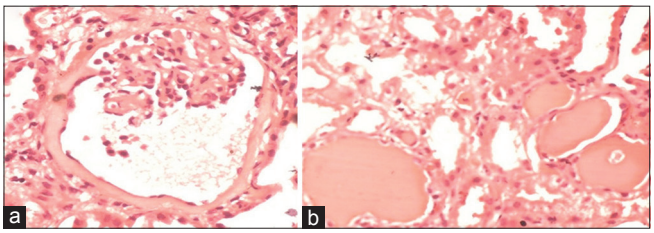

Figure 2: Classical features of HIVAN a) collapsing focal segmental glomerular sclerosis (H and E × 400); b) Showing microcystic dilatation of tubules. These tubules are filled with the hyaline cast. (H and E × 400)

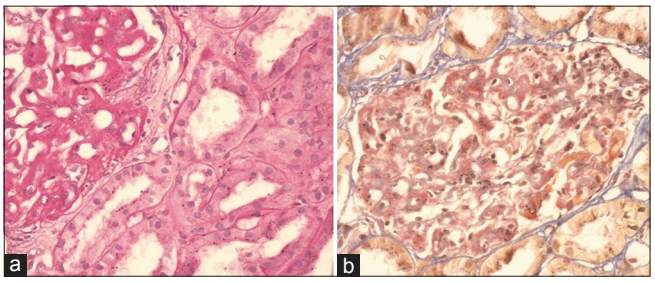

Figure 3: Histopathology in MPGN a) severe GBM thickening and its duplication along with proliferation (PAS × 400); b) increased leucocyte, mesangial cell proliferation, and heavy subendothelial and mesangial deposits. (AFOG × 400)

as mesangiocapillary glomerulonephritis. Tubulointerstitial lesions were seen in 2 cases in isolation (CIN, chronic interstitial nephritis), and in 4 cases combined with other glomerular lesions (overall 6 patients, 60%). Ultrasonography of abdomen revealed enlarged kidneys in seven patients (70%), which included both patients of HIVAN.

Discussion

We have presented a single center, observational study on histological spectrum of renal disease in HIV/AIDS patients. Renal histopathology in 1o patients with proteinuria ≥1 g/day has been described with particular attention towards presence of HIVAN.

HIVAN is associated with characteristic glomerular, tubulointerstitial, and ultrastructural lesions. The most consistent findings include collapsing FSGS, cystic tubular dilatation, interstitial infiltrates, and dilated tubules filled with proteinaceous casts. Immunofluorescence is nonspecific and ultrastructural changes on electron microscopy are not unique to HIVAN.[16] The pathogenesis of HIVAN involves local infection of the kidney, systemic HIV infection and systemic immune dysfunction.[17] Patients with HIVAN typically present with nephrotic range proteinuria and rapidly progressive renal insufficiency, accompanied by varying degrees of azotemia.[18] Most patients with HIVAN are young men (mean age 33 years; male to female ratio of 10:1) and >90% of patients are Blacks.[19,20] HIVAN is associated with large echogenic kidneys, but this relation is non-specific.[21] Both of our patients with HIVAN were young males presenting with nephrotic range proteinuria, CD4 count <200 cells/mm^3 and large echogenic kidneys on ultrasound. The absence of both nephrotic range proteinuria and CD4 count <200 cells/mm^3, are useful to exclude diagnosis of HIVAN, with a negative predictive value of 90%.[22] However, HIVAN can be definitively diagnosed only by kidney biopsy.[6]

The distribution of HIVAN is not uniform and the true prevalence is not known, mainly due to relatively infrequent kidney biopsies. In an autopsy study of HIV-infected persons the overall prevalence of HIVAN was 6.9%.[23] Screening study for HIVAN in HIV patients with proteinuria >1.5 g/day found an overall prevalence of 3.5%.[24] Prevalence of HIVAN in kidney biopsy series outside India have reported prevalence of HIVAN ranging from 0% to 83% [Table 2].[7,25-33] HIVAN has been consistently and frequently reported in studies from North America, Western Europe and African countries. However, studies from Italy and Thailand have shown complete absence of this entity.[7,32] The prevalence of HIVAN in our study was 20%, with the most common glomerular lesion being mesangioproliferative glomerulonephritis (30%) [Table 1]. MesPGN is an inconsistent finding American, African, and European studies, but represents a substantial proportion in Asia accounting for 1/3rd to 2/3rd of all renal lesions.[7,10] The probable cause of this discrepancy may lie in racial predisposition, viral genotype, environmental factors, and host susceptibility factors. On the contrary, tubulointerstitial lesions are consistently seen in all kidney biopsy series across world, and may be present in up to 70% cases.[12,13,30,33]

