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ORIGINAL ARTICLE

In-hospital mortality of HIV-positive patients with acute kidney injury a decade after the roll-out of anti-retroviral therapy in Cape Town, South Africa

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ABSTRACT

Background: Acute kidney injury (AKI) in HIV-infected patients in sub-Saharan Africa is a common cause of hospitalisation and is associated with high morbidity and mortality. There is a paucity of comparative data regarding the outcomes of AKI in those patients with and without HIV infection from the African continent.

Methods: This was a single-centre retrospective study of all consecutive adult patients with AKI referred to the renal unit at Tygerberg Hospital for the period January 2015 to December 2016. The diagnosis of AKI required evidence of the following: a recent normal serum creatinine and/or normal kidney sizes on ultrasound examination and/or granular casts on urine microscopy. Kaplan–Meier curves and logistic regression were used to assess survival and identify factors predicting mortality.

Results: We identified a total of 291 patients with AKI of whom 116 (40%) were HIV positive. HIV-positive patients had a mortality rate of 34.5% vs. 29.1% in the HIV-negative patients ($P = 0.34$). At hospital admission, HIV-positive patients had a higher admission serum creatinine (551 µmol/L vs. 190 µmol/L, $P < 0.01$). Of those who died, the HIV-positive patients were younger (41 vs. 52 years, $P < 0.01$), predominantly Black (87.5% vs. 23.5%, $P < 0.01$) and were mostly admitted to medical wards (92.5% vs. 41.2%, $P < 0.01$). There was no difference in mortality related to the use of renal replacement therapy ($P = 0.50$). Logistic regression identified mixed ancestry (OR 2.47, $P = 0.02$), HIV infection (OR 2.69, $P < 0.01$) and surgical ward admission (OR 2.05, $P = 0.03$) as predictors of death.

Conclusions: In-hospital mortality of AKI was high, and HIV infection was associated with a greater risk of death. This may be the result of late presentation of both the AKI as well as the HIV infection.

Keywords: acute kidney injury; HIV; outcomes; mortality; South Africa.

BACKGROUND

In 2015, there were 2.1 million new HIV infections worldwide, and a total of 36.7 million people living with HIV, half in sub-Saharan Africa [1]. Advances in treatment are largely responsible for a 26% decline in HIV-related deaths globally since 2010, from an estimated 1.5 million in 2010 to 1.1 million in 2015.

Despite these advances, South Africa continues to have the highest rate of HIV infection in the world, which has had a huge impact on the incidence of both chronic kidney disease (CKD) and acute kidney injury (AKI) [2]. In South Africa, there has been a 67% rise in deaths related to nephritis/nephrosis from 1999 to 2006 [3].

Causes of AKI in HIV-infected patients include hypovolaemia due to diarrhoeal disease, opportunistic infections and sepsis, nephrotoxicity due to drugs such as tenofovir and thrombotic microangiopathies [4,5].

In South Africa, AKI in HIV-infected patients remains a common cause of admission to emergency units. AKI independently predicts worse outcomes and is associated with additional costs on an already overburdened health-

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care system [6,7]. The reported incidence of AKI in HIV-infected persons ranges from 2.7 to 5.9 per 100 person-years with higher rates occurring in those recently diagnosed with HIV. The associated mortality is as high as 27–33% [8].

Risk factors for AKI include low CD4+ T-cell counts. Counts of < 200 cells/mm^3 was one of the most important predictors of morbidity and mortality related to AKI in HIV-infected patients. Decreasing levels of CD4+ T-cell counts have been associated with an increase in the incidence of AKI [9,10]. Other risk factors include AIDS-defining illnesses, chronic liver disease due to hepatitis B and C, and nephrotoxicity related to highly active antiretroviral therapy (HAART) [4]. Many South African patients present late and usually develop AKI as a complication of diarrhoeal disease or opportunistic infections such as tuberculosis [11]. Drug-induced nephrotoxicity related to agents such as tenofovir, rifampicin and amphotericin B are other common causes of AKI. Less common causes include thrombotic microangiopathies and the use of traditional remedies [12]. A mortality rate of 45.5% was observed among adult South African patients presenting with AKI who report the use of traditional medicines [13,14].

