Arthritis in association with human immunodeficiency virus infection in Black African children: causal or coincidental?

K. Chinniah, G. M. Mody1, R. Bhimma and M. Adhikari

Objectives. To compare human immunodeficiency virus (HIV)-infected and HIV-uninfected children with arthritis of unknown origin to determine whether the association between HIV infection and arthritis is causal or coincidental.

Method. Retrospective review of 132 children with arthritis who were tested for HIV infection.

Results. Thirty-five (27%) of the children were HIV infected and the male to female ratio was 2.5:1 (P = 0.02). Arthritis was the presenting feature of HIV infection in 78% of these children. The remaining 97 (73%) were diagnosed as having juvenile idiopathic arthritis. ‘Spondyloarthropathy-like’ features were found in 34% of HIV-infected children compared with 5% of uninfected children.

Conclusion. The high prevalence of HIV infection in 27% of children, the predominance of males and the increased prevalence of ‘spondyloarthropathy-like’ features, supports a causal relationship between HIV infection and arthritis.

Key words: Arthritis, Juvenile idiopathic arthritis (JIA), Spondyloarthropathy (SPA), HIV infection, Children.

Reports on the association of arthritis with human immunodeficiency virus (HIV) infection in children have been limited to case reports and small case series [1–6]. In adults, there are many studies implicating HIV infection in the pathogenesis of arthritis [7–11]. The spectrum of articular syndromes reported in adults includes arthralgia, spondyloarthropathy (SPA), reactive arthritis (ReA), psoriatic arthritis, undifferentiated SPA, HIV-associated arthritis, septic arthritis and avascular necrosis [12]. The experience in adults in Africa, where SPA was uncommon prior to the HIV epidemic, has provided support for a true association between arthritis and HIV infection in adults [13–17]. Studies from Argentina and Mexico have provided clinical evidence to support a direct role for HIV infection as a cause of the articular syndromes [18, 19].

Articular manifestations have been reported to occur at any stage of HIV infection but tend to be more prevalent in the late stages. The pathogenesis of arthritis in patients with HIV infection is not completely understood but is probably multifactorial and may involve direct viral invasion of joint tissue, indirect involvement via an activated immune system, genetic and environmental factors.

HIV disease has reached pandemic proportions with an estimated 40 million people worldwide living with AIDS of whom about 26.6 million are estimated to be residing in sub-Saharan Africa [20]. An antenatal survey in 2002, in KwaZulu-Natal, South Africa, documented an HIV prevalence rate of 37% in pregnant women [21]. The vertical transmission rate in Africa is estimated to be between 30 and 35% resulting in the majority of paediatric HIV/AIDS being due to vertical transmission [22]. It is estimated that in the absence of antiretroviral therapy only 50% of vertically infected children will survive to 2 yr and 40% to 5 yr with subsequent low mortality for an undetermined period [23]. The only data on HIV prevalence in South African children come from a recent Human Science Research Council Household Survey in 2002, of all population groups, which reported a 5.6% prevalence of HIV infection in children between the ages of 2 and 14 yr [24].

A review of published literature revealed only a few reports of children with HIV infection and arthritis. These reports include a study by Martinez-Rojano et al. [1] who examined 26 HIV-infected children for rheumatological manifestations and found two children with joint pathology, one with septic arthritis and the other with arthralgia; Smith et al. [2] detected HIV infection in three of 61 children with septic arthritis of the shoulder; Pappo et al. [3] reported HIV positivity in three of four children with septic arthritis in a survey of 139 children with haemophilia; Schlesinger et al. [4] noted a child with concomitant HIV infection and Lyme arthritis, and recently Ahuja et al. [5] reported a 5-yr-old boy with HIV infection and arthritis of the large joints.

From 1997 an increasing number of children referred to our clinic with arthritis of unknown origin were found to be HIV infected. Despite extensive investigations no other cause for the arthritis, except HIV infection, could be identified. This created a dilemma as to whether these children had JIA and coincidental HIV infection or whether HIV infection was causally related to the arthritis. Thus we undertook this study at our Paediatric Rheumatology Clinic to determine the prevalence of HIV infection in Black African children with arthritis of unknown origin, and to compare the pattern of arthritis in children who were HIV infected with those who were HIV uninfected with JIA.