Kidney biopsy series in HIV patients from India, have reported wide variability in prevalence of HIVAN, ranging from 0% to 85% [Table 3].[8-13,34,35] These widely variable results emphasize limitations on drawing conclusions from single-center studies with small sample sizes. Therefore, we did a systemic review of literature on kidney biopsy in adult HIV patients from India. Search was made on Pubmed, Scopus, and Google Scholar, excluding case reports. We pooled biopsy data from all the 10 available biopsy series (including the current study). Among 194 patients, the most frequent glomerular lesion was HIVAN (16.5%) followed by MesPGN (13.9%) and FSGS in 7.7%. Pooling of studies though has obvious limitations, gives a fair degree of insight into the histological spectrum. The current study and pooled analysis show that HIVAN, though less common than Western countries, still has an appreciable prevalence in HIV patients from India. This is in contrast to popular belief that HIVAN is rare in Asian countries. Urinalysis should therefore be done in all HIV/AIDS patients and quantification of proteinuria should be performed wherever feasible.[2] Kidney biopsy, being the only definite way to diagnose HIVAN, must be done in all patients with significant proteinuria. Diagnosis of HIVAN is very essential as it carries both prognostic and therapeutic implications. Several studies have reported significant improvements in renal function and proteinuria for patients with HIVAN receiving cART, corticosteroids, and ACE inhibition.[6]

Table 2: Prevalence of HIVAN in kidney biopsies of HIV/AIDS patients in major studies across world

Study	Region	Year	No. of cases	Prevalence of HIVAN
D'agati *et al.* (63)	USA	1997	112	64.7%
Szczech *et al.* (28)	USA	2002	89	47.2%
Berliner *et al.* (64)	USA	2008	152	35%
Gerntholtz *et al.* (65)	S Africa	2006	104	27%
Han *et al.* (66)	S Africa	2006	30	83%
Nochy *et al.* (67)	France	1993	60	43%
Gutiérrez *et al.* (68)	Spain	2007	27	14.8%
Williams *et al.* (69)	London	1998	17	40%
Casanova *et al.* (66)	Italy	1995	26	None
Cavalcante *et al.* (29)	Brazil	2007	6	50%
Praditpornsilpa *et al.* (17)	Thailand	1999	26	None
Present Study	India		10	20%

HIVAN: Human immunodeficiency virus associated nephropathy

Implication for primary care/family physicians/healthcare professionals

In view of rising prevalence of renal disorders in HIV/AIDS patients, urinalysis, and estimation of renal function must be done all patients. Contrary to prior belief, HIVAN has an appreciable prevalence in HIV patients with proteinuria in this region. Renal biopsy is recommended wherever feasible, because the treatment options and prognosis are influenced by the actual histological diagnosis.

Study Limitations

The most important limitation was small number of patients undergoing kidney biopsy. This was because we have limited renal biopsy only to patients with proteinuria ≥1 g/day, as HIVAN most often presents with significant degrees of proteinuria. Second, we have not performed immunofluorescence (IF) and electron microscopy (EM) studies in the biopsy samples. However, this is unlikely to have affected the finding of HIVAN in our study, because its diagnosis is mainly based on light microscopy as IF and EM findings are non-specific.[16] But, this may have caused failure to detect cases of immune complex diseases. Finally, the results of this study are subject to confounding and bias that are inherent to all observational studies.

Conclusions

With improvement in survival due to availability of cART, renal involvement has become quite common in HIV/AIDS patients. Proteinuria as detected by dipstick was present in 29% patients and of this proteinuria ≥1 g/day was seen in 10.2%. We have reported a prevalence of HIVAN in 20% of kidney biopsies. HIVAN is associated with rapid deterioration in renal function and carries a high mortality, but has good response to cART, steroids, and ACE inhibition. Moreover, unlike popular belief, HIVAN appears to be a common entity

Table 3: Renal histopathology in HIV/AIDS patients in Indian studies

Study	Year	No. of cases	Renal Histopathology
Madiwale (72)	1999	20	HIVAN 85%, MPGN 5%, MCD 5%, Lupus Nephritis 5%
Varma *et al.*(20)	2000	25	Mes PGN (32%), FSGS (16%), HIVAN (4%)
Janakiraman *et al.* (21)	2008	10	HIVAN 70%, DPGN 10%, MN 10%, CIN 10%
Vali *et al.* (19)	2012	27	HIVAN (Collapsing FSGS) 11.1%, FSGS 7.4%, DPGN 7.4%, IN 30%
Gupta *et al.* (18)	2013	26	MesPGN 38%, Collapsing FSGS 7%, MPGN 7%, IN 19%
Sunil *et al.* (71)	2016	32	HIVAN (6%), MPGN (6%), DN (6%), MesPGN (6%), FSGS (3%), IN (19%)
Prakash *et al.* (23)	2017	14	MesPGN 31.2%, FSGS 12.5%, MPGN 12.5%, DPGN 12.5%, IN 71%
Satish *et al.* (22)	2018	30	DN 23.3%, FSGS 13.3%, IgAN 10%, DPGN 6.6%, IN 26.6%
Present Study		10	MesPGN 30%, HIVAN 20%, MPGN 10%, DPGN 10%, DN 10%, IN 60%
Pooled analysis* (10 studies)		194	HIVAN 16.5%, MesPGN 13.9%, FSGS 7.7%, DPGN 4.1%, MPGN 4.1% Tubulo-interstitial lesions: 22.7%