AKI is the most frequent organ failure in the setting of severe sepsis, with large multicentre studies suggesting that 40% of all AKI in critically ill patients could be attributed to severe sepsis [15,16]. A local study reported that 43.6% of AKI in HIV-infected patients was due to sepsis [17]. An earlier study, conducted during the pre-HAART era at Tygerberg Hospital, reviewed AKI outcomes in an intensive care unit (ICU) over one year and identified only 3 of 46 patients who were HIV positive. This likely reflected the local practice at the time not to admit HIV-infected patients to the ICU due to nihilism regarding the overall prognosis [18].

Morbidity and mortality have been demonstrated to be higher in those with HIV who develop AKI [8,19,20]. A study of the long-term consequences of AKI reported high rates of subsequent cardiovascular disease, end-stage renal disease and increased mortality [21]. In the developed world, the incidence of AKI and HIV-associated nephropathy (HIVAN) has decreased substantially as a result of early administration of HAART.

Much work has been done regarding HIVAN and HIV-related CKD; however, there remains a paucity of data regarding AKI and the outcomes of renal replacement therapy (RRT) in the HIV-infected as compared to HIV-uninfected patients. In this paper we report on a cohort of patients with AKI at a tertiary hospital in South Africa and provide mortality data based on HIV serological status.

METHODS

This was a retrospective, cohort study of patients with AKI admitted to Tygerberg Hospital from 1 January 2015 to 31 December 2016. The hospital is a 1380-bed, public sector teaching hospital in Cape Town and provides secondary and tertiary level care to approximately 1.5 million people in the Western Cape province of South Africa. It is one of two tertiary hospitals in the province responsible for providing acute and chronic adult dialysis services. The modality of dialysis most frequently employed is intermittent haemodialysis. Patients who are haemodynamically unstable are offered slow, low-efficiency dialysis (SLED). Haemodialysis is initiated at the discretion of the attending nephrologist and in most cases this is when traditional indications such as refractory hyperkalaemia or pulmonary oedema have arisen.

This study included all adult patients (≥ 18 years) referred to the Division of Nephrology with AKI. The Kidney Disease: Improving Global Outcomes (KDIGO) criteria for AKI staging was not used because only 105 patients (36%) had information on baseline kidney function available. Most of the patients had community-acquired AKI and presented with established AKI at the time of referral. Urine output data were also not available for many of the patients. Therefore, the diagnosis of AKI was determined based on the following information: (1) where available, a normal serum creatinine concentration and an estimated glomerular filtration rate of > 60 mL/min/1.73 m^2 in the preceding 3 months and/or (2) normal kidney sizes by ultrasound examination (>100 mm in length) and/or (3) the documentation of granular casts on urine microscopy. Patients were excluded if they had CKD or evidence of glomerular disease.

Data that were extracted from clinical files included demographics, AKI diagnosis, additional comorbidities, administration of HAART, use of renal replacement therapy versus conservative management, the ward to which the patient was admitted (medical versus surgical), and laboratory data including serum creatinine and CD4+ T-cell counts.

Patients were divided into two groups: HIV positive and HIV negative. These groups were further subdivided into those that did and did not receive RRT. Differences in mortality between these groups were compared as a primary endpoint.

All numerical data with a normal distribution were described using means ± standard deviations. Non-normal data were reported as medians and interquartile ranges (IQR). The chi-squared test was used to analyse mortality in different

groups. Univariable and multivariable logistic regressions were used to identify predictors of death. The t-test was employed to compare the means of continuous data where the data had a normal distribution. Where data did not have a normal distribution, the Mann–Whitney U test was used. Statistical significance was set at a P value of < 0.05.

Permission to perform this study was granted by the Health Research Ethics Committee at Stellenbosch University (approval number S17/01/006) and was conducted in accordance with the Declaration of Helsinki.

RESULTS

A total of 291 cases of AKI were identified. Of these, 116 (40%) were HIV positive. At baseline, the median age was 39 years (IQR 32–46 years), and in the HIV-positive and HIV-negative groups it was 38 years (IQR 32–46 years) and 42 years (IQR 29–56 years), respectively ($P = 0.06$). One hundred and forty-seven patients (51%) were female, with no significant difference between the groups ($P = 0.27$). More patients in the HIV-positive group were Black than in the HIV-negative group (90.5% versus 40.6%, respectively, $P < 0.01$). See Table 1.

