Kidney Injury in Children Infected with HIV, Followed at the Teaching Hospital of Borgou (Benin): Epidemiological and Clinical Aspects

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Abstract

The history of kidney disease associated with HIV infection dates back to the years of HIV breakthrough. The objective was to study kidney damage in children infected with HIV at the Teaching Hospital of Borgou (Benin) in 2019. This was a cross-sectional, descriptive, analytical, matching-type study carried out from June 1, 2019 to September 30, 2019 at the pediatrics department of Teaching Hospital of Borgou (Benin). The study included HIV-positive children, followed in consultations, and whose parents gave their consent. The biological markers were demonstrated with urine dipstick. Glomerular filtration rate was calculated using the Schwartz test and classified according to stages. The dependent variable was the presence of at least one impairment (biological or functional). Sample size was determined by Schwartz's method on the basis of one case for two controls. Sociodemographic, clinical, biological, and therapeutic data were collected. Comparisons were made using the Chi-square test or Fisher’s exact test. The identification of associated factors was possible using a multiple logistic regression model at 5% threshold.

In total, we included 117 children, including 39 HIV-positive children. The average age was 8 ± 4.81 years and the gender ratio was 1:17. The frequency of kidney damage was 76.5%. Permanent proteinuria and at least two crosses on urine dipstick were present in 20.5%, leukocyturia in 2.6%, and proximal tubular dysfunction in 5.1%. Glomerular hyperfiltration was found in 38.5%, acute kidney injury in 38.5%, and chronic kidney injury in 5.1%. Associated factors were age (P = 0.004), presence of opportunistic infections (P = 0.00), and treatment adherence (P = 0.004). Kidney damage is common in HIV-positive children. Careful follow-up is necessary to avoid complications.

Keywords: Benin; HIV-positive; kidney disease; pediatrics

Introduction

At the end of 2017, 36.9 million people were living with HIV worldwide (1). Every day, some 1500 children aged less than 15 years are infected with HIV. An estimated 90% of them live in Saharan Africa. One in seven persons who die from HIV-related illnesses is a child aged less than 15 years. Globally, HIV is now responsible for 3% of deaths in children aged 5 years and 6% in Sub-Saharan Africa (2). In Benin, an estimated 70,000 people were living with HIV, including 6700 children. The history of kidney disease associated with HIV
infection dates back to the years of HIV discovery. In 1984, in New York and Miami, clinicians reported the first cases in black adults (3,4). Later in 1989, HIV-related nephropathy was confirmed in children in Miami (5–7). In sub-Saharan Africa, little is known about the extent of kidney damage in HIV infection, especially in children. However, this region could represent the largest HIV infection globally (8). In Benin, a 2013 study carried out at the National Teaching Hospital of Cotonou reported a 9.8% frequency of kidney injury in people living with HIV (PLHIV) receiving antiretroviral treatment (9). In Parakou, a study carried out at the teaching hospital of Cotonou reported a 44.19% incidence of kidney injury in adult patients on antiretroviral treatment (10). However, in Benin no study has been conducted on this subject in children infected with HIV. Hence, this study was initiated in 2019 with the objective of studying the frequency and factors associated with kidney damage in children infected with HIV at the pediatrics department of the Teaching Hospital of Borgou (Benin).

Patients and Study Method

This was a prospective and analytically descriptive study, pairing type with a 5-month collection from June 3, 2019 to October 5, 2019 in the pediatrics department of Teaching Hospital of Borgou (Benin). Inclusion criteria was all children aged 2 to 15 years, infected with HIV naive, followed at the pediatrics ward and whose parents had given their consent. Children followed in the ward for various non-renal conditions and who were not infected with HIV were considered as controls. Matching was made according to gender, ethnicity, parental education, and nutritional status. It was considered a case for two witnesses. The sampling method for cases is an exhaustive census of patients meeting the inclusion criteria despite a minimum size of 33 children, calculated by the Schwartz formula for a frequency of HIV infection at the pediatrics department of Teaching Hospital of Borgou reported a 4.41% incidence of kidney injury in adult patients on antiretroviral treatment (10). However, in Benin no study has been conducted on this subject in children infected with HIV. Hence, this study was initiated in 2019 with the objective of studying the frequency and factors associated with kidney damage in children infected with HIV at the pediatrics department of the Teaching Hospital of Borgou (Benin).