Patients and methods

We reviewed the records of Black African children with arthritis of unknown origin in whom the HIV test was performed. These children were seen at the Paediatric Rheumatology Clinic, King Edward VIII Hospital, Durban, South Africa from January 1998 to December 2002. Children of parents who refused HIV testing...
were excluded from this review. All children underwent extensive investigation to identify a cause for the arthritis. Children in whom the cause of the arthritis could be identified were excluded. A small number of children from other ethnic groups seen at our clinic were also excluded to eliminate the effect of ethnicity and genetic factors on the expression of the disease. At the time of the study King Edward VIII Hospital served as a tertiary referral centre for all children with rheumatic diseases for the province of KwaZulu-Natal, South Africa. This province has a population of 9.4 million people, about 79% of whom are Black Africans [25].

An enzyme linked immunosorbent assay [HIV-1/2 ELISA, Abbott (USA), Axsym] for HIV-1 and -2 antibodies was performed in children in whom informed consent was obtained following appropriate pre-test counselling. A duplicate unlinked test for antibodies to HIV-1 and -2 using the HIV ELISA Abbott (USA) kit was performed on children with positive or indeterminate results. A diagnosis of HIV infection was made when two ELISA tests were positive in children over the age of 15 months and appropriate post-test counselling was undertaken. HIV/AIDS was classified according to the Centers for Disease Control (CDC) clinical criteria [26], and when available, the CD4 counts were measured to determine the CDC immunological stage of HIV disease. T-cell subsets were analysed using flow cytometry. HLA B27 was tested by polymerase chain reaction (PCR) using sequence-specific primers on DNA obtained from peripheral blood. Joint-related disability was graded according to the Steinbrocker functional class [27].

The children were divided into two categories based on whether they were HIV infected or HIV uninfected. The demographic data, clinical findings, laboratory investigations and management were analysed in these two groups of children. Laboratory results recorded (other than HIV testing) included a full blood count, erythrocyte sedimentation rate, rheumatoid factor, antinuclear factor, complement, blood culture, rubella, hepatitis B and parvovirus serology, tests for streptococcal infection, rheumatic fever, chest X-ray, tuberculin test and other relevant investigations based on the clinical findings (e.g. joint fluid aspiration, bone marrow aspirate, synovial biopsy, rickettsial serology and clotting profiles). None of the children received antiretroviral therapy as it was not available in public sector hospitals in South Africa during the period of review. Children with HIV-associated arthritis received non-steroidal anti-inflammatory drugs (ibuprofen, indometacin, diclofenac, naproxen) as first-line therapy. Chloroquine was the first disease-modifying agent added followed by sulphasalazine or penicillamine. Corticosteroids were given either orally or as intravenous pulse therapy to children with resistant disease. Methotrexate was not prescribed due to its immuno-suppressive effects. Children with JIA received current recommended treatment except tumour necrosis factor antagonists.

JIA was classified according to the 1997 International League Against Rheumatism (ILAR) subgroups of JIA, namely systemic onset juvenile idiopathic arthritis (SOJIA), polyarthritis, oligoarthritis and enthesitis-related arthritis (ERA) [28]. HIV arthritis was classified as oligoarthritis (fewer than five joints involved), polyarthritis (five or more joints involved) or ‘spondyloarthropathy-like’ disorder.

The term ‘SPA like’ is used to emphasize the diversity of disorders that comprise this group and the fact that some children only partially fulfilled the criteria set out by the European Spondyloarthropathy Study Group (ESSG) for SPA [29]. ‘SPA-like’ disorder comprises children with arthritis and enthesitis, children with arthritis and extra-articular features suggestive of reactive arthritis (ReA), like urethritis, acute diarrhoea and conjunctivitis, and children with extra-articular infections associated with ReA (Salmonella, Yersinia). Ethics approval for the study was obtained from the Biomedical Research Ethics Committee of the Nelson R. Mandela School of Medicine, University of KwaZulu-Natal.

