The world is a dangerous place; not because of people who are evil, but because of the people who don’t do anything about it.

Albert Einstein

In 1984, Pardo et al described what is now widely known as the “collapsing glomerulopathy” of human immunodeficiency virus (HIV)-associated nephropathy (HIVAN). This initial description has been substantiated by the works of Rao et al and D’Agati et al. HIVAN, with a predilection for black Africans, is now known to consist of a constellation of findings involving glomerular, interstitial, and tubular changes. Genetic factors appear to involve the apolipoprotein L1 (APOL1) gene, which is in strong linkage disequilibrium with nonmuscle myosin heavy chain 9 (MYH9). The role of these genes in sub-Saharan Africa (SSA) has yet to be determined.

The interference of HIV-1 viral proteins with the fully differentiated podocyte causes it to proliferate. There also appears to be disruption of contact between the glomerular basement membrane and the podocyte, allowing the podocyte to “fall off” into Bowman space, with loss of glomerular integrity and ultimately collapse of the glomerulus. This gives rise to the well described collapsing focal segmental glomerulosclerosis (FSGS) seen in HIVAN.

Renal epithelial cell infection by HIV-1 was demonstrated in 2000. However, renal epithelial cells lack CCR5 and CXCR4 receptors, the mechanism by which the virus classically enters to infect T cells, macrophages, and dendritic cells. Evidence suggests that T cells have virologic synapses that allow transfer of the virus from T cells to epithelial cells.

Thus, a significant complication of HIV infection is kidney disease. Because up to 30% of HIV-positive patients may have abnormal kidney function, HIV-associated kidney disease has emerged as a
relatively common cause of end-stage renal disease and death.9,10

In Africa, the first report of a biopsy-proven case of HIVAN, featuring a collapsing focal segmental glomerulopathy with tubular microcysts, was published in the *South African Medical Journal* in 1994.11 By that time, the epidemic of HIV in Africa had commenced many years before. In the ensuing years, there have been very few studies from Africa addressing HIVAN and other HIV-related kidney diseases, and most of what we know today about HIVAN comes from non-African countries.

**HISTORY OF THE EPIDEMIC**

HIV-1 is the dominant type of HIV found in SSA. Its viral ancestor appears to have been a simian immunodeficiency virus found in several chimpanzee colonies in Cameroon, Western Africa.12 The first transfer of simian immunodeficiency virus to humans is believed to have occurred around 1930.13 It is likely that HIV-2 originated in the 1940s after transfer from monkeys in Guinea-Bissau (Fig 1).14,15 The prevalence of this HIV type remains low in SSA.

By the 1970s, the Congolese capital of Kinshasa was experiencing the first epidemic of HIV-1. It is thought that the virus was carried to the Congo by an infected individual from Cameroon and the virus entered an urban sexual network. The epidemic spread from west to east, and by the 1980s, the Ugandan population in particular had a high level of infection. At this time, physicians were aware of acquired immunodeficiency syndrome (AIDS) in the United States; however, the connection was not yet made with the epidemic occurring in Africa.

Interactions of itinerant groups, such as truck drivers and soldiers, with sex workers hastened the spread along transport and trade routes. As the decade progressed, the epidemic moved south and SSA became the epicenter of the HIV epidemic by the early 1990s. SSA now accounts for more than two-thirds of all people living with HIV and was the site of almost three-quarters of the AIDS-associated deaths that occurred in 2008.16 In 2009, an estimated 1.8 million people in SSA became infected with HIV, and the overall population of those living with HIV in this region reached 22.5 million. The country with the largest number of people living with HIV (5.6 million) is South Africa.

Blacks are affected more commonly than any other race group. In almost all SSA countries, most of those living with HIV are women, particularly girls and young women aged 15-24 years. In South Africa, there is an ~21% prevalence in women aged 20-24 years versus ~7% in men of the same age group.17

There has been an absence of surveillance, documentation, and confirmation of renal histologic types in HIV-positive patients in SSA. In addition, little is known about the prevalence and effects of treatment on outcome, and most areas of Africa lack access to analysis by biopsy. The prevalence of HIVAN in the United States has been calculated to be 3.5%-12%;18 its prevalence in SSA has been reported as 6%-45%.19 However, these prevalence rates have been derived mostly from studies making assumptions of HIVAN without actual histologic confirmation of the disease.

