Curr Rheumatol Rep (2014) 16:421
DOI 10.1007/s11926-014-0421-z

Spondyloarthritis in Sub-Saharan Africa

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Published online: 18 April 2014
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Abstract Spondyloarthritis (SpA) is generally uncommon in sub-Saharan Africa, in part because of the rarity of HLA-B27 in this region. However, the relationship between HLA-B27 and SpA, particularly ankylosing spondylitis (AS), is complex. Despite the HLA-B*27:05 risk allele occurring in some West African populations, associated AS is not seen. In fact, most patients with AS are HLA-B27-negative, although there is emerging evidence that another class I HLA molecule, HLA-B*14:03, is associated with AS in black Africans. The Assessment of SpondyloArthritis International Society criteria for detecting early axial disease are of limited value in sub-Saharan Africa, because of both the rarity of HLA-B27 and very limited access to magnetic resonance imaging. Reactive arthritis (ReA), psoriatic arthritis, and undifferentiated SpA are seen mainly in the context of HIV infection, although the exact effect of the virus in the pathogenesis of arthritis is unclear. In Zambia, ReA is associated with the HLA-B*57:03 allele, which is paradoxically also associated with slow progression of HIV infection. HIV-associated ReA has a more protracted and aggressive course than standard ReA. Enthesitis-related arthritis is more common in children infected with HIV by vertical mother-to child transmission. Use of TNF inhibitors for axial disease is problematic, mainly because of cost, but also because of potential safety problems, especially reactivation of tuberculosis.

Keywords Seronegative arthritis · Spondyloarthritis · Spondyloarthropathy · Sub-Saharan Africa · Psoriatic arthritis · Ankylosing spondylitis · Reactive arthritis · Undifferentiated spondyloarthritis · Juvenile seronegative arthritis · Enthesitis-related arthritis · HIV · HIV-related arthritis · HLA-B27 · HLA-B14 · HLA-B57

Introduction

Spondyloarthritis (SpA), also referred to as seronegative spondyloarthritis or seronegative arthritis, was until recent times believed to be rare in sub-Saharan Africa, especially compared with other types of inflammatory arthritis including rheumatoid arthritis and gout. The standard explanation for the rarity of SpA is the almost complete absence of the HLA-B27 gene in many black African populations [1•, 2], which in European and Asian populations is a strong genetic risk factor [3••]. However, recent studies suggest that the relationship between HLA-B27 and SpA in sub-Saharan Africa is perhaps more complex. This has been most evident for HIV-associated seronegative arthritis, which has been increasingly recognised in this part of the world, but which is not associated with HLA-B27. In this article we review ankylosing spondylitis (AS), psoriatic arthritis (PsA), reactive arthritis (ReA), juvenile idiopathic arthritis (JIA), and HIV-associated arthritis in sub-Saharan Africa.

This article is part of the Topical Collection on *Spondyloarthritis*

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Ankylosing Spondylitis

Ankylosing spondylitis, the prototypic SpA, has substantial inter-ethnic variation in prevalence, being most common among people of European extraction and least common among sub-Saharan Africans [4••]. The condition is especially rare in southern and central African populations, where the frequency of HLA-B27 is <1 % [5]. After the initial report in 1975 of a single case of AS in a cross-sectional survey of 1352 black South Africans [6], a succession of studies supported the theory that AS was rare in black races of sub-Saharan Africa. Recent additions to the studies confirming this view come from central Africa, where only three cases of AS were recorded among 2370 patients with rheumatic complaints seen over a 15-year period in Kinshasa, Democratic Republic of the Congo [7]; and only four cases among 10,000 rheumatic patients seen over an 11-year period in neighbouring Congo Brazzaville [8].