Approximately half (155 patients, 53%) of the patients had comorbid diseases. Non-communicable diseases were more common in HIV-negative cases. Hypertension occurred in 7.8% of HIV-positive patients as compared to 18.3% of HIV-negative patients ($P = 0.01$). More HIV-infected patients had a diagnosis of tuberculosis than those who were HIV-negative (19.0% versus 2.9%, respectively, $P < 0.01$).

Regarding baseline laboratory data, the overall median serum creatinine at admission was 240 µmol/L (IQR 114–656 µmol/L). In the HIV-positive group, the median creatinine on admission was much higher at 552 µmol/L (IQR 173–837 µmol/L) than in the HIV-negative group at 190 µmol/L (IQR 104–416 µmol/L, $P < 0.01$). The overall median serum creatinine at the start of renal replacement therapy was 738 µmol/L (IQR 575–1112.5 µmol/L), with the HIV-positive group having a median creatinine of 923 µmol/L (IQR 61–1221 µmol/L) as compared to the HIV-negative group of 722 µmol/L (IQR 559–1044 µmol/L, $P = 0.11$). At hospital discharge, the overall median creatinine concentration was 120 µmol/L (IQR 83–181 µmol/L), with the HIV-positive group having a median value of 137 µmol/L (IQR 93–194 µmol/L) as compared to the HIV-negative group of 108.5 µmol/L (IQR 77–169 µmol/L, $P = 0.06$). The median CD4+ T-cell count was 132 cells/mm^3 in the HIV-positive group. See Table 1.

The most common causes of AKI in the HIV-positive patients included pneumonia (27%) and diarrhoeal disease (22%), whereas pneumonia (14%), rhabdomyolysis (19%) and obstetric (11%) aetiologies predominated in the HIV-negative patients. All the patients with cardiac failure were HIV negative.

The leading category of AKI was ischaemic acute tubular necrosis (ATN) (46%), followed by septic ATN (38%). More HIV-positive patients had septic ATN than the HIV-negative group (53% versus 27%, $P < 0.01$). Other causes of AKI included toxic ATN (13%), acute tubulointerstitial nephritis (3%) and pre-renal azotaemia (0.7%).

A total of 67 (23%) patients received RRT of whom 19 (16%) were HIV positive and 48 (27%) were HIV negative ($P = 0.03$). The most common indication for RRT initiation was hyperkalaemia.

Regarding the primary outcome of in-hospital mortality, the overall number of deaths was 91 (31%) with 40 (35%) from the HIV-positive group and 51 (29%) from the HIV-negative group ($P = 0.34$, see Figures 1A and 1B). The overall median age was 45 years (IQR 35–54 years), while for the HIV-positive patients it was 41 years (IQR 34–47 years) as compared to 52 years (IQR 37–60 years) for the HIV-negative group ($P < 0.01$). See Table 2.

Overall mortality was higher in males (53/91, 58%) and more males in the HIV-positive group died than in the HIV-negative group (73% versus 47%, $P = 0.02$). Black patients represented approximately half of the overall mortality with the overwhelming majority being HIV positive (88% versus 24%, $P < 0.01$). The mortality rate was highest for those patients in the medical wards (58 patients, 64%) with most HIV positive patients (93%) dying in the medical wards as compared to most HIV-negative patients dying in the surgical wards (57%, $P < 0.01$). There was no difference in survival between those who received and those who did not receive RRT. See Figure 1C.

Seventy-four (64%) of the HIV-positive group had a CD4+ T-cell count < 200 cells/mm^3. This group had higher mortality than HIV-positive patients, with a CD4+ T-cell count of > 200 cells/mm^3 ($P = 0.01$) (Table 3). Sixty-seven patients were on HAART of whom 22 (33%) died; 48 were not receiving treatment of whom 18 (38%) died.

Univariate logistic regression analysis identified age, male sex and mixed ancestry as predictors of death; however, multivariate logistic regression analysis revealed mixed ancestry, being HIV positive and admission to surgical wards as predictors of mortality. See Table 4.

Table 1. Baseline characteristics of HIV-positive and HIV-negative patients with AKI.