Studies from South Africa have tended to more accurately report the prevalence of kidney disease in HIV-positive patients using histologic data.20-24 Data from our

![Figure 1. The origins of the human immunodeficiency virus (HIV) epidemic in Africa. Reproduced from www.avert.org15 with permission of AVERT (Averting HIV and AIDS).](image-url)
center in Cape Town has shown that the diagnosis of HIVAN was made in 25% of all renal biopsies performed in 2009. In the past in South Africa, a deliberate decision was made not to perform biopsy on patients with kidney disease in HIV-positive patients. This decision was made due to the poor prognosis of HIVAN without the availability of combination antiretroviral therapy (cART). Since the arrival of cART, this attitude has changed.

The largest reliable kidney biopsy series in HIV-positive patients has recently been published by us. We describe the spectrum of renal pathology seen in 192 biopsies from HIV-positive patients. The patients were predominantly black Africans and there were no whites in the study. We viewed HIVAN as a disease not only of the glomerulus, but accepted that, in addition, HIVAN encompassed interstitial and tubular changes. However, glomerular changes predominated in HIVAN. Collapsing FSGS was found to be the most common variant of HIVAN, and pseudocrescents also featured in this series (Fig 2). There were other histologic subtypes, including the perihilar variant of FSGS. In the case of any variant of FSGS, additional histologic features needed to be present (these included parietal or podocyte hypertrophy or hyperplasia, microcysts, and plasma cell interstitial infiltrate) to be able to attribute the pathology to HIV infection. We described a new variant of FSGS we termed the “fetal” variant, which resembles the histologic conformation of a fetal glomerulus (Fig 3). The interstitial component of HIVAN has a characteristic lymphocytic infiltrate that is composed predominantly of plasma cells. The diagnostic clincher for HIV involvement in the interstitium is the presence of microcysts (Fig 4).

Immune complex glomerulonephritis may be seen in conjunction with HIVAN. The most common pattern is mesangiocapillary glomerulonephritis. A second variant is characterized by large subepithelial deposits surrounded by base-

**Figure 2.** (A) Collapsing focal segmental glomerulosclerosis with protein reabsorption granules within podocytes (center) (hematoxylin and eosin stain; original magnification, ×400) (B) Prominent epithelial cells surround a collapsed glomerulus. This lesion can be confused with a crescent (pseudocrescent) (hematoxylin and eosin stain; original magnification, ×400).

**Figure 3.** The glomerulus shows global collapse and prominence of the epithelial cells. This resembles the histologic configuration of a fetal glomerulus (hematoxylin and eosin stain; original magnification, ×400).
ment membrane extensions.\textsuperscript{21,23} This lesion can be seen on hematoxylin and eosin–stained sections (Fig 5). These deposits may represent either a form of postinfectious glomerulonephritis or a variant of membranous glomerulopathy. Immune complex glomerulonephritis seen without features of HIVAN also appears in this series.\textsuperscript{26} The most common coincidental immune complex glomerulonephritis was membranous glomerulonephritis (Box 1).

Data from Groote Schuur Hospital, Cape Town, South Africa, showed 50% mortality at 4.47 months for patients with HIVAN.

**Figure 4.** Typical microcysts. Massively distended tubules with atrophied and flattened epithelial cells. They contain acellular eosinophilic material (hematoxylin and eosin stain; original magnification, ×400).

**Figure 5.** (A) Large subepithelial deposits surrounded by basement membrane extensions can be seen at original magnification ×400 on light microscopy. These deposits usually are larger than the typical subepithelial deposits seen in membranous glomerulopathy, but are more numerous than in postinfectious glomerulonephritis (hematoxylin and eosin stain). (B) Immunohistochemistry staining is positive for immunoglobulin G (and C3; not shown); original magnification, ×400. (C, D) These lesions are best resolved and identified at the ultrastructural level (electron microscopy; original magnification, ×20,000 [D]; ×15,000 [E]).
not receiving cART. The outcome for all forms of the disease was improved greatly with the initiation of cART (Fig 6).