The dependent variable was the presence of renal impairment, which could be functional, biological, morphological, and/or anatomoclinical.

Renal failure, glomerular hyperfiltration, and tubular dysfunction which appeared during the follow-up were considered as functional renal impairment. Renal failure is defined by a glomerular filtration rate (GFR) of less than 100 mL/min, calculated according to the Schwartz formula, simplified and readjusted in 2009:

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GFR \ (\text{mL/min} / 1.73 \text{m}^2) = \frac{0.413 \times \text{height (in cm)}}{\text{serum creatinine (mg/dL)}} (12).
\]

Glomerular hyperfiltration was defined by GFR > 120 mL/min. Biological markers included presence of proteinuria, hemoglobinuria or glucosuria on the urine dipstick. Proximal tubular dysfunction retained in front of proteinuria was greater than or equal to a cross and normoglycemic glucosuria on the urine dipstick. Morphological involvement was confirmed with change in the size and structure of the kidneys on ultrasound. The morphology was assessed using the Konu et al. scale (13). Renal hypertrophy was considered for a kidney size greater than normal according to both height of the children and Konu et al. scale (13).

Moderate malnutrition is defined by a weight/height ratio and/or a height/age ratio of between –3 z-scores to –2 z-scores to the median (14).

Severe acute malnutrition is defined by a weight/height ratio that is –3 z-scores lower than the median or by severe and visible emaciation or by the presence of nutritional edema (14). Severe acute malnutrition is complicated if there is severe wasting (14).

Sociodemographic, clinical, paraclinical, therapeutic, and evolutionary variables were studied. The data were collected through an individual interview with individual and medical files.

Double data entry was made in the French version of the Epi Data 7.2.3.1 software after each monitoring period. These data were then analyzed using SPSS 24 and Stata 13 software.

The averages are presented with their standard deviation values and the proportions/frequencies of quantitative variables. The measure of association used was the odds ratio (OR). Statistically, Chi-square test, Fischer exact test, or Yates-correction Chi-square test were used as appropriate to determine the degree of significance of association (P-value). A multivariate analysis by logistic regression by carrying out successive descending step-by-step iterations was carried out to identify the factors associated with the occurrence of kidney damage. The significance level was set at 0.05. The present work was a research study of the Faculty of Medicine of the University of Parakou, and it obtained the approval of the ethics and professional conduct committee. Following verbal consent of parents, the rules of human dignity, anonymity, and confidentiality were respected.

Results

For this case-control study, 39 children living with HIV were included as HIV-positive patients and 78 children included as controls (HIV-negative), that is, one case for two controls.

Sociodemographic characteristics of HIV-positive subjects

The average age was 8 years (±4.81 years) in HIV-positive subjects. The most represented age group was that of 5–10
Among infected children, 33.3% had maternal and 20.5% paternal deaths.

Clinical characteristics of HIV-positive subjects

The majority of patients were admitting through HIV-positive consultations (74.4%). Most of them had no specific medical history (97.4%). More than three of the four subjects (76.9%) were in good nutritional status. However, 12.8% presented with Moderate Acute Malnutrition (MAM), 5.1% with Uncomplicated Severe Acute Malnutrition (UcSAM), and 5.1% with Severe Acute Malnutrition with Complication (SAMC).

In relation to the general condition, 66.7%, 30.8%, and 2.5% of children were, respectively, found in stage I, II, and IV. Of the 39 subjects, a significant and severe immunological deficit was found in 10.26% and 12.82%, respectively (Table 2). Of the 39 HIV-positive subjects, 25 (83.3%) were on antiretroviral therapy.

Renal Disorders

Renal markers

On the urine dipstick, of the 39 HIV-positive patients, proteinuria, leukocyturia, hemoglobinuria, and glucosuria were found in 20.5%, 2.6%, 0%, and 5.1%, respectively. Proximal tubular dysfunction was found in 5.1% of patients.