Baseline characteristics	All n = 291	HIV+ n = 116	HIV– n = 175	P value
Demographic data				
Age, median (IQR)	39 (30–53)	38 (32–46)	42 (29–56)	0.06
Female, n (% of group)	147 (50.5)	54 (46.6)	93 (53.1)	0.27
Ethnicity, n (% of group)				
Black	176 (60.5)	105 (90.5)	71 (40.6)	< 0.01
White	28 (9.6)	2 (1.7)	26 (14.9)	–
Mixed ancestry	87 (29.9)	9 (7.7)	78 (44.5)	–
Comorbid diseases, n (% of group)				
Hypertension	41 (14.1)	9 (7.8)	32 (18.3)	0.01
Diabetes mellitus	31 (10.7)	10 (8.6)	21 (12.0)	0.36
Tuberculosis (pulmonary and extrapulmonary)	27 (9.3)	22 (19.0)	5 (2.9)	< 0.01
Other*	56 (19.2)	10 (8.6)	46 (26.3)	< 0.01
Laboratory data, median (IQR)				
Admission creatinine (µmol/L)	240 (114–656)	551.5 (173.5–837)	190 (101–416)	< 0.01
Creatinine at RRT initiation (µmol/L)	738 (575–1113)	923 (691–1221)	722 (559–1044)	0.11
Creatinine at hospital discharge	120 (83–181)	137 (93–194)	108.5 (77–169)	0.06
CD4+ T-cell count, n=115	–	132 (47–292)	N/A	–
Causes of AKI, n (% of group)				
Pneumonia	55 (18.9)	31 (26.7)	24 (13.7)	< 0.01
Rhabdomyolysis	38 (13.1)	5 (4.3)	33 (18.9)	–
Obstetric	23 (7.9)	3 (2.6)	20 (11.4)	–
Urosepsis	18 (6.2)	11 (9.5)	7 (4.0)	–
Diarrhoea	42 (14.4)	25 (21.6)	17 (9.7)	–
Cardiac failure	10 (3.4)	0 (0)	10 (5.7)	–
Drugs/toxins	19 (6.5)	9 (7.8)	10 (5.7)	–
Pancreatitis	8 (2.7)	1 (0.9)	7 (4.0)	–
GSW	7 (2.4)	0 (0)	7 (4.0)	–
Ruptured AAA	2 (0.7)	0 (0)	2 (1.1)	–
Bowel ischaemia	3 (1.0)	0 (0)	3 (1.7)	–
Bowel perforation	4 (1.4)	0 (0)	4 (2.3)	–
Other#	59 (20.3)	29 (25.0)	30 (17.1)	
Type of AKI, n (% of group)				
Septic ATN	110 (37.8)	62 (53.4)	48 (27.4)	< 0.01
Ischaemic ATN	133 (45.7)	40 (34.5)	93 (53.1)	–
Toxic ATN	37 (12.7)	7 (6.0)	30 (17.1)	–
ATIN	9 (3.1)	7 (6.0)	2 (1.1)	–
Pre-renal AKI	2 (0.7)	0 (0.0)	2 (1.1)	–
Ward, n (% of group)				
Medical	182 (62.5)	99 (85.3)	83 (47.4)	< 0.01
Surgical	109 (37.5)	17 (14.7)	92 (52.6)	–
Indications for RRT, n (% of group)				
Hyperkalaemia	28 (9.6)	5 (4.3)	23 (13.1)	0.01
Metabolic acidosis	2 (0.7)	1 (0.9)	1 (0.6)	1.00
Pulmonary oedema	18 (6.2)	6 (5.2)	12 (6.9)	0.56
Fluid overload	13 (4.5)	5 (4.3)	8 (4.6)	0.92
Encephalopathy	7 (2.4)	5 (4.3)	2 (1.1)	0.12
Pericarditis	1 (0.3)	1 (0.9)	0 (0.0)	0.40
Offered RRT, n (% of group)				
Yes	67 (23.0)	19 (16.4)	48 (27.4)	0.03
No	224 (77.0)	97 (83.6)	127 (72.6)	–