The situation in West African populations is strikingly different, and intriguing. Despite allele frequencies for HLA-B27 of up to 7.8 % in Fula in the Gambia [9] and 9.7 % in Mali [10], AS is rarely seen at hospital and community level. Extensive studies of the Fula tribe failed to identify a single case of AS, despite carrier rates of 6 % for HLA-B27 and 32 % and 69 % for the HLA-B*27:03 and HLA-B*27:05 subtypes [11], respectively. Although HLA-B*27:03 is believed not to confer susceptibility to SpA, the absence of AS among carriers of HLA-B*27:05 remains unexplained; in Caucasians, this subtype is strongly associated with AS [3••]. Possible explanations for this difference among the Fula are the absence of an infectious initiator, or an unidentified protective genetic or environmental factor.

Conversely, black Africans who do develop AS seldom carry HLA-B27. The prevailing view is that the clinical and demographic features resemble HLA-B27-negative AS, as observed in other populations; that the age of onset is typically older, often in the fourth or fifth decade; that there is rarely a family history of SpA; and that there is a relative absence of extra-articular features, notably uveitis [12–14]. However: this may be an oversimplification of the genetic and clinical features of AS in black Africans, and HLA class I antigens may have a function in AS in black Africans. In Burkina Faso, 13 patients (0.9 % of 1439 patients) seen by a rheumatology service in the first two years of opening were diagnosed with AS, and six of 11 tested carried HLA-B27 [15]. This was the first study from sub-Saharan Africa to reveal a significant association between AS and HLA-B27 in black Africans, approximating to the prevalence in African Americans [16]. Subsequent work revealed that the HLA-B*27:05 allele occurred in seven (50 %) of the 14 cases of AS, compared with one (3.7 %) of 27 matched controls (OR=26) [17••]. Two small studies have indicated a possible function for the HLA-B*14:03 allele as a genetic risk factor, with the allele found in four of eight Togolese patients with AS, and in two of three ethnically unrelated Zambians with AS, but not in healthy controls in either population [18, 19]. In a review of 25 cases from different parts of Africa (three, eight, and 14 patients from Zambia, Togo, and Burkina Faso, respectively), the presence of HLA-B*14:03 and B*27:05 alleles was significantly increased in AS patients compared with healthy controls (32 % vs. 0.2 %, respectively, OR=191.5, $p=5.65\times10^{-16}$). On the basis of these findings, the authors suggest that the HLA-B*14:03 and HLA-B*27:05 alleles may have a common pathogenetic function in AS. In the same study they revealed the average age at disease onset to be 26 years, similar to the mean age of 25 years found in Togo in an earlier study [20].

In the clinical setting, diagnosis of AS in black Africans is often delayed until late in the course of the disease, when there is established spinal deformity and substantial associated physical disability [15]. The delay in diagnosis probably has many causes, including the shortage of health care professionals with rheumatology skills, and the lack of specialised tests, for example magnetic resonance imaging, in many parts of sub-Saharan Africa. The Assessment of SpondyloArthritis International Society classification criteria for axial SpA [21] will probably therefore be of limited clinical value in sub-Saharan Africa, because of the rarity of HLA-B27 and limited access to magnetic resonance imaging.

Treatment for AS is for the most part limited to non-steroidal anti-inflammatory agents and physiotherapy. Use of TNF inhibitors is limited, not only because of cost but also because of the risk of tuberculosis associated with this class of biological agents [22]. Moreover, given the high prevalence of HIV in sub-Saharan Africa, the safety of TNF inhibitors in this setting is questionable, although there are a few anecdotal case reports of these agents being safe for patients with early HIV infection [23].

Psoriatic Arthritis

The incidence of psoriasis and, by extension, PsA is low in sub-Saharan Africa, with some ethnic and geographical variability. Psoriasis was diagnosed in only 0.4 % of 2254 Ghanaians with a newly diagnosed skin condition [24], and 0.9 % of 1091 new patients seen in a dermatology clinic in Ibadan, Nigeria [25]. In a survey of 7039 patients seen over a one-year period in dermatology clinics at three teaching hospitals in Johannesburg, South Africa, psoriasis was diagnosed in 2.9 % of all patients, being most common in patients of Indian extraction at 9.6 %, and lowest in blacks at 2.1 % [26]. Not surprisingly, PsA is rarely seen outside the HIV setting in sub-Saharan Africa (see below).

