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Research Article

CLINICAL SPECTRUM, ETIOLOGY AND ASSOCIATED FACTORS OF ACUTE KIDNEY INJURY IN HIV-INFECTED PATIENTS: A PROSPECTIVE OBSERVATIONAL STUDY

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Abstract

Background: Acute kidney injury (AKI) is characterised by a rapid decline in renal function and is associated with poor outcomes. People living with HIV are at increased risk of AKI through HIV-dependent factors, HIV-independent factors and nephrotoxic drugs, particularly tenofovir. Identifying those at risk and the predominant causes can guide prevention and management.

Objectives: To determine the incidence, clinical spectrum, etiology and associated factors of AKI in HIV-infected patients at a tertiary-care centre.

Methods: This single-centre, prospective observational study over two years enrolled 103 HIV-infected adults with AKI (KDIGO definition). Patients with chronic kidney disease or prior renal transplant were excluded. History, examination and laboratory evaluation (including CD4 count, serum albumin and renal function) were recorded; AKI was staged by AKIN criteria and etiology assigned. Data were analysed with SPSS v22; $p<0.05$ was significant.

Results: The incidence of AKI among HIV admissions was 7.5% (103/1361). Mean age was 46.7 ± 10.5 years; 59.2% were men and 40.8% belonged to the 41–50 year group. The cohort was characterised by low CD4 count (<250/mm³ in 74.7%; mean 213 ± 99), hypoalbuminaemia (<3.5 g/dL in 92.2%), anaemia (Hb <10 g/dL in 83.4%) and low BMI (<18.5 kg/m² in 66.0%). Most patients presented in AKIN stage 3 (57.3%) or stage 2 (40.8%). The commonest etiologies were tenofovir-induced nephropathy (26.2%),

multifactorial causes (25.2%), sepsis (22.3%) and dehydration (20.4%); nephrotoxic drugs other than tenofovir (4.9%) and obstructive uropathy (1.0%) were uncommon.

Conclusion: AKI is an important cause of admission among HIV-infected patients (incidence 7.5%). Tenofovir-induced nephropathy was the leading etiology, followed by multifactorial causes, sepsis and dehydration. AKI was associated with male sex, age 41–50 years, low CD4 count, low BMI and hypoalbuminaemia. Judicious use of tenofovir and early attention to sepsis and volume status are warranted.

Keywords: HIV; Acute kidney injury; Tenofovir; CD4 count; Serum albumin; Sepsis.

INTRODUCTION

Human immunodeficiency virus (HIV) infection remains a major public-health challenge in India, which has one of the largest populations of people living with HIV in the world.[1] With the widespread availability of antiretroviral therapy (ART), survival has improved and the clinical focus has shifted towards non-AIDS complications, including renal disease.

Acute kidney injury (AKI) is characterised by an abrupt decline in glomerular filtration and is associated with prolonged hospitalisation, progression to chronic kidney disease and increased mortality. HIV-infected patients are at heightened risk of AKI through several mechanisms: HIV-dependent factors (HIV-associated nephropathy, immune-complex disease, opportunistic infections), HIV-independent factors (sepsis, volume depletion, hypotension) and nephrotoxic drugs.[2],[3] Among antiretrovirals, tenofovir disoproxil fumarate is a well-recognised cause of proximal tubular injury and nephropathy.[4] Advanced immunodeficiency, reflected in a low CD4 count, has also been identified as a risk factor for AKI in this population.[5],[6]

The increased risk of AKI in HIV-infected patients and its adverse impact on prognosis underline the need to identify those at risk, characterise the predominant causes and inform prevention strategies. We therefore studied the incidence, clinical spectrum, etiology and associated factors of AKI in HIV-infected patients at our centre.

MATERIAL AND METHODS

Study design, setting and participants

This single-centre, prospective observational study was carried out over two years in the Departments of General Medicine and Nephrology of a tertiary-care teaching hospital that serves as a regional nodal centre for HIV/AIDS care. HIV-infected/AIDS patients older than 18 years who were admitted with AKI, defined per the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, were enrolled after informed consent.[7] Patients with pre-existing chronic kidney disease and those who had undergone renal transplantation were excluded.