Abbreviations: HIV, human immunodeficiency virus; RRT, renal replacement therapy; AKI, acute kidney injury; ATN, acute tubular necrosis; ATIN, acute tubulointerstitial nephritis; GSW, gunshot wound; AAA, abdominal aortic aneurysm. *Other comorbidities: carcinoma, liver failure, alcohol dependence, chronic obstructive pulmonary disease, asthma, chronic hepatitis B, gout, systemic lupus erythematosus, rheumatoid arthritis, interstitial lung disease, cerebrovascular accident, mitral valve replacement, rheumatic heart disease, aortic dissection, deep vein thrombosis, abdominal aortic aneurysm, meningioma, peptic ulcer disease, major depressive disorder, atrial fibrillation, pulmonary embolism, epilepsy, gastroesophageal reflux disease, mycobacterium other than tuberculosis, schwannoma, panhypopituitarism, peripheral vascular disease, hypothyroidism. #Other causes of AKI: necrotizing fasciitis, meningitis, peptic ulcer perforation, cholecystitis, contrast nephropathy, compartment syndrome, diabetic ketoacidosis, burn wounds, acute liver failure and sepsis of unknown origin.

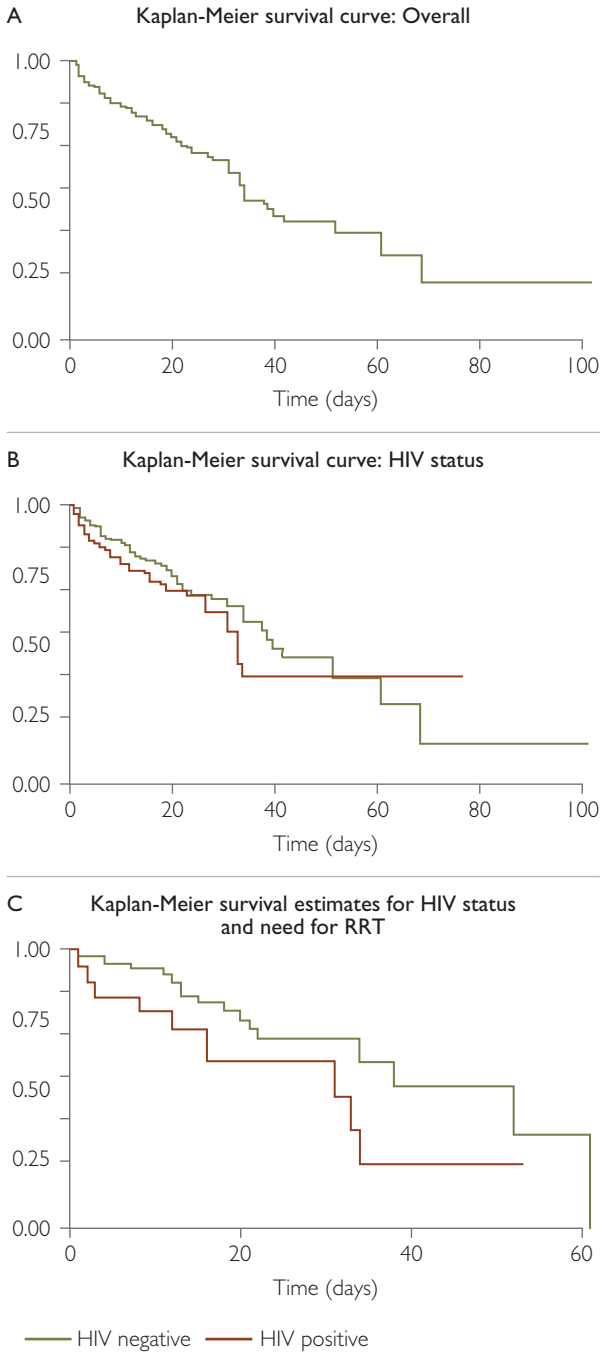

Figure 1. Survival analysis for all patients with AKI and by HIV status and need for renal replacement therapy. A, Overall survival. B, Comparison of survival between HIV-positive and HIV-negative patients (log-rank, P = 0.22). C, Comparison of survival between HIV-positive and HIV-negative patients receiving RRT (log-rank, P = 0.46).