The effect of genetic factors is unclear because one of the risk alleles for psoriasis in Caucasians, HLA-CW6, has a

frequency of as high as 15.1 % among black Africans, compared with 9.6 % among Caucasians [27•]. Dietary factors that may have a protective effect include a low intake of polyunsaturated fatty acids and a high intake of maize, which is the staple food in much of Africa and may have a protective effect because of its high linoleic acid content. Linoleic acid is a precursor of prostaglandin E2, which suppresses cellular immunity, and may thus reduce the risk of psoriasis [28].

Reactive Arthritis and Undifferentiated Spondyloarthritis

The reduction in gastrointestinal and sexually transmitted infections in much of the industrialized world has been accompanied by a reduced incidence of ReA [29]. Despite these infections being rife in sub-Saharan Africa, ReA initiated by bacterial pathogens in immunocompetent individuals remains uncommon, and it is mainly seen in the setting of HIV infection (see below). At a single rheumatology centre in the Democratic Republic of Congo, the most common inflammatory rheumatic disorder encountered in patients between 1988 and 2002 was SpA, with 185 cases of undifferentiated SpA and 55 of ReA reported [7], compared with no cases observed in studies from the same centre in previous decades [30]. In the West African state of Togo, 93 cases were documented among 13,517 patients over a period of 16 years [31]. These variations probably reflect the lower adult HIV prevalence in West African regions: approximately 2–4 %, compared with >15 % in southern and central Africa [32•].

A possible relationship between HIV infection and SpA was observed in Africa in the late 1980s [33]. In prospective studies from the 1990s in Zimbabwe, Zambia, and the Democratic Republic of Congo, a variety of HIV-associated arthritis was documented, including standard ReA, PsA, undifferentiated SpA, and "arthritis alone" [34–36]. In an attempt to better understand the clinical range, radiological features and prognostic factors of HIV-associated ReA, Njobvu and McGill identified 170 ReA patients over a five-year period [37]. Tellingly, only 59 % of patients met the diagnostic criteria for ReA at presentation, with a further 20 % deemed to have an undifferentiated SpA. Of 65 patients tested for HIV infection, 94 % tested positive (91 % of men, 100 % of women). Whereas the four patients without underlying HIV infection were all disease-free at six months, just over half of the 61 HIV-positive patients had polyarticular, lower-limb-predominant, progressive, and erosive arthritis, with six patients having evidence of sacroiliac erosions. Extra-articular features were common: anterior uveitis in a third of patients, keratoderma blenorrhagicum in 14.3 %, and stomatitis in 9.5 %. Both sexes had similar clinical, diagnostic, and radiographic features. This study emphasises the importance of long-term follow-up for accurate diagnosis. In a retrospective study of 3042 in-patients with rheumatic complaints, seen over a 15-and-a-half-year period in Congo Brazzaville, 178 patients were found to have HIV-associated arthritis [38]. Most patients had a non-erosive symmetrical arthritis (97.5 %) and polyarthritis (83.5 %), and in most cases the disease affected the lower limbs (99.3 %), with involvement of the big toe in approximately a quarter of cases. All patients were seronegative for rheumatoid factor. None of the patients met the European Spondylarthropathy Study Group classification criteria for SpA [39]. In this study, acute bouts responded to non-steroidal anti-inflammatory drugs within two to five weeks.

Although the pathophysiology of HIV-associated SpA is not clear, studies of Zambian subjects have established an intriguing association with the HLA-B*57:03 allele. In a study of 64 patients with SpA, 57 HIV-infected subjects without SpA, and 43 healthy controls, 54 (84 %) of the SpA patients tested positive for HIV infection [40]. The frequency of HLA-B*57:03 was increased in HIV-positive patients with SpA compared with HIV-positive individuals without SpA (OR= 8.3). In addition, HLA-B*5703 was over-represented in the subgroup of HIV patients with slow progression to AIDS compared with the subgroup of rapid progressors to AIDS, irrespective of the presence or absence of SpA (OR=10.8). This association was even more evident for the HIV group of slow progressors with SpA compared with those without SpA