AKI was defined by KDIGO criteria as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, or to ≥1.5 times baseline within the prior 7 days, or urine output <0.5 mL/kg/h for 6 hours, and was staged using the Acute Kidney Injury Network (AKIN) classification.[7],[8]

Sample size

The sample size was calculated as $4pq/d^2$, with p = prevalence of AKI among HIV

patients at the centre (5.5%), q = 1 − p and absolute precision d = 0.05, giving a minimum of 84; 103 patients were enrolled.

Assessments

Each patient underwent a structured history, general and systemic examination and laboratory evaluation, including complete blood count, renal function tests, serum electrolytes, random blood sugar, serum albumin, CD4 count, HBsAg, anti-HCV, 24-hour urine protein, urine routine, urine culture and sensitivity, liver function tests and ultrasonography of the abdomen. ART regimens were recorded per national guidelines, under which a tenofovir–lamivudine–efavirenz (TLE) regimen is first-line for most adults.[9] The etiology of AKI was assigned after clinical, laboratory and imaging assessment.

Statistical analysis

Data were entered in Microsoft Excel and analysed with SPSS version 22 (IBM Corp.). Categorical variables were expressed as frequencies and proportions and compared with the chi-square test; continuous variables were expressed as mean ± standard deviation and compared with the independent-samples t test. A p value <0.05 was considered statistically significant.

Ethics

The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from every participant.

RESULTS

During the study period, 1361 HIV-infected patients were admitted, of whom 103 developed AKI, giving an incidence of 7.5% (103/1361).

The mean age was 46.7 ± 10.5 years, and the largest group (40.8%) was aged 41–50 years. There were 61 men (59.2%) and 42 women (40.8%), a male-to-female ratio of 1.45:1. The mean duration since HIV diagnosis was 4.6 years; 8 patients (7.8%) were newly detected, and the majority presented within the first two years of diagnosis. Demographic, clinical and laboratory characteristics are shown in Table 1.

The cohort showed advanced immunosuppression and poor nutritional status: the mean CD4 count was 213 ± 99 cells/mm³, with 74.7% having a CD4 count below 250 cells/mm³; hypoalbuminaemia (<3.5 g/dL) was present in 92.2% (mean albumin 2.45 ± 0.62 g/dL); anaemia (Hb <10 g/dL) in 83.4% (mean 8.6 ± 1.3 g/dL); and low BMI (<18.5 kg/m²) in 66.0% (mean 18.3 ± 1.3 kg/m²). The prevalence of these features is shown in Figure 3. Most patients (86.4%) were on a TLE-based ART regimen. The mean serum creatinine at presentation was 3.63 ± 1.66 mg/dL against a mean baseline of 0.98 ± 0.64 mg/dL.

Reduced urine output was the commonest presenting symptom (75.7%), followed by fever (39.8%), vomiting (38.8%), bilateral lower-limb swelling (36.9%), loose stools (28.2%) and breathlessness (19.4%). Pulmonary tuberculosis was the commonest comorbidity (19.4%), followed by systemic hypertension (12.6%), other opportunistic infections (9.7%), extrapulmonary tuberculosis (5.8%) and diabetes mellitus (5.8%).

By AKIN staging, most patients presented late: 57.3% in stage 3, 40.8% in stage 2 and only 1.9% in stage 1 (Figure 2).

The etiology of AKI is summarised in Table 2 and Figure 1. Tenofovir-induced

nephropathy was the leading cause (27 patients, 26.2%), followed by multifactorial causes (26, 25.2%), sepsis (23, 22.3%) and dehydration (21, 20.4%); nephrotoxic drugs other than tenofovir (5, 4.9%) and obstructive uropathy (1, 1.0%) were uncommon. Among the 23 patients with sepsis-related AKI, the commonest sources were urinary tract infection (10) and bronchopneumonia (9). Multifactorial AKI (n=26) most often combined dehydration with sepsis (9) or tenofovir with obstructive uropathy (7), tenofovir with antitubercular therapy (6), or tenofovir with co-trimoxazole or an NSAID (2 each). Among the 5 patients with AKI from other nephrotoxic drugs, amphotericin B accounted for 4 and amikacin for 1