Although HIVAN and the components thereof have come to represent the common manifestations of kidney disease in HIV, the occurrence of acute kidney injury (such as acute tubular necrosis) is common in HIV-positive patients. Studies have reported the incidence of acute kidney injury to be as high as $\frac{1}{10}$ for all HIV-infected patients presenting with decreased kidney function. The cause of acute tubular necrosis in such instances has been shown to vary, but often is attributed to sepsis, malaria, diarrheal illnesses, rhabdomyolysis, or nephrotoxins (aminoglycosides, co-trimoxazole, antituberculosis drugs, and tenofovir; Box 2).

### SOCIOECONOMICS

The social and economic impact of the HIV epidemic in Africa, in particular HIV/AIDS-related poverty and the increasing number of orphans (giving rise to families with children parenting children), perhaps has reached its devastating climax on the continent. The present period of abatement has been brought about by the eventual rollout of cART to an ever increasing number of HIV-positive patients.

President Thabo Mbeki of South Africa (immediate past president) and other leaders in Africa had denied that HIV was the only cause of the wasting and destructive disease (initially called “slimmer’s disease”) that was seen in the community. Instead, these denialists blamed poverty and consequent malnourishment as the driving force behind the disease process. Dr “Manto” Tshabalala-Msimang served as Minister of Health under Mbeki, and her emphasis on treating South Africa’s AIDS epidemic with garlic, beetroot, lemons, and potatoes rather than with antiretroviral medicines set back the public sector rollout of cART by years. This almost certainly caused many thousands to die and perpetuated the ongoing epidemic (new infections and mother-to-baby transmission). She was ridiculed in the local press, and an example of this is shown in a cartoon (Fig 7), which originally appeared in the national newspaper, the Sunday Times, on February 15, 2004. Following pressure from activists, notably the Treatment Action Campaign and health care workers, South Africa

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### Box 1. The UCT Classification of HIVAN

**Glomerulus**
- Glomerular variants of HIVAN
  - FSGS collapsing variant
  - FSGS noncollapsing variant with additional features
  - Global sclerosis with epithelial cell involvement: “fetal” variant
- Additional features to glomerular variants of HIVAN
  - Parietal and or visceral epithelial cell hypertrophy and hyperplasia
  - Presence of pseudocrescents

**Interstitial**
- Fibrosis
- Lymphocytic infiltrate
- Plasma cells within the lymphocytic infiltrate
- Diffuse inflammatory lymphocytic syndrome

**Tubules**
- Presence of microcysts
- Epithelial cell hyperplasia and hypertrophy

**HIV + ICGN**
- With additional features of HIVAN (as described)
  - Mesangiocapillary glomerulonephritis
  - Ball in cup: very large subepithelial immune deposits
  - Any other type of glomerulonephritis
- Without additional features of HIVAN (immune complex disease alone)

**Others**
- Diseases unrelated to HIV, eg, granulomas, acute tubular necrosis, drug reactions, lymphoma

Abbreviations: FSGS, focal segmental glomerulosclerosis; HIVAN, human immunodeficiency virus–associated nephropathy; ICGN, immune complex glomerulonephritis; UCT, University of Cape Town.
Box 2. Causes of Kidney Failure in HIV-Positive Patients in Cape Town

Acute
- Sepsis
- Rhabdomyolysis
- Drug toxicity
  - Rifampicin
  - Co-trimoxazole
  - Aminoglycosides
- GN
- Pyelonephritis
- Renal tuberculosis

Acute-on-chronic
- HIVAN (± immune complex disease) with
  - ATN
  - Nephrotic drug
  - Granulomatous disease
  - Malignant hypertension
  - Lymphoma

Chronic
- HIVAN
- HIVAN + other GN
  - Membranous GN
  - Mesangiocapillary GN
  - IgA nephropathy
  - Mesangiproliferative GN
  - Idiopathic FSGS
  - Hypertensive nephrosclerosis
  - Diabetic nephropathy

Abbreviations: ATN, acute tubular necrosis; FSGS, focal segmental glomerulosclerosis; GN, glomerulonephritis; HIVAN, human immunodeficiency virus–associated nephropathy; IgA, immunoglobulin A.
Source: Arendse et al.24

started providing cART drugs only in 2004.