Morphological markers of HIV-positive subjects

Of the 39 HIV-positive children, 20.5% had enlarged kidneys and 25.64% had a hyperechoic cortex despite the corticomedullary differentiation preserved in 100%.

Functional renal impairment

Of the 39 HIV-positive subjects, 33.3% and 5.1% presented with acute renal failure and chronic renal failure, respectively. In relation to glomerular filtration rate, glomerular hyperfiltration, mild renal failure, and preterminal renal failure were found in 38.5%, 33.3%, and 5.1%, respectively (Table 2).

Identification of HIV-related kidney damage

Renal impairment statistically related to HIV in infected subjects were proteinuria (P = 0.00), mild renal failure (P = 0.01), and glomerular hyperfiltration (P = 0.00) (Table 3).

Multivariate analysis

In addition, there was an association between renal hypertrophy and glomerular hyperfiltration (P = 0.03) (Table 4).

### Table 1: Clinical and therapeutical characteristics of HIV-positive subjects (n = 39).

| Reason for admission                             | Number | Percentage |
|--------------------------------------------------|--------|------------|
| Routine consultation                             | 29     | 74.4       |
| Consultation for symptoms                        | 05     | 12.8       |
| Other reasons                                    | 05     | 12.8       |
| Asthma                                           | 01     | 2.6        |
| Moderate acute malnutrition                      | 05     | 12.8       |
| Uncomplicated severe acute malnutrition          | 02     | 5.1        |
| Severe acute malnutrition with complication      | 02     | 5.1        |
| Good nutritional status                          | 30     | 76.9       |
| Moderate acute malnutrition                      | 05     | 12.8       |
| Uncomplicated severe acute malnutrition          | 02     | 5.1        |
| Severe acute malnutrition with complication      | 02     | 5.1        |
| Stage I                                          | 26     | 66.7       |
| Stage II                                         | 12     | 30.8       |
| Stage IV                                         | 01     | 2.5        |
| Normal                                           | 32     | 82.0       |
| Opportunistic infections                         | 02     | 5.1        |
| Other ailments                                   | 04     | 12.9       |
| Not significant                                  | 27     | 67.23      |
| Way                                              | 03     | 7.69       |
| Important                                        | 04     | 10.26      |
| Severe                                           | 05     | 12.82      |
| On antiretroviral therapy                        | 25     | 64.10      |

years, that is, 38.5%. Male subjects were 21 (53.8%) with a gender ratio of 1:17 (Table 1). Of the 39 infected children surveyed, 38 (97.4%) were from Beninese and 61.7% resided within 5 km of the Borgou Regional Teaching Hospital (CHUD/B).
Table 2: Renal impairment in HIV-positive subjects included in the study.

| Renal markers          | Number (N) | Percentage (%) |
|------------------------|------------|----------------|
| Proteinuria            |            |                |
| Negative               | 31         | 79.5           |
| Positive               | 8          | 20.5           |
| Leucocyturia            |            |                |
| Negative               | 38         | 97.4           |
| Positive               | 01         | 2.6            |
| Hemoglobinuria          |            |                |
| Negative               | 39         | 100            |
| Glycosuria              |            |                |
| Negative               | 37         | 94.9           |
| Positive               | 02         | 5.1            |
| Proximal tubular dysfunction | 02     | 5.1            |

| Morphological disorders |          |                |
|-------------------------|----------|----------------|
| Kidney size             |          |                |
| Normal                  | 31       | 79.5           |
| Increased               | 08       | 20.5           |
| Echostucture            |          |                |
| Echogenic iso cortex     | 29       | 73.9           |
| Hyperechoic cortex       | 10       | 26.1           |
| Cortico-medullary differentiation preserved | 39 | 100 |
| Functional renal impairment |        |                |
| Acute kidney injury      | 13       | 33.3           |
| Chronic renal failure    | 02       | 5.1            |
| Glomerular filtration rate |       |                |
| Glomerular hyperfiltration | 15     | 38.5           |
| Normal kidney function   | 09       | 23.1           |
| Mild renal failure       | 13       | 33.3           |
| Preterminal renal failure | 02     | 2.5            |