### Table 1. Comparison of the demographic data and extra-articular manifestations in HIV-infected and HIV-uninfected children

<table>
<thead>
<tr>
<th></th>
<th>HIV infected, n=35 (%)</th>
<th>HIV uninfected, n=97 (%)</th>
<th>P value</th>
<th>Odds ratio</th>
<th>Confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male:female</td>
<td>25:10 (2.5:1)</td>
<td>46:51 (0.9:1.0)</td>
<td>0.02</td>
<td>2.15</td>
<td>1.12–4.11</td>
</tr>
<tr>
<td>Fever</td>
<td>6 (17)</td>
<td>22 (23)</td>
<td>0.47</td>
<td>0.71</td>
<td>0.23–2.08</td>
</tr>
<tr>
<td>Wasting</td>
<td>14 (40)</td>
<td>10 (10)</td>
<td>&lt;0.001*</td>
<td>5.8</td>
<td>2.06–16.57</td>
</tr>
<tr>
<td>Lymphadenopathy</td>
<td>32 (91)</td>
<td>67 (69)</td>
<td>0.01*</td>
<td>4.78</td>
<td>1.32–26.02</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>15 (42)</td>
<td>12 (12)</td>
<td>&lt;0.001*</td>
<td>10.63</td>
<td>3.51–33.06</td>
</tr>
<tr>
<td>Skin rashes</td>
<td>10 (29)</td>
<td>9 (9)</td>
<td>0.005*</td>
<td>3.91</td>
<td>1.29–11.97</td>
</tr>
<tr>
<td>Eye involvement</td>
<td>5 (14)</td>
<td>4 (4)</td>
<td>0.06</td>
<td>3.88</td>
<td>0.77–20.60</td>
</tr>
<tr>
<td>Gastroenteritis</td>
<td>2 (6)</td>
<td>1 (1)</td>
<td>0.17</td>
<td>5.82</td>
<td>0.29–346.72</td>
</tr>
<tr>
<td>Bronchopneumonia</td>
<td>7 (20)</td>
<td>7 (7)</td>
<td>0.04</td>
<td>3.21</td>
<td>0.91–11.37</td>
</tr>
<tr>
<td>Pulmonary tuberculosis</td>
<td>9 (25)</td>
<td>1 (1)</td>
<td>&lt;0.001*</td>
<td>33.23</td>
<td>4.15–1470.92</td>
</tr>
</tbody>
</table>

*P < 0.05.
children was 5.5 yr (range 2–11 yr) and 7.8 yr (range 8 months–12 yr) in HIV-uninfected children, the difference was not statistically significant.

Wasting, lymphadenopathy, skin rash and hepatomegaly were significantly more prevalent in HIV-infected children (Table 1). The spectrum of skin rashes in HIV-infected children included chicken pox, herpes zoster lesions, eczema and papular urticaria. The predominant skin rash seen in the HIV-uninfected children was the evanescent, erythematous, maculopapular rash associated with systemic onset juvenile idiopathic arthritis.

There was no significant difference in the prevalence of fever or eye involvement in HIV-infected children compared with HIV-uninfected children. The ocular manifestations in the five HIV-infected children included anterior uveitis in three, bilateral conjunctivitis in one and chorioretinitis in the remaining patient. All four (4%) of the HIV-uninfected children had anterior uveitis. There was a significant increase in pulmonary tuberculosis in HIV-infected children. The mean haemoglobin and erythrocyte sedimentation rate were similar in both groups.

**Comparison of the pattern of arthritis in HIV-infected and HIV-uninfected children (Table 2)**

The 97 HIV-uninfected children fulfilled the ILAR criteria for JIA. All these children had had active arthritis for more than 6 weeks and no other cause for the arthritis could be identified despite extensive investigations. These 97 children were classified according to the ILAR criteria for JIA, namely SOJIA (16%), oligoarthritis (39%), polyarthritis (39%) and ERA (5%). None of the children had psoriatic arthritis. Rheumatoid factor (RF) was present in 9% of children with polyarthritis. Children with RF-positive polyarthritis had persistent and aggressive disease. Antinuclear antibody (ANA) was present in 14% of children with JIA, but none of these children had uveitis. There was no significant relationship between ANA, oligoarthritis and uveitis.

Polyarthritis (40%) was the most common form of arthritis in the HIV-infected children, followed by the ‘SPA-like’ disorder (34%) and oligoarthritis (26%). The systemic manifestations in HIV-infected children included fever in 6 (17%), lymphadenopathy in 32 (91%), hepatomegaly in 15 (42%) and skin rashes in 10 (29%). The pyrexia in all the HIV-infected children could be explained on the basis of associated infections, and it was not the characteristic persistent, quotidian pattern of fever seen in the HIV-uninfected children with SOJIA. The skin rashes in the HIV-infected children were fixed and not associated with fever. Therefore, none of our children with HIV infection were classified as having systemic onset arthritis.