Mes PGN: Mesangioproliferative glomerulonephritis; MPGN: Membranoproliferative glomerulonephritis; FSGS: Focal segmental glomerulosclerosis; AIN: Acute interstitial nephritis; DPGN: Diffuse proliferative glomerulonephritis; MN: Membranous nephropathy; CIN: Chronic interstitial nephritis; HIVAN: Human immunodeficiency virus associated nephropathy; MCD: Minimal change disease; DiabeticN : Diabetic nephropathy; IgAN: IgA nephropathy. *Pooled kidney biopsy data including all observational studies from India

in this part of world too, and thereby requires a high degree of clinical suspicion. Kidney biopsy, being the only definite way to diagnose HIVAN, must be done in all patients with significant proteinuria.

Authors' contribution

BV; concept, study design, experiments, data acquisition, analysis of information, and draft of the manuscript. AS; data analysis, information analysis, design, and approval of final manuscript.

Acknowledgements

The authors would like to thank Prof Jai Prakash, Dept of Nephrology, IMS, BHU; Prof Usha Singh, Dept of Pathology, IMS, BHU; and Prof S S Agarwal, Dept of Internal Medicine and ART Centre, IMS, BHU for their valuable guidance and support throughout the thesis work.

Financial support and sponsorship

This article is part of the thesis work in internal medicine specialty by Dr. Bhupendra Verma at IMS, BHU, India. However, there was no funding/financial support.

Conflicts of interest

There are no conflicts of interest.

