Rheumatoid arthritis in sub-Saharan Africa

Paul McGill

Throughout sub-Saharan Africa non-infective diseases, such as hypertension, diabetes, and epilepsy, are important causes of mortality and morbidity in populations already stressed by infection and malnutrition.1 Now, rheumatoid arthritis (RA), a one time rarity,2 is increasingly recognised in Africans.3-14 The Ugandan experience is typical. From Mulago Hospital, Kampala, an early report1 was followed by progressively larger series,3-5 culminating in a review of no less than 404 patients with classical RA, the largest series by far to come out of Africa.6 Rheumatoid arthritis appeared as a mild disorder with few extra-articular features, low morbidity, and low seropositivity in the earlier reports of 39 and 65 patients.4,5 The latest study6 accumulated over a decade reported many cases of severe disease, a high rate of seropositivity, the usual range of extra-articular features, and much disability. Reports from Kenya,6-9 central10-12 and southern Africa13,14 have mirrored the Ugandan experience. An exception may be Nigeria where Greenwood15 noted that RA was a mild and uncommon disorder, and this seems to apply to the present time.16 Thus although most reports suggest a changing pattern, formal epidemiological studies are necessary to establish whether this is real or apparent and to examine causative factors.

In Uganda planned population studies were shelved in the wake of war and are unlikely to be revived in the foreseeable future. Elsewhere there have been five community surveys in South Africa and one in West Africa reporting a variable but mainly low prevalence (0%-0.9%).17-22 The South African studies are, individually, relatively small in epidemiological terms (543-1070 respondents), but the uniform criteria used and overlapping investigators allow merging of the results. A total of 3955 adults over 15 years of age were examined and 12 cases of definite RA detected, a prevalence of 0.3%. In Western societies the average prevalence of definite RA is around 1%23 according to the 1958 American Rheumatism Association (ARA) criteria.24

A number of factors both artefactual and biological might contribute to the lower prevalence recorded in these African studies. Firstly, criteria: the South African investigators used the Rome criteria for inactive RA25 modified to exclude morning stiffness and a past history of polyarthritis and requiring positive clinical, serological, and radiological criteria for a diagnosis of definite RA. As shown in the West African study22 the historical and clinical criteria24 designed for use in developed societies when applied unmodified to rural African populations lead to overdiagnosis of RA. Such symptoms as morning stiffness and a past history of polyarthritis are unreliable as may be the clinical assessment of pain and stiffness in the tender swollen feet and hands of a largely barefoot population who undertake manual labour. In a recent European review26 of assignment criteria the Rome criteria for inactive RA and the New York clinical criteria27 achieved better epidemiological discrimination than both the 1958 and 1987 revised ARA criteria.28 Cathcart and O'Sullivan29 in Sudbury, Massachusetts, USA, using the New York criteria found only 16 (0.4%) of 4552 respondents compared with 39 (0.9%) who fulfilled the ARA criteria for definite RA. The Rome criteria are equally strict (in reality recognising that syndrome which a doctor would diagnose as RA in a patient seeking medical advice) and are likely to record a lower prevalence of RA than the ARA criteria.

A second factor to influence the prevalence of RA is the age structure of the population. If RA in the African is a disease predominantly of middle aged women then the younger populations of Africa will dilute the prevalence. When the combined South African results are adjusted for age the prevalence is seven adults per 1000 or 0.7%.

Genetic factors might also affect the prevalence of RA in various races. The familiar HLA-DR4 associations with RA in white subjects have recently been confirmed in South African Zulus.30 The prevalence of DR4 in patients (40%) and controls 10%, however, is lower than the respective 70%/30% in groups of white subjects. The significance of these observations is unclear at the present time and further studies including DR4 subsets in Africans are awaited with interest.

It is most likely, however, that the environment determines the prevalence and expression of RA, and there are some interesting differences in the various South African populations. The highest prevalence and most severe cases were found in a mixed urban community in Soweto (0.9%). In rural communities fewer and milder cases were encountered among the Tswana of West Transvaal 0.1%,31 the Xhosa of the Transkei 0.68%,19 and the Sotho in Lesotho 0.3%.20 The lowest prevalence of all occurred among the Venda peoples (0%) where even traditional healers failed to recognise the typical deformities of rheumatoid arthritic hands from photographs.32 So far there is no explanation for the relative paucity of RA in rural African
communities. Genetic differences are unlikely. The various Bantu tribes of South Africa (Zulu, Xhosa, Sotho, and Tswana) are genetically related. In the rural Venda a paucity of middle aged and elderly women might account for the very low prevalence in that population, but otherwise there were no outstanding differences in population structure of the remaining communities. Indeed the urban-rural difference becomes more striking in women over 50; in Soweto 3-8% compared with 0-8% in the combined rural populations. No convincing environmental factor has emerged to explain these differences either of a provocative nature—for example, stress associated with metropolitan living, or of a suppressive nature—for example, immunosuppression associated with infective disease, especially malaria. The explanation may quite simply be that RA, especially severe disease, carries a higher than usual mortality in rural communities of developing countries, where underdevelopment and lack of modern facilities are in stark contrast with Western rurality. Perhaps the better medical facilities in cities allow patients with RA to survive to develop more recognisable and severe features of the disorder.

For the future, development of optimum sets of criteria for use in rural communities of Africa and elsewhere in developing countries is desirable. These could be applied to compare the prevalence of RA in members of similar tribal groups living in their traditional homeland with the prevalence in those who have become established city dwellers. Perhaps these and other studies will be encouraged by the expansion of rheumatological expertise throughout sub-Saharan Africa.

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