The prevalence of ‘SPA-like’ features was 34% in HIV-infected children, compared with 5% in HIV-uninfected children; these results were statistically significant ($P < 0.001$; CI 2.75–37.56). When the data in Table 2 are analysed according to the pattern of arthritis we note that 12 (71%) of the 17 children with ‘SPA-like’ features were HIV infected compared with 5 (21%) who were uninfected ($P < 0.001$; $\chi^2 = 24.03$).

The records of the patients with HIV infection were further analysed with respect to the pattern of joint involvement, clinical stage of HIV infection, Steinbrocker functional assessment [27] at presentation, duration of arthritis, and the clinical course of the disease (Table 3). Tests for CD4 counts were performed on 20 of the 35 children (Table 4).

**HIV-associated oligoarthritis (Table 3)**

Children with oligoarthritis were mainly males, with a male to female ratio of 3.5:1.

The mean number of affected joints was 2 (range 1–4). Three children (33%) had monoarthritis. The joints most frequently involved were the knees (66%), ankles (55%) and the small joints of the foot (22%). The synovial fluid was examined in three children, and showed few pus cells but the culture was sterile. Synovial biopsies examined in two children showed non-specific synovial inflammation. The HLA B27 antigen was negative in the five children who were tested. ANA were present in one child but eye involvement was absent in this group. Early signs of HIV infection (CDC clinical category A), were present in 77% of these children. Seven (77%) were in Steinbrocker functional class 2.

The median duration of arthritis, until last follow-up, was 12 months (range 1–84 months). The records of the clinical course of the disease were available for seven children (77%) who were followed up for a mean period of 12 months (range 3–36 months). The arthritis resolved within 6 months in four (57%) of the children (one within 6 weeks), two had persistent arthritis and one had recurrent episodes of arthritis despite treatment.

**HIV-associated polyarthritis (Table 3)**

There was no significant difference in the ratio of males and females in this group. The mean number of affected joints was 7 (range 5–13). The joints of both upper and lower limbs were frequently involved; knees 12 (85%), ankles 10 (71%), wrists 8 (61%), elbows 7 (54%) and small joints of the hands in 8 (57%). The hips and sacroiliac joints were each clinically involved in three children (23%). One child had a synovial biopsy which showed chronic non-specific inflammation. Rheumatoid factor was positive in two children. The HLA B27 antigen was negative in the five children tested. Moderate to severe HIV disease (CDC clinical category B/C) was present in 71% of children with polyarthritis. Ten children (77%) had significant disability (Steinbrocker class 3 and 4).

The median duration of arthritis until last follow-up was 17 months (range 3–56 months). Records of the clinical course of the disease were available for nine (64%) of the children who were followed up for a mean period of 26 months (range 2–60 months). Four children (44%) resolved within 6 months, three had persistent arthritis and two had recurrent arthritis despite treatment.

**HIV-associated ‘spondyloarthropathy-like’ disorder (Table 3)**

Seven of the 12 children with ‘SPA-like’ features (all males) had arthritis and enthesisitis (ERA). The mean number of active joints was 7 (range 1–14). The joints most frequently involved were the knees (86%), ankles (71%), hips (57%), midtarsal (43%) and

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**Table 2. Patterns of arthritis in HIV-infected and HIV-uninfected children**

<table>
<thead>
<tr>
<th>Condition</th>
<th>HIV infected, $n=35$ (%)</th>
<th>HIV uninfected, $n=97$ (%)</th>
<th>$P$ value</th>
<th>Odds ratio</th>
<th>Confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic onset arthritis</td>
<td>0 (0)</td>
<td>16 (16)</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oligoarthritis</td>
<td>9 (26)</td>
<td>38 (39)</td>
<td>0.15</td>
<td>0.54</td>
<td>0.21–1.37</td>
</tr>
<tr>
<td>Polyarthritis</td>
<td>14 (40)</td>
<td>38 (39)</td>
<td>0.93</td>
<td>1.04</td>
<td>0.44–2.45</td>
</tr>
<tr>
<td>Spondyloarthropathy</td>
<td>12 (34)</td>
<td>5 (5)</td>
<td>&lt;0.001</td>
<td>9.6</td>
<td>2.75–37.56</td>
</tr>
</tbody>
</table>
Mean CD4