References

1. Fine DM, Perazella MA, Lucas GM, Atta MG. Kidney biopsy in HIV: Beyond HIV associated nephropathy. Am J Kidney Dis 2008;15:504-14.
2. Swanepoel CR, Atta MG, D'Agati VD, Estrella MM, Fogo AB, Naicker S, *et al.* Kidney disease in the setting of HIV infection: Conclusions from a kidney disease: Improving global outcomes (KDIGO) controversies conference. Kidney Int 2018;93:545-59.
3. Ross MJ, Klotman PE. Recent progress in HIV- associated nephropathy. J Am Soc Nephrol 2002;13:2997-3004.
4. Phair J, Palella F. Renal disease in HIV infected Individuals. Curr Opin HIV AIDS 2011;6:285-9.
5. Mocroft A, Kirk O, Gatell J, Reiss P, Gargalianos P, Zilmer K, *et al.* Chronic renal failure among HIV-1-infected patients. AIDS 2007;21:1119-27.
6. Gupta SK, Eustace JA, Winston JA, Boydstun II, Ahuja TS, Rodriguez RA, *et al.* Guidelines for the management of chronic kidney disease in HIV-infected patients: Recommendations of the HIV medicine association of the infectious diseases society of America. Clin Infect Dis 2005;40:1559-85.
7. Praditpornsilpa K, Napathorn S, Yenrudi S, Wankrairot P, Tungsaga K, Sitprija V. Renal pathology and HIV infection in Thailand. Am J Kidney Dis 1999;33:282-6.
8. Gupta V, Gupta S, Sinha S, Sharma SK, Dinda AK, Agarwal SK, *et al.* HIV associated renal disease: A pilot study from north India. Indian J Med Res 2013;137:950-6.
9. Vali PS, Ismal K, Gowrishankar S, Sahay M. Renal disease in human immunodeficiency virus-Not just HIV-associated nephropathy. Indian J Nephrol 2012;22:98-102.
10. Varma PP, Prasher PK, Deshpande GU, Mani NS, Nema SK, Sayal SK. Spectrum of renal lesions in HIV patients. J Assoc Physicians India 2000;48:1151-4.
11. Janakiraman H, Abraham G, Matthew M, Kuruvilla S, Panikar V, Solomon S, *et al.* Correlation of CD4 counts with renal disease in HIV positive patients. Saudi J Kidney Dis Transpl 2008;19:603-7.
12. Satish S, Nadig DM, Vankalakunti M, Sanjeev Shetty MS. Renal disease in human immunodeficiency virus infection; a divergent array. J Nephropathol 2018;7:255-62.
13. Prakash J, Ganiger V, Prakash S, Sivasankar M, Sunder S, Singh U. Kidney disease in human immunodeficiency virus-seropositive patients: Absence of human immunodeficiency virus-associated nephropathy was a characteristic feature. Indian J Nephrol 2017;27:271-6.
14. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR Recomm Rep 1992;41:1-19.
15. KDIGO, CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation, prevention and management of chronic kidney disease-mineral and bone disorder. Kidney Int 2013;3:1-150.
16. Markowitz GS, Appel GB, Fine PL, Fenves AZ, Loon NR, Jagannath S, *et al.* Collapsing focal segmental glomerulosclerosis following treatment with high-dose pamidronate. J Am Soc Nephrol 2001;12:1164-72.
17. Wyatt CM. Kidney disease and HIV infection. Top Antivir Med 2017;25:13-6.
18. Rao TK. Human immunodeficiency virus infection and renal failure. Infect Dis Clin North Am 2001;15:833-50.
19. Wyatt CM, Klotman PE, D'Agati VD. HIV-associated nephropathy: Clinical presentation, pathology, and epidemiology in the era of antiretroviral therapy. Semin Nephrol 2008;28:513-22.
20. Di Fiori JL, Rodrigue D, Kaptein EM, Ralls PW. Diagnostic sonography of HIV-associated nephropathy: New observations and clinical correlation. AJR Am J Roentgenol 1998;171:713-6.
21. Atta MG, Choi MJ, Longenecker JC, Haymart M, Wu J, Nagajothi N, *et al.* Nephrotic range proteinuria and CD4 count as noninvasive indicators of HIV-associated nephropathy. Am J Med 2005;118:1288.
22. Shahinian V, Rajaraman S, Borucki M, Grady J, Hollander WM, Ahuja TS. Prevalence of HIV-associated nephropathy in autopsies of HIV-infected patients. Am J Kidney Dis 2000;35:884-8.
23. Ahuja TS, Borucki M, Funtanilla M, Shahinian V, Hollander M, Rajaraman S. Is the prevalence of HIV-associated nephropathy decreasing? Am J Nephrol 1999;19:655-9.
24. D'Agati V, Appel GB. HIV infection and the kidney. J Am Soc Nephrol 1997;8:138-52.
25. Szczech LA, Gange SJ, van der Horst C, Bartlett JA, Young M, Cohen MH, *et al.* Predictors of proteinuria and renal failure among women with HIV infection. Kidney Int 2002;61:195-202.
26. Cavalcante MA, Coelho SN, Lacerda HR. Prevalence of persistent proteinuria in stable HIV/AIDS patients and its association with HIV nephropathy. Braz J Infect Dis 2007;11:456-61.
27. Berliner AR, Fine DM, Lucas GM, Rahman MH, Racusen LC, Scheel PJ, *et al.* Observations on a cohort of HIV-infected patients undergoing native renal biopsy. Am J Nephrol 2008;28:478-86.

28. Gerntholtz TE, Goetsch SJ, Katz I. HIV-related nephropathy: A South African perspective. Kidney Int 2006;69:1885-91.
29. Han TM, Naicker S, Ramdial PK, Assounga AG. Cross-sectional study of HIV-seropositive patients with varying degrees of proteinuria in South Africa. Kidney Int 2006;69:2243-50.
30. Nochy D, Glotz D, Dosquet P, Pruna A, Guettier C, Weiss L, *et al*. Renal disease associated with HIV infection: A multicentric study of 60 patients from Paris hospitals. Nephrol Dial Transplant 1993;8:11-9.
31. Gutiérrez E, Morales E, Gutiérrez Martínez E, Manzanares MJ, Rosello G, Mérida E, *et al*. Glomerulopathies associated to HIV infection: A Spanish perspective. Nefrologia 2007;27:439-47.
32. Williams DI, Williams DJ, Williams IG, Unwin RJ, Griffiths MH, Miller RF. Presentation, pathology, and outcome of HIV-associated renal disease in a specialist centre for HIV/AIDS. Sex Transm Infect 1998;74:179-84.
33. Casanova S, Mazzucco G, Barbiano di Belgiojoso G, Motta M, Boldorini R, Genderini A, *et al*. Pattern of glomerular involvement in human immunodeficiency virus-infected patients: An Italian study. Am J Kidney Dis 1995;26:446-53.
34. Sunil R, Sandeep R, Mahesh V, Chethana R, Reddy SS, Subbarao KV. Changing spectrum of renal disease in HIV. Int J Med Res Health Sci 2016;12:31-5.
35. Madiwale C, Venkataseshan VS. Renal lesions in AIDS: A biopsy and autopsy study. Indian J Pathol Microbiol 1999;42:45-54.