Aside from the denialsists, several governments in SSA (except Uganda, Zaire, Zambia, and Senegal) often refuted that there was an epidemic of HIV/AIDS in Africa and showed a cynical skepticism, in which they were willing to accept international aid but were unwilling to combat the virus.29 Also, a study done in Burkina Faso, West Africa, provided interesting data.30 The results showed that the “treat poverty, treat HIV” theory was flawed. The authors demonstrated that fighting poverty did not necessarily decrease the prevalence of HIV/AIDS. They found that the predicted probability of having HIV was highest in the rich, and the growth of HIV in countries in conflict had been known to be slower than in surrounding countries at peace.31

In studies in South Africa, black women have been found to have the highest incidence of HIV infection. This disproportionate increase of females over males is a direct result of the sociocultural differences and economic constraints.32 These factors may have compelled women to initiate sexual partnerships for favors and financial gain (eg, “sugar daddy” relationships).33 Black females have little bargaining power in this society, which leaves them with limited control over their own sexual behavior.34

The low gross domestic product of most African countries, as well as the burden of communicable diseases, has elevated health care costs above what many African countries are able to afford. The price for urgent or long-term dialysis is far too high for most African countries and dialysis is offered to only those who are able to afford to pay privately (eg, in Nigeria). Alternatively, when governments offer dialysis to their populace, as in South Africa, the finances for dialysis are limited. This restriction in funding has necessitated the development of exclusion criteria by hospital authorities/physicians. These exclusion criteria are artificial and merely serve to act as a gatekeeper to prevent patients from being accepted for dialysis when funding is unavailable. A good example is the development of categories for acceptance that has been in use in Groote Schuur Hospital (Box 3). Category 1 patients will always be treated, category 2 patients will be placed on a waiting list, and category 3 patients will not be accepted into the dialysis program. In reality, category 2 patients seldom “make it” onto the program. As shown in Box 3, HIV-positive status relegates the patient to a category 2 or 3 level.

These exclusion criteria were challenged by a patient with diabetic nephropathy who had been refused acceptance into a dialysis program in Durban, South Africa.35 The case was heard in the Constitutional Court of South Africa (Case CCT 32/97) in 1997. The judge noted that everyone was entitled to the health care services supported by the state “within its available resources” and ruled in favor of the state. In the judge’s summary, he said, “Unfortunately, the resources are limited, and I can find no reason to interfere with the allocation undertaken by those better equipped than I to deal with the agonising choices that had to be made.”35(p34)

DISPARITY IN HEALTH CARE WORKFORCE AND EXPENDITURE

Compared with other parts of the world, the health personnel to population ratios of Africa have fallen behind figures of other regions with higher gross domestic products (Fig 8).36 For example, in the 1990s, 10 countries in SSA had one physician per 30,000 population, compared with 1:2,000 or 1:3,000 for comparable countries like Bolivia, Honduras, and India.37 This therefore means that the number of nephrologists in many SSA countries will be equally low compared with developed countries. Even now within SSA, there is wide variation in the number of
nephrologists, with figures generally being fewer than 1 per million population in many countries (Table 1).38,39 This translates into inadequate screening and late referral of patients with kidney disease.

SSA has 11% of the world population but contributes 24% to the global disease burden. However, SSA has only 3% of the global health workforce and allocates <1% of global financial resources to health care.40 With such a small budget made available for health care already beleaguered by the ravaging epidemic of HIV/AIDS, there is little wonder that such funds end up for use in “curable” conditions such as malaria, tuberculosis, and diarrheal illnesses rather than for the provision of cART. Little or nothing is made available for chronic kidney diseases.

LACK OF INFRASTRUCTURE

The basic facilities to practice good clinical nephrology, such as laboratory, radiologic services, and renal biopsies, often are nonexistent in many SSA countries. In many studies, there have been few (or no) renal biopsies performed to establish causality with HIV. Emem et al41 from Nigeria reported a high prevalence of 152 of 400 patients (38%) with kidney disease in patients with HIV/AIDS. Ten of these patients underwent a kidney biopsy, most of which showed features of classic HIVAN; only 3 had normal kidney histology.41 Although the number of biopsies was low, they were able to use results of their study to draw attention to the fact that there is still a gap in our knowledge of HIVAN in SSA. In a large study (373 patients) from Western Kenya, kidney disease was defined by abnormal creatinine level and dipstick proteinuria.42 Renal biopsies were not performed. Biopsies would have given valuable information about the underlying pathologic processes in this region.