Discussion

Our study is the first one carried out on the subject at the pediatrics department of Teaching Hospital of Borgou (Benin). This was a case-contro study matched by gender, ethnicity, parents’ level of education, and nutritional status, which made it possible to recruit all infected and uninfected children who came to consult in this service and fulfilled the inclusion criteria. In addition to serum creatinine, we included proteinuria, hematuria, and leukocyturia as biological markers, already used in other studies (15,16). The validity of these parameters is questionable for chronic kidney disease but easy to use. To avoid possible errors, we considered subjects with two crosses and more for proteinuria and one cross and more for hematuria. Small sample size was a limitation of our study. It would have been particularly interesting to measure certain additional parameters, such as serum calcium, for the evaluation of renal damage, and other markers of proximal tubular dysfunction, namely, phosphoremia, phosphaturia, aminoaciduria, etc. The renal biopsy was not performed to better assess the pathological damage. Incidentally, kidney biopsy is not performed in our country. It is a capital exploration for diagnosis and therapeutic evaluation.

Sociodemographic characteristics

Most of the children included in our study were males, with an overall gender ratio of 1:21, that is, 1:17 in HIV-positive subjects and 1:23 in uninfected subjects (controls), thus 53.2% males in HIV-positive and 55.1% in HIV-negative groups. Soumana et al. (17) had also found a male predominance with a gender ratio of 1:6, close to ours, in children infected with HIV. In contrast, Mfutu Ekulu et al. (8) reported a predominance of females with 49.5% of males in infected children and 46.2% in uninfected children. The average age was 8 years (±4.81 years) in HIV-positive subjects. On the other hand, in Kinshasa, Congo, the median ages were 76 months or 6 years in infected children and 96 months or 8 years in uninfected children. Given the significant difference in parental status between infected and uninfected children with 33.3% maternal deaths in infected children and 0% (P = 0.00) in uninfected children, it was deduced that mother-to-child transmission was the major mode of transmission in these children, which agreed with Soumana et al. (17).

Kidney damage

Renal damage in HIV infection is manifold in terms of its onset, its course, and the involved renal structures. In the black race, HIV-associated nephropathy (HIVAN) is the most common condition, marked by a gradual progression to end-stage renal disease. It begins with glomerular...
Table 3: Renal impairment associated with HIV in the subjects included in the study.

|                              | Total | HIV+ | HIV- | OR      | [IC 95%] | P   |
|------------------------------|-------|------|------|---------|----------|-----|
|                              | N (117)| (39) | (78) |         |          |     |
| **Dipstick**                 |       |      |      |         |          |     |
| Proteinuria                  |       |      |      |         |          |     |
| Negative                     | 109   | 31   | 79.5 | 78      | 100      |     |
| Positive                     | 8     | 8    | 20.5 | 0       | 0        |     |
| **Leucocyturia**             |       |      |      |         |          | 0.8 |
| Negative                     | 115   | 38   | 97.4 | 77      | 98.7     |     |
| Positive                     | 2     | 1    | 2.6  | 1       | 1.3      | 2.0 |
| **Hemoglobinuria**           |       |      |      |         |          | 1   |
| Negative                     | 117   | 39   | 100  | 78      | 100      |     |
| Positive                     | 0     | 0    | 0    | 0       | 0        |     |
| **Functional renal impairment** |      |      |      |         |          |     |
| Acute kidney injury          | 59    | 13   | 33.3 | 46      | 59.0     | 0.7 |
| Chronic kidney disease       | 8     | 2    | 5.1  | 6       | 7.7      | 0.81|
| **Glomerular filtration rate** |      |      |      |         |          |     |
| End-stage renal disease      | 1     | 0    | 0    | 1       | 1.3      | 0.72|
| Preterminal renal failure    | 7     | 2    | 5.1  | 5       | 6.4      | 0.98|
| Mild renal failure           | 59    | 13   | 33.3 | 46      | 59.0     | 0.7 |
| Normal kidney function       | 31    | 9    | 23.1 | 22      | 28.2     | -   |
| Glomerular hyperfiltration   | 19    | 15   | 38.5 | 4       | 5.1      | 9.2 |