Table 1 Juvenile idiopathic arthritis subtypes in sub-Saharan Africa

Country	Nigeria	Zambia	KwaZulu Natal, South Africa	Western Cape, South Africa
Sample size	23	85[a]	97[b]	78
Polyarticular RF negative	9 (39.1 %)	27 (34.6 %)	29 (29.9 %)	21 (26.9 %)
Polyarticular RF positive	4 (17.4 %)	9 (11.5 %)	9 (9.3 %)	11 (14.1 %)
Oligoarticular arthritis	7 (30.4 %)	25 (32 %)	38 (39.2 %)	21 (26.9 %)
Systemic onset	3 (13.0 %)	11 (14.1 %)	16 (16.5 %)	6 (7.7 %)
Enthesitis-related arthritis	–	5 (6.4 %)	5 (5.2 %)	18 (23 %)
Psoriatic arthritis	–	1 (1.1 %)	0 (0 %)	1 (1.3)

[a] All HIV-negative except seven HIV patients

[b] All HIV-negative

(OR=26.3). Altogether, these findings suggest that the HLA-B*57:03 allele, although protecting HIV patients against rapid progression of HIV infection, simultaneously increases the risk of SpA. In another study of 72 patients with SpA and 92 matched controls there was a significant increase in the incidence of undifferentiated SpA and of ReA with features of Reiter's syndrome among those who carried the HLA-B*5703 allele [19].

The function of bacterial pathogens as initiators for HIV-associated ReA has not been well studied, but at least in Lusaka, Zambia, there seems to have been a temporal relationship between an outbreak of dysentery in the 1990s and ReA. With public health measures to control dysentery in recent years there seems to have been a reduction in ReA [41].

Periarticular pain also seems to be more common with HIV infection. A Zambian study investigating the relationship between soft tissue lesions and early, asymptomatic HIV infection (WHO stage 1) selected 120 patients with soft tissue lesions seen in a rheumatology clinic. All of the patients who had sacroiliac pain (14), heel pain (14), costochondritis (3), and polyenthesitis at four or more sites (20) tested positive for HIV, compared with an HIV seroprevalence in Lusaka of 30 % in the 30–40-year age range at the time [42]. These findings suggest that isolated soft-tissue lesions and an unexplained elevated erythrocyte sedimentation rate in patients under 45 years are highly predictive of early HIV infection. Experience in Kenya has been similar. In a cross-sectional study of 193 anti-retroviral-treatment-naive HIV-positive patients seen at a comprehensive care HIV clinic in Nairobi, a substantial proportion complained of joint symptoms, most having no objective evidence of arthritis and only three having an inflammatory arthritis [43].

Juvenile Idiopathic Arthritis

Little was published on JIA in indigenous populations of sub-Saharan Africa until recently. In recent studies from Nigeria [44], South Africa [45, 46•], and Zambia [47••], all of the ILAR subtypes of JIA were identified (Table 1). The most common subtypes were rheumatoid-factor-negative polyarticular arthritis and oligoarticular arthritis. Of particular interest is that the oligoarthritis variant associated with uveitis and antinuclear antibodies, affecting mainly girls under the age of six years and commonly seen in Caucasians, is distinctly rare in black Africans [47••, 48].

In a South African study of juvenile idiopathic arthritis in children who contracted HIV through vertical mother-to-child transmission, inflammatory arthritis was strongly associated with extra-articular features of SpA [45]. Indeed, in sub-Saharan Africa, the presence of enthesitis and uveitis in children with inflammatory arthritis should serve as a prompt to test for possible underlying HIV infection [45, 49].

Conclusions

The knowledge base regarding SpA in sub-Saharan African has increased substantially over the past two decades. Recent studies confirm the overall rarity of SpA, but there is emerging evidence that HLA class I alleles may have a function in the etiopathogenesis of this group of inflammatory arthritis. There is also evidence that HIV infection in sub-Saharan Africa is associated with a range of musculoskeletal features of SpA, both in adults and children.

Compliance with Ethics Guidelines

Conflict of Interest Mohammed Tikly, Panganani Njobvu, and Paul McGill declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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