Number tested 5 8 7

CDC immunological staging of HIV disease:

Number tested 5 8 7

Mean CD4 count (cells/μl) 564 346 850

Range 121–1512 24–1131 214–2471

sacroiliac joints (29%). One child with recurrent arthritis and enthesitis had significant involvement of the small joints of the hand. Three of these seven children had anterior uveitis and one had chorioretinitis. The HLA B27 antigen was negative in four of five children tested and one child with sacroilitis, anterior uveitis and enthesitis tested positive. Three (43%) had significant disability (Steinbrocker class 4). The median duration of arthritis in this group was 23 months (range 3–72 months). Five children (71%) were followed up for a mean period of 26 months (range 3–54 months). Arthritis resolved within 6 months in two (40%), two had recurrent episodes of arthritis and enthesitis and one had persistent arthritis despite treatment.

Discussion

This is the first report of a large series of children with arthritis who have HIV infection. Previous reports of children with arthritis and HIV infection have been limited to single case reports or small case series [1–6]. Our 35 children with HIV infection accounted for 27% of the total of 132 children with arthritis of unknown origin who were seen in our clinic during the study period.

One of the important questions which arises from this report is whether there is a true association between arthritis and HIV infection in children or whether this is a coincidental finding due to the high background prevalence of HIV infection in our population. Fortunately, this study was able to compare patients with HIV infection and arthritis with other HIV-uninfected children with idiopathic arthritis from the same ethnic group at a single centre.

The evidence for the association of HIV infection with arthritis in adults has included electron microscopy findings of tubuloreticular structures within the endothelial cells in synovial tissue [30, 31], isolation of HIV from the synovial fluid [9], detection of P24 antigen in cells of the synovial lining layer and in mononuclear cells of subsynovial inflammatory infiltrates [10] and the detection of HIV DNA within dendritic cells isolated from the synovium and peripheral blood of HIV-infected individuals [11]. Clinical studies which support a direct role of HIV in producing rheumatic manifestations include two reports on HIV-positive and HIV-negative patients in Argentina [18] and Mexico [19] which showed a significant increase in the prevalence of rheumatic manifestations in HIV-positive patients. Further support was provided by a longitudinal study of 117 patients, followed up for a mean period of 24.6 months (range 0.5–85 months), which showed that the majority of symptoms developed during the longitudinal evaluation [32].

Some of the observations arising out of this report, which suggest that there is a true association, will be discussed. First, the prevalence of HIV infection in 27% of our children with arthritis is higher than would be expected in children in the 2–12 yr age group. A recent Human Science Research report recorded the prevalence of HIV infection in children, of all ethnic groups between the ages of 2 and 14 yr, in South Africa to be 5.6% [24]. The prevalence of HIV infection in children aged 5–12 yr in a rural community in Uganda was noted to be 0.4% [33]. Our finding of an incidence of 27% HIV infection, in a fairly low risk group of 2–12 yr-old children with arthritis, supports an association between HIV infection and the development of arthritis in children.

Secondly, there was a significantly higher proportion of males, with a male to female ratio of 2.5:1, in our HIV-infected children compared with a nearly equal ratio of 0.9:1.0 for HIV-uninfected children. The male:female ratio in 521 Caucasian children with JIA...
in the United Kingdom was reported as 0.51% [34]. The higher proportion of males among HIV-infected children may be a reflection of the increased incidence of SPA-like features in this group.

Thirdly, SOJIA accounts for about 14–20% of JIA in most large studies of children with arthritis, and was recorded in 14% of 521 children in the UK study. We recorded SOJIA in 16% of our HIV-uninfected children. All HIV-infected children with systemic manifestations had concomitant infections to explain their systemic illness. The pattern of fever and types of skin rashes were different from those seen with SOJIA and therefore none of our HIV infected children were considered to have a systemic onset pattern of arthritis.

The fourth important observation is the significant increase in the prevalence of ‘SPA-like’ features, which was noted in 34% of HIV-infected children compared with only 5% of HIV-uninfected children \( (P < 0.001) \). Seventy-one per cent of all children with ‘SPA-like’ features were HIV infected. The male:female ratio in this group was 5.5:1. Burgos-Vargas et al. [35] describe classical SPA as a group of HLA B27-associated disorders that are characterized by enthesitis and arthritis affecting the lower extremities. Additional features include a variety of extra-articular manifestations with bacterial infections as triggers in some cases. ReA is defined as a form of arthritis that appears following an infection [35]. This term is usually restricted to HLA B27-associated ReA triggered by arthritogenic bacteria such as Salmonella, Yersinia, Campylobacter, Shigella and Chlamydia [35, 36]. The link between the SPA and ReA is the HLA B27 antigen.