If Africa is to make a valuable contribution to the understanding of HIVAN, the performance of renal biopsies, a cornerstone of clinical nephrology, will be critical toward achieving this goal. We also have been able, through retrospective analysis of biopsies performed at Groote Schuur Hospital, to show that secondary glomerular diseases (lupus nephritis and HIVAN) have become the dominant causes of kidney disease in our population. HIVAN is the primary cause of nephrotic syndrome in black South Africans.22,25

Many African researchers do not have access to funds or other necessary research support, including mentorship from their institutions. Few research laboratories in SSA have the skills and equipment to support research work in HIVAN, and local laboratories may not be able to cope with the complexities of prospective clinical trials. Many medical school libraries in SSA do not have access to relevant nephrology literature, which means that there may be perpetuation in the gap of our knowledge of HIVAN from SSA.

The International Society of Nephrology Global Outreach program specifically sponsors young African doctors to study in recognized dialysis centers in Africa. This is with the specific purpose of them returning to their own countries to start the appropriate management of chronic kidney disease.

EFFECTS OF WARS AND UNRESOLVED CONFLICTS ON AIDS

Wars and protracted conflicts create ideal conditions for poverty, famine, destruction of the health care infrastructure, large population movements, and the breakdown of family units.43 Beginning in the mid-1970s, 7 countries in SSA (Sierra Leone, Liberia, Democratic Republic of Congo, Angola, Mozambique, Somalia, and South Sudan) have endured civil disorders and wars for 10 years or more. Others (Zimbabwe, Nigeria, and Ivory Coast) are threatened by imminent political, religious, or ethnic divisions that often erupt into
violent conflicts and social unrest. Violence and the threat of violence may make it difficult for an individual to protect against HIV infection and make healthy choices in terms of sexual behavior. In many countries, sex workers, those who use drugs, and sexual minorities are vulnerable to violence, rape, harassment, and unjustified arrest at the hands of police forces that abuse their authority.

POLITICAL WILL

An equally important factor that constrained access to treatment relates to the difficulties that African pharmaceutical companies encountered before they could obtain licenses to produce cheap generic antiretroviral drugs. In December 1999, President Bill Clinton announced that when countries are facing a public health crisis, the United States would treat the enforcement of drug patent laws flexibly. With an escalating epidemic and the high cost of providing antiretroviral therapy, the death toll continued to rise, as well as the number of new infections. Many SSA countries began mass rollout of cART in only the early to mid-2000s. Some of the effects of the rollout on worldwide HIV statistics already are being seen. No SSA country currently is experiencing an increased incidence of HIV. Most have had a >25% decrease in the incidence rate of HIV infection from 2001 to 2009.

WHAT OF THE FUTURE?

Since the initial descriptions of HIVAN, renal histologic findings and the accompanying immune complex diseases have been described inconsistently. Further histologic descriptions and combinations of histologic findings have been identified by us (Figs 3 and 5) and others in South Africa.

Some authors have grouped features of HIVAN with immune complexes as being “HIVAN,” whereas others have distinguished between the 2. The distinction between the 2, we suggest, is very important because immune complex disease on its own may respond differently to cART. Clearly defining HIVAN, HIVAN plus immune complex diseases, immune complex diseases without HIVAN, and diseases unrelated to HIV would enable standardization of outcomes of treatment regimens. It also would assist in prognosticating outcomes based on histology (Box 1).
In South Africa, we are now able to offer renal replacement therapy to selected HIV-positive patients. Because it is early days and the numbers are small, there have been no reported evaluations of outcomes of these patients on long-term dialysis therapy.

In addition to the conventional HIV-negative–donor transplant, our unit has formally established a transplantation program for HIV-positive donors to HIV-positive recipients. To date, we have performed transplants on 14 HIV-positive patients using HIV-positive donors. Our experience in transplant has shown that even with heavy immunosuppression, HIV-positive patients have an excellent outcome.49

Barriers to receiving cART, such as the threshold for commencing treatment based on CD4 count, have been changed. In 2009 on World AIDS Day, the South African government announced that patients would be eligible to receive cART when their CD4 count decreased to \( \leq 350 \text{ cells}/\mu L \); the cut-off previously had been established at 200 cells/\( \mu L \).50

**WHAT STILL NEEDS TO BE DONE**

The prevalence of chronic kidney disease and the patterns of kidney disease in HIV-positive patients in SSA still need to be de-