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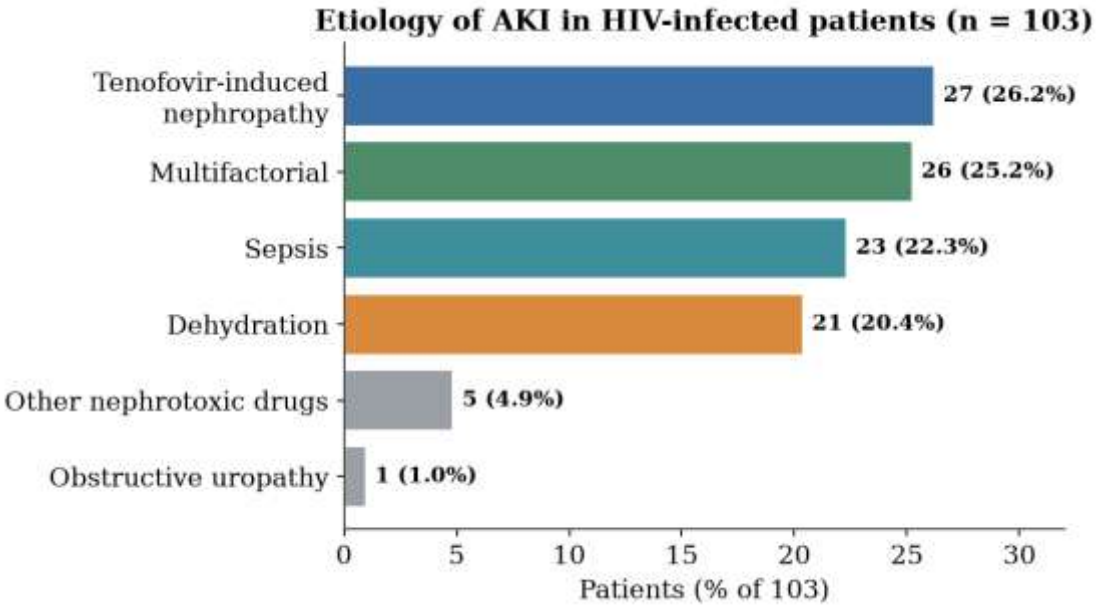

Figure 1. Etiology of acute kidney injury in HIV-infected patients (n = 103). Tenofovir-induced nephropathy was the leading cause, followed by multifactorial causes, sepsis and dehydration.

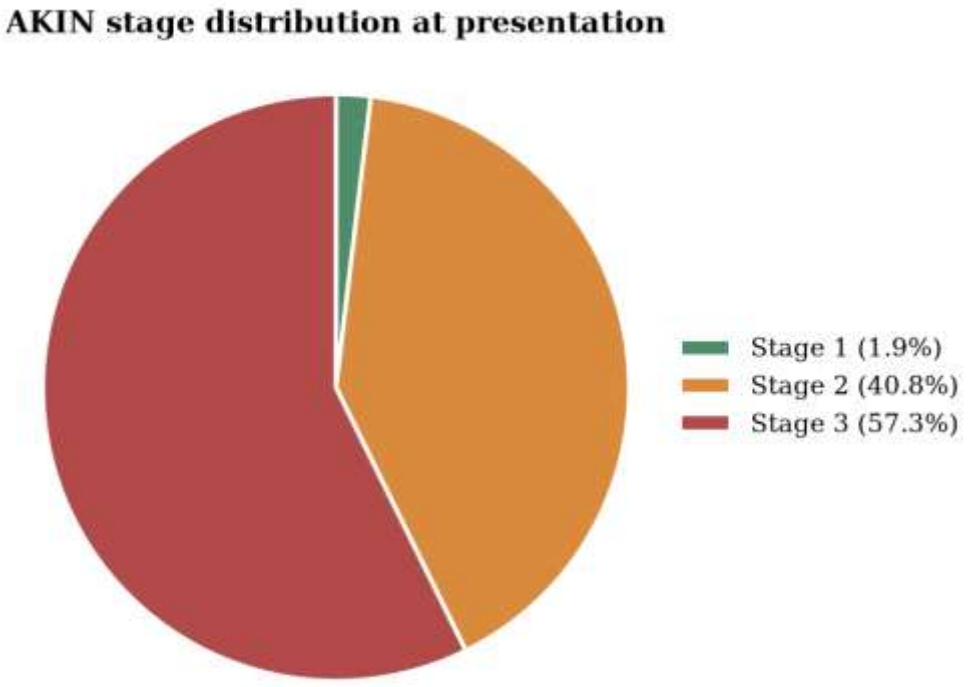

Figure 2. AKIN stage distribution at presentation. The majority of patients presented in AKIN stage 3, reflecting late presentation.