The pattern of SPA in HIV-infected children differed from the classical SPA in that the HLA B27 was negative in seven of the eight children tested, four (33%) had involvement of the upper limbs and the arthritis resolved within 6 months in six (50%) of these children. Therefore we describe them as a ‘SPA-like’ disorder. Features that were suggestive of SPA in this group included arthritis and enthesitis (compatible with the ESSG criteria for SPA) and extra-articular manifestations of ReA like acute gastroenteritis, urethritis, conjunctivitis and ReA-triggering infections like Salmonella enteritides. HLA B27-negative ReA triggered by Yersinia and Campylobacteria infection is reported to follow a rather short, benign course of disease [35], similar to our cohort. This suggests that HIV virus or an unidentified co-infection may trigger a form of ReA in HIV-infected children. Upper limb involvement, although unusual, has been reported in patients with Salmonella and Yersinia ReA arthritis [35].

Our observation of an increase in the incidence of ‘SPA-like features’ in HIV-infected children is consistent with the findings of HIV arthritis reported in adult Black Africans [15]. SPA was considered a rare disease in Africa prior to the HIV epidemic. A previous report by Lutalo [37] identified only three adults with SPA in a series of 52 patients with rheumatic disease in Zimbabwe. The low prevalence of SPA in Africa was attributed to the low prevalence of HLA B27 (0.68% in Zimbabwe). With the HIV epidemic, large cohorts of adults from Zimbabwe, Rwanda and Zambia [13–17] were reported with HIV infection and arthritis and specifically a significant increase in HIV-associated SPA was noted. The largest series came from Zambia where Njobvu et al. [15] reported on 344 adults with arthritis who were tested for HIV infection; 289 were HIV infected. Of these 289 adults, 223 were considered to have HIV-associated SPA and 66 were classified as having arthritis alone.

The pattern of arthritis in 521 UK children with JIA [34], was reported as SOJIA (14%), oligoarthritis (45%), polyarthritis (27%), ERA (7%) and psoriatic arthritis (7%). In our cohort of JIA we recorded SOJIA (16%), polyarthritis (38%), oligoarthritis (38%) and ERA (5%). Juvenile chronic arthritis in Black South African children was described in 1984 by Haffejee et al. [38]. A detailed description of the pattern of joint involvement, the severity of arthritis and the associated features is contained in that paper.

The clinical findings are similar to our series of JIA, except that more children had polyarthritis (50 vs 38%). RF-positive polyarthritis was present in 9% of our JIA cohort compared with 7% in the UK cohort [34]. There was no significant relationship between ANA, oligoarthritis and uveitis in our series of JIA. A retrospective review of 35 African American and 137 Caucasian children with pauciarticular and polyarticular JIA reported racial differences in disease expression [39]. African American children were less likely to be ANA positive (33 vs 70.7%) and more likely to be RF positive (20 vs 3.8%). Uveitis was less common in African American children and was associated with a negative ANA. Haffejee et al. [38] did not find any relationship between ANA and oligoarthritis with uveitis among Black South African children.

Polyarthritis (40%) was commoner than oligoarthritis (26%) in HIV-infected children. Arthritis was the presenting feature of HIV infection in 78% of the children and it occurred at any stage of the HIV disease. HIV-associated arthritis may present as oligoarthritis, polyarthritis, with ‘SPA-like’ features or with systemic features. This study emphasizes the difficulty in distinguishing between HIV-associated arthritis and JIA at presentation and that HIV arthritis may mimic JIA.

Conclusion

The 27% HIV prevalence in a relatively low risk group of 2–12-yr-old Black African children with arthritis, together with the predominance of males and an increased prevalence of ‘SPA-like features’, suggests that HIV infection may be causally associated with arthritis in children. HIV-associated arthritis can mimic JIA and must be considered in the differential diagnosis of arthritis in children in an HIV endemic area. Arthritis may be the presenting feature of HIV infection or may occur at any stage of the HIV disease.

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References