DISCUSSION

We found a high overall in-hospital mortality of 31%, whereas others have reported lower mortality rates. Nash et al. focused on hospital-acquired AKI and found an overall mortality rate of 19.4% [22]. Liangos et al. reported a mortality rate of 21.3% in hospitalized patients with AKI, which was nearly 10 times greater than patients without AKI [23]; Wang et al. documented an in-hospital mortality in incident AKI patients of only 10% [24]. These studies had relatively lower mortality rates despite patients being older, having more non-communicable diseases and being mostly HIV uninfected. For patients who developed hospital-acquired AKI, the earlier identification and initiation of treatment may have resulted in improved outcomes. Most of the patients in our study had community-acquired AKI and late presentation may have contributed to poorer outcomes.

We found no difference in crude mortality between HIV-positive and HIV-negative patients. Few studies have reported direct comparisons. A 2012 systematic review concluded that there were insufficient data for this purpose [25]. More recently, some studies from South Africa have reported on these outcomes. One of these studies compared the outcomes of AKI in critically ill adult patients treated with continuous RRT and reported very high 90-day mortality rates in the HIV-positive patients (60% vs. 10%) [26]. This was thought to be related to late presentation. The lower mortality in our study is probably due to the inclusion of patients who had AKI but recovered kidney function without the need for dialysis, the inclusion of less critically ill patients and our focusing on only in-hospital mortality. A Johannesburg study reported slightly higher in-hospital mortality rates than in our study [27]. This study included patients with chronic kidney disease, which may have affected mortality. We identified HIV as an independent predictor of death. The reason for this is uncertain but may be related to late presentation of both the AKI as well as the HIV infection.

We found no difference in mortality related to RRT. A study of HIV-positive patients with AKI requiring dialysis noted that patients who died had lower CD4+ counts, fewer dialysis days as well as shorter length of hospital stay relative to those who recovered kidney function or remained dialysis-dependent [17]. Our findings support the conclusions of others [17,27] that HIV-infected patients with AKI should be considered for acute dialysis support because their outcomes are similar to those of HIV-negative patients with AKI.

Most of the HIV-positive patients who died were relatively young, Black, male, predominantly admitted to medical wards and had septic AKI. These findings were nearly identical to those documented by other South African studies [17,26,27]. The median CD4+ count at baseline was 132 cells/mm^3, indicating late HIV diagnosis. A low CD4+ count is known to be a strong risk factor for the development of AKI as well as death [9,17]. Our study also found a higher mortality when CD4+ counts were less than 200 cells/mm^3.

Table 2. Mortality in HIV-positive and HIV-negative patients with AKI.

	All	HIV+	HIV–	P value
Overall mortality, n (% of group)	91 (31)	40 (35)	51 (29)	0.34
Variables in patients who died				
Age, median (IQR)	45 (35–54)	41 (34–47)	52 (37–60)	< 0.01
Sex, n (% of group)				
Male	53 (58.2)	29 (72.5)	24 (47.1)	0.02
Ethnicity, n (% of group)				
Black	47 (51.6)	35 (87.5)	12 (23.5)	< 0.01
White	12 (13.2)	2 (5.0)	10 (19.6)	–
Mixed	30 (33.0)	2 (5.0)	28 (54.9)	–
Unknown	2 (2.2)	1 (2.5)	1 (2.0)	–
Ward admission, n (% of group)				
Medical	58 (63.7)	37 (92.5)	21 (41.2)	< 0.01
Surgical	32 (35.2)	3 (7.5)	29 (56.9)	–
OBGYN	1 (1.1)	0 (0)	1 (2.0)	–
Medical ICU only	17 (18.7)	7 (17.5)	10 (19.6)	0.06
Surgical ICU only	24 (26.4)	3 (7.5)	21 (41.2)	–
Cause of ATN, n (% of group)				
Septic	49 (53.8)	25 (62.5)	24 (47.1)	0.04
Ischaemic	33 (36.3)	13 (32.5)	20 (39.2)	–
Toxic	6 (6.6)	0 (0)	6 (11.8)	–
Received RRT, n (% of group)				
No	65 (71.4)	30 (75.0)	35 (68.6)	0.46
Yes	26 (28.6)	10 (25.0)	16 (31.4)	–

Abbreviations: HIV, human immunodeficiency virus; IQR, interquartile range; ICU, intensive care unit; OBGYN, obstetrics and gynaecology; ATN, acute tubular necrosis; RRT, renal replacement therapy.