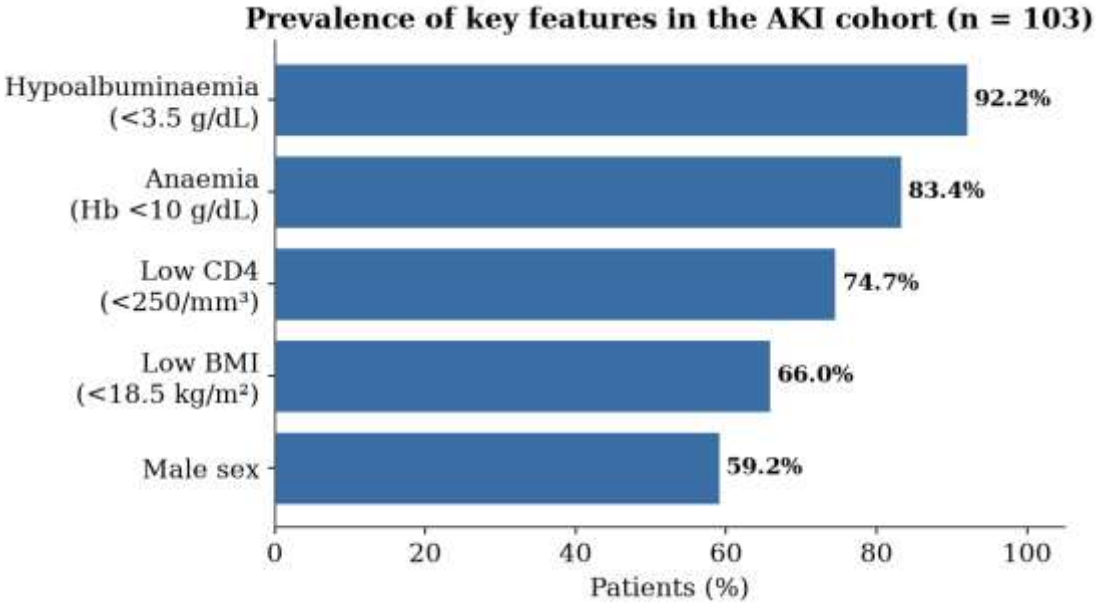

Figure 3. Prevalence of key features in the AKI cohort (n = 103). Hypoalbuminaemia, anaemia and low CD4 count were highly prevalent.

Table 1. Demographic, clinical and laboratory profile of the study population (n = 103)

Variable	Category / statistic	Value
Age (years)	Mean ± SD	46.7 ± 10.5
Age 41–50 years	n (%)	42 (40.8)
Sex	Male / Female	61 (59.2) / 42 (40.8)
Duration since HIV diagnosis	Mean (years)	4.6
On TLE ART regimen	n (%)	89 (86.4)
CD4 count (cells/mm³)	Mean ± SD	213 ± 99
CD4 <250 cells/mm³	n (%)	77 (74.7)
Pulmonary tuberculosis	n (%)	20 (19.4)
Systemic hypertension	n (%)	13 (12.6)
Diabetes mellitus	n (%)	6 (5.8)
Body-mass index (kg/m²)	Mean ± SD	18.3 ± 1.3
Haemoglobin (g/dL)	Mean ± SD	8.6 ± 1.3
Serum albumin (g/dL)	Mean ± SD	2.45 ± 0.62
Hypoalbuminaemia (<3.5 g/dL)	n (%)	95 (92.2)
Total leucocyte count (/mm³)	Mean ± SD	10,626 ± 6,560
Baseline serum creatinine (mg/dL)	Mean ± SD	0.98 ± 0.64
Serum creatinine at presentation (mg/dL)	Mean ± SD	3.63 ± 1.66
AKIN stage	1 / 2 / 3 (%)	1.9 / 40.8 / 57.3

SD, standard deviation; TLE, tenofovir–lamivudine–efavirenz; ART, antiretroviral therapy; AKIN, Acute Kidney Injury Network.

Table 2. Etiology of AKI and sub-classification (n = 103)

Etiology	n	% of 103
Tenofovir-induced nephropathy	27	26.2
Multifactorial causes	26	25.2
Dehydration + sepsis	9	—
Tenofovir + obstructive uropathy	7	—
Tenofovir + antitubercular therapy	6	—
Tenofovir + co-trimoxazole	2	—
Tenofovir + NSAID	2	—

Etiology	n	% of 103
Sepsis	23	22.3
Urinary tract infection	10	—
Bronchopneumonia	9	—
Cellulitis / necrotising fasciitis / appendicitis / cholecystitis	1 each	—
Dehydration	21	20.4
Other nephrotoxic drugs (amphotericin B 4, amikacin 1)	5	4.9
Obstructive uropathy	1	1.0

NSAID, non-steroidal anti-inflammatory drug. Percentages are of the total 103 patients; each patient was assigned one primary etiological category.