**Table 4:** Association between renal hypertrophy and glomerular hyperfiltration in HIV+ subjects included in the study.

|                                   | Hyperfiltration | No hyperfiltration | OR      | IC       | P    |
|------------------------------------|----------------|--------------------|---------|----------|------|
|                                   | n = 11 | %     | n = 12 | %      |       |     |
| Renal hypertrophy                  |        |       |        |        |      | 0.03|
| Yes                                | 4      | 36.4  | 1      | 8.3     | 0.14 | 0.003–1.11 |
| No                                 | 7      | 63.6  | 11     | 91.7    |      |     |

hyperfiltration, which most often goes unnoticed (18). It is a compensatory mechanism of the body in the face of nephronic reduction, which is accompanied by the hypertrophy of the remaining healthy nephrons. It is secondary to vasodilation of afferent arterioles and vasoconstriction of efferent arterioles (19). This glomerular hyperfiltration was found in our study in subjects infected with HIV (38.5%), while in uninfected subjects, it was 5.1% (P = 0.00). Even though the literature is poor with regard to glomerular hyperfiltration in children infected with HIV, its mechanism remains the same as that found in glomerular nephropathy and even in diabetic nephropathy and sickle cell patients (20). It was, nevertheless,
confirmed by renal ultrasound that renal hypertrophy was significantly linked to glomerular hyperfiltration, 26.1% (P = 0.03) in children infected, with HIV with a hyperchoic cortex in 21.7% of them as described by Geoffray et al. (21).

It remains important to emphasize this phenomenon in order to avoid the occurrence of the main manifestation of nephropathy linked to HIV. This produces a nephrotic syndrome which was found in 2.9% in the entire group of children infected with HIV by Mfutu Ekulu et al. (8) with similar results in the study conducted by Chaparro et al. (22). While none of the infected children in our study presented with nephrotic syndrome, this discrepancy could then be explained by the small size of our sample. The direct role of virus and its proteins and that of certain genes on glomerular, tubular, and parietal epithelial cells is cited in the pathogenesis of lesions that lead to proteinuria (23). Proteinuria remains the most reliable diagnostic factor in the definitive diagnosis of HIV-related kidney damage based on histopathological examination (24). Particular attention must therefore be paid to its evaluation in the management of children infected with HIV. In this regard, the protocol for the management of renal impairment during HIV infection, developed by the American Society for Infectious Diseases (ISDA) (25), recommends regular screening for signs of kidney damage, including proteinuria, upon diagnosis of HIV infection. This strategy, aimed at early diagnosis and management, may help to slow the progression of end stage kidney disease. This is strongly recommended in Saharan Africa countries with limited resources. The prevalence of proteinuria in our study was 20.5% in infected children and 0% in uninfected children; this is the same (23.8%) in infected subjects compared with 4.3% in uninfected children found in Kinshasa, Congo (8). This shows the importance of instituting a low-salt and hypoproteimuric diet in these children. The frequency of renal failure in children infected with HIV was (38.5%) close to that found in Parakou, Benin among PLWHIV using tenofovir (44.2%) (10). In children not infected with HIV, we found that 59% had acute renal failure because of various ailments presented during hospitalization; however, this rate was very high compared to Tondi et al. (26), who found 5.8% among children hospitalized in Niger. This discrepancy is explained by the size of our sample, which was 200 times smaller than theirs. However, it emerges that the renal assessment must be requested in all children hospitalized for other than kidney disease. Several authors (8,10,17) have found severe immunosuppression at clinical stage as a determining factor, which was not the case in our study, and the reason remains unknown to us.

Conclusion

Hyperfiltration and markers of renal damage such as proteinuria and leukocyturia were the hallmarks of kidney damage in HIV-infected children monitored at CHUD Borgou/Alibori in Parakou. These results could be used after validation for the implementation of renal protection and prevention measures in children infected with HIV in order to preserve their renal capital.

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