Mixed ancestry, HIV-positive status and surgical ward admission were independent predictors of death. Mixed ancestry as a predictor was probably a reflection of this ethnic group constituting nearly half of the Western Cape population [28]. Since most of the HIV-positive patients who died had CD4+ counts less than 200 cells/mm^3, it is likely that the late diagnosis of infection was a major factor contributing to this risk. Most of the surgical patients who died were HIV negative; however, these cases were older, had more comorbidities, and were predominantly admitted to the surgical ICU. AKI in this sub-group of critically ill patients was associated with poor outcomes.

We found that ATN was the most common cause of AKI with septic ATN as the dominant cause in the case of HIV-infected patients whereas ischaemic ATN predominated in the HIV-negative patients. This finding was comparable to other studies [17,27,29]. Prakash et al. reported that 53% of their HIV patients had pre-renal azotaemia at presentation [29], whereas none of our HIV patients had this condition. Many of our patients' first contact with medical

Table 3. Mortality in HIV-positive patients based on CD4+ cell count and use of HAART.

	All	HIV+	P value
CD4+ count (cells/mm^3)	n = 116 (%)		
< 200	74 (63.8)	32 (27.6)	0.01
≥ 200	42 (36.2)	8 (6.9)	
HAART	n = 115 (%)		
Yes	67 (58)	22 (33)	0.65
No	48 (42)	18 (38)	

Abbreviations: HIV, human immunodeficiency virus; HAART, highly active anti-retroviral therapy.

Table 4. Predictors of mortality using univariable and multivariable logistic regression.

Univariable logistic regression	Odds ratio	P value	95% CI
Age	1.02	< 0.01	1.01–1.04
Male sex	1.70	0.04	1.02–2.80
Ethnicity (reference Black)			
White	2.05	0.08	0.91–4.67
Mixed ancestry	1.76	0.047	1.00–3.06
HIV-positive	1.27	0.34	0.77–2.11
Surgical ward	1.60	0.10	0.92–2.79
Multivariable logistic regression			
Age	1.01	0.20	0.99–1.03
Male sex	1.22	0.47	0.71–2.11
Ethnicity (reference Black)			
White	2.28	0.10	0.84–6.16
Mixed ancestry	2.47	0.02	1.18–5.15
HIV-positive	2.69	< 0.01	1.28–5.61
Surgical ward	2.05	0.03	1.09–3.96

Abbreviation: HIV, human immunodeficiency virus.

services is at their peripheral hospitals where treatment, such as a fluid challenge, is initiated. Only if the response to therapy is judged as poor, or the referring physician anticipates that RRT may soon be needed, are patients referred to our facility for possible dialysis. Therefore, many patients with pre-renal azotaemia do not reach our facility because they recover kidney function at their peripheral hospitals and are not referred.

The serum creatinine concentration at the time of presentation was much higher in the HIV-positive patients. Difficulty in accessing healthcare facilities, delays in diagnosis and transfer to tertiary centres are some of the issues that contribute to late presentation. The median serum creatinine concentration at initiation of RRT was not different between the HIV-positive and HIV-negative groups. Arendse et al. reported a pre-dialysis serum creatinine value of 998 µmol/L, which was similar to our patients at the time dialysis was started [17]. In general, due to resource constraints, the initiation of acute dialysis in the South African public sector is usually delayed until more traditional indications have evolved. This practice may vary within centres, from one nephrologist to the next, as well as between the public and private healthcare sectors.

LIMITATIONS

This was a single-centre, retrospective study. We could not define AKI using more contemporary staging criteria such as the KDIGO or Acute Kidney Injury Network (AKIN) criteria because most of the patients included did not have baseline kidney function available and urine output was not well monitored. Only referrals to our service were assessed. Patients who were not referred may either have died or had mild injury that did not require referral. This may have influenced mortality outcomes. Also, we were unable to score the severity of the illness at presentation and therefore were unable to comment on the effect that this may have had on outcomes. Finally, since this study was conducted in a public sector hospital, mortality outcomes may differ from those of the private sector due to differences in case mix as well as variations in practice.

CONCLUSIONS

In patients with AKI, there was no difference in crude mortality rates based on HIV status or the need for RRT. However, HIV infection was associated with increased mortality. This may be related to late presentation of both the HIV diagnosis as well as the AKI.

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