Table 3. Comparison of key findings with previous studies

Parameter	Present study	Prakash et al.	Franceschini et al.	Hubert et al.
Incidence of AKI	7.5%	3.9%	5.9%	—
Mean age (years)	46.7	35.3	—	42
Male sex	59.2%	68.8%	—	34.8%
Mean CD4 (cells/mm³)	213	201	—	<250 in 95%
AKIN stage 3	57.3%	48.5%	—	57.2%

Values for comparator studies are as reported in the respective publications; "—" denotes not reported/not comparable.

DISCUSSION

In this prospective study, the incidence of AKI among admitted HIV-infected patients was 7.5%. This is comparable to reports by Franceschini et al. (5.9%) and Roe et al. (5.7%), higher than Prakash et al. (3.9%) and lower than Lopes et al. (18%); differences reflect variation in case definitions, admission thresholds and study populations.[5,10,11,12] The mean age of 46.7 years and male predominance (59.2%) are consistent with the demographics of HIV infection in India and with several comparator cohorts, although sub-Saharan series report a female predominance.[10,13,14] The cohort was characterised by advanced immunosuppression: the mean CD4 count was 213 cells/mm³ and three-quarters had a CD4 count below 250 cells/mm³. A low CD4 count reflects a higher stage of HIV infection, predisposing to opportunistic infections, sepsis and hence AKI, and has been identified as a risk factor for AKI in HIV across several studies.[5,6,15] Most patients (86.4%) were receiving a tenofovir-based (TLE) regimen, in line with national

ART guidelines; tenofovir nephrotoxicity typically develops within 6–9 months of initiation and manifests as proximal tubular dysfunction.[4,9]

Tenofovir-induced nephropathy was the single commonest etiology (26.2%), followed by multifactorial causes (25.2%), sepsis (22.3%) and dehydration (20.4%). Other series have variably reported sepsis, hypovolaemia or multifactorial causes as predominant: Prakash et al. found hypovolaemia (44.2%) leading, Hubert et al. found infections in 65.8%, and Vachiat et al. reported sepsis in 62%.[10,13,14] The prominence of tenofovir in our cohort probably reflects its near-universal first-line use under current guidelines, and highlights the importance of baseline and periodic renal monitoring in patients on tenofovir, together with dose adjustment and avoidance of concomitant nephrotoxins.

Hypoalbuminaemia (<3.5 g/dL) was present in 92.2% and low BMI (<18.5 kg/m²) in 66.0%, reflecting the poor nutritional state of the cohort. Hypoalbuminaemia has a recognised causal association with AKI and has been described as a risk factor in HIV-related AKI.[16,17] Most patients presented late, in AKIN stage 3 (57.3%) or stage 2 (40.8%), comparable to Hubert et al. (stage 3 in 57.2%), underscoring the need for earlier recognition.[14]

Limitations

This was a single-centre, hospital-based study, and referral patterns may have influenced the case mix. Follow-up was not performed, so outcomes such as renal recovery, dialysis requirement and mortality could not be assessed. Importantly, the study did not include an HIV-infected comparison group without AKI; the demographic and laboratory features described (male sex, age 41–50 years, low CD4 count, low BMI, hypoalbuminaemia) therefore represent characteristics of the AKI cohort rather than statistically established independent risk factors. A larger, controlled study with longitudinal follow-up would allow formal risk-factor and outcome analysis.

CONCLUSION

AKI is an important cause of hospital admission among HIV-infected patients, with an incidence of 7.5% in this cohort. Tenofovir-induced nephropathy was the leading etiology, followed by multifactorial causes, sepsis and dehydration. AKI occurred predominantly in men aged 41–50 years and was associated with a low CD4 count, low BMI and hypoalbuminaemia, and most patients presented in advanced AKIN stages. Judicious use of tenofovir with renal monitoring, prompt treatment of sepsis, correction of volume depletion and attention to nutrition may help prevent and mitigate AKI in this population.

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