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**Table 1.** Distribution of Nephrologists and RRT in Selected African Countries and Comparators

<table>
<thead>
<tr>
<th>Country</th>
<th>Nephrologists</th>
<th>Dialysis Modality</th>
<th>HD</th>
<th>CAPD</th>
<th>CAPD Fraction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. pmp</td>
<td>No. pmp</td>
<td>No. pmp</td>
<td>No. pmp</td>
<td>No. pmp</td>
</tr>
<tr>
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<td>33,000 421</td>
<td>45 0.3</td>
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<td>Morocco</td>
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<td>4,800 162</td>
<td>30 1</td>
<td>0.62</td>
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<tr>
<td>Tunisia</td>
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<td>6,500 650</td>
<td>200 20</td>
<td>3.0</td>
<td></td>
</tr>
<tr>
<td>Nigeria(^b)</td>
<td>100 0.7</td>
<td>1,000 8</td>
<td>0 —</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Ghana(^b)</td>
<td>2 0.1</td>
<td>35 2</td>
<td>0 —</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Senegal(^b)</td>
<td>2 0.2</td>
<td>50 4.2</td>
<td>26 1</td>
<td>34.2</td>
<td></td>
</tr>
<tr>
<td>Sudan</td>
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<td>1,610 46</td>
<td>111 3</td>
<td>6.5</td>
<td></td>
</tr>
<tr>
<td>Kenya(^h)</td>
<td>15 0.5</td>
<td>260 7.5</td>
<td>30 3.7</td>
<td>10.3</td>
<td></td>
</tr>
<tr>
<td>Rwanda(^h)</td>
<td>1 —</td>
<td>0 —</td>
<td>30 3.7</td>
<td>100.0</td>
<td></td>
</tr>
<tr>
<td>South Africa(^h)(^c)</td>
<td>80 1.8</td>
<td>3,360 75</td>
<td>1,449 32.2</td>
<td>28.7</td>
<td></td>
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<td>United Kingdom</td>
<td>525(^d) 8.4(^d)</td>
<td>— 670.1</td>
<td>— 119.7</td>
<td>15.1</td>
<td></td>
</tr>
<tr>
<td>United States(^a)</td>
<td>— —</td>
<td>370,269 1,684.2</td>
<td>27,559 125</td>
<td>6.9</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CAPD, continuous ambulatory peritoneal dialysis; HD, hemodialysis; pmp, per million population; RRT, renal replacement therapy.

\(^a\)Represents the prevalence of peritoneal dialysis expressed as a percentage of the total dialysis population.

\(^b\)Located in sub-Saharan Africa.

\(^c\)These numbers indicate more recent (2009) data for South Africa.

\(^d\)Data courtesy of Dr P. Mason (personal communication, January 2012).

\(^e\)Data from the US Renal Data System (2011).39

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fined accurately. Because renal replacement therapy is not widely available, it is essential that we establish better treatment regimens for HIVAN. We currently are conducting a randomized controlled trial using, in addition to cART, steroids for the treatment of HIVAN.

CONCLUSIONS

A complex interplay of factors, including viral infection, genetic predisposition, and immune activation, contribute to the pathogenesis of HIV-associated kidney diseases. There is a dearth of local epidemiologic data for HIV-associated kidney disease. Strategies to prevent or slow progression to end-stage renal disease should include standardization of laboratory data collection, specifically, performing a urinalysis and measuring kidney function, for all HIV-positive individuals at initial presentation. Initiating cART at a higher CD4 count is progress, which will further reduce the incidence of HIV, including kidney disease. The outcome of HIVAN can be dramatically altered by using cART (Fig 6). The goal is to prevent renal replacement therapy, and strategies include fast tracking patients for cART, awareness of tenofovir\(^\text{11}\) and trimethoprim and sulfamethoxazole (Bactrim) toxicity, avoidance of nephrotoxic agents such as nonsteroidal anti-inflammatory drugs, adequate fluid resuscitation, and efficient treatment of sepsis.

Patients are living longer on cART and hence are more at risk of developing hypertension and diabetes. We may find this to be the new epidemic in Africa. The International Society of Nephrology Outreach Program has gone a long way in attempting to correct the imbalance in knowledge and education.

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