

Ethnicity in rheumatic disease

Editorial

Definition of ethnicity varies depending on the context, precise purposes of clinical practice, census, national surveillance of vital and health statistics, etc. It defines particular groups on the basis of language in common, nation of origin, customs, and culture [1]. In contrast, 'race' is a biological construct and is used to classify human being biologically and genetically into homogeneous groups [1]. Both terminologies are often use interchangeably, though incorrectly; and in medical research use of 'ethnicity' is scarce [1,2]. Today's globalization and hence ethnic distribution of a nation is vastly influenced by the movement of ideologies across traditional national border, pattern of migration, and political debate; and to confront with the challenges of globalization, reshaping of healthcare delivery system is a necessity [3]. In this write-up, I address how ethnicity affects rheumatic conditions.

Ethnic disparity has been widely studied in SLE. Though SLE is less prevalent among native West-Africans, West-Africans descent settled in Europe/America experience the condition more than in native Europeans/Americans, and to be intriguing, many of them get the condition before migration [4]. In the UK, lupus is 6-7 times more prevalent in Afro-Caribbean women than that in same of European. In San Francisco study, age-adjusted prevalence of SLE was four-times higher in African-American women than in Caucasian women. The only data concerning lupus in Caribbean women revealed higher prevalence than that of women of European descent in the UK [5,6]. Among Asians, SLE was reportedly frequent with Chinese settling outside the China. In their study Wang et al. found that SLE was most common in Malaysian Chinese ethnic group as well [7]. Same result echoed with Chinese-Americans in Hawaii

[8,9]. Among New Zealanders', the high SLE risk was documented in Polynesian islanders [4]. In a systematic review, McDougall et al. described, the age-adjusted rate of SLE in various Alaska Native population, Oklahoma American Indian population, Phoenix American Indian population with lowest being in Crow, Arapahoe and Sioux tribes [10]. In Canadian First Nations population, though, the prevalence of SLE in British Columbia's Nuu-Chah-Nulth population was estimated at 0.3%, in the Manitoba First Nations population it was even higher. Several studies from Australia highlight SLE with indigenous prevalence being two fold to fourfold higher than the non-indigenous population [10].

On the other hand, among various Native Americans, the highest RA prevalence has been recorded in the Pima, Chippewa and Yakima tribes [4]. In Canada, RA prevalence varies in various indigenous people groups. In New Zealand, RA has been reported to be higher in Maori than that in non-Maori [10]. Although people of Afro-Caribbean ethnic UK people (mainly first-generation and second-generation migrants) appear to have low RA prevalence, in the US the condition is seen to affect both African-Americans and European-Americans equally. In South Africa, prevalence of RA in urban African populations was almost as equal as in European descent. However, prevalence of RA in rural South African populations was noted to be even lower than in urban populations, signifying differed environmental events in genetically homogenous communities might play some roles. In a recent published work, Samanta and colleagues mentioned about insufficient data regarding RA prevalence among South Asians living in UK [11]. They reported RA is less prevalent in native Chinese than that in European descent. Moreover, RA is

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more prevalent in Pakistanis living in England than that in native Pakistanis, nevertheless, the ratio is not as high as in the indigenous white populations.

Once, large vessel vasculitis had been considered to be common in the western world. Nowadays, it is being reportedly frequent among Indian populations, but, its exact prevalence in Indian subcontinent is yet to enumerate [2]. In their recent work, Pearce et al. have described, in UK, the incidence of ANCA-associated vasculitis does not differ among various ethnic groups [12]. Among New Zealand indigenous people, gout was found to be prevalent in Maori [10]. And acute rheumatic fever was prevalent in New Zealanders from under-privileged Maori, Pacific Islanders and Polynesians than other ethnic groups [13,14]. Lyme disease is frequent in Europe and Scandinavia and Familial Mediterranean fever (FMF) affects populations of Mediterranean descent; periodic fever associated with MKD is found in Dutch; Cryopyrin-associated periodic syndromes documented periodically among British and American neonates [15].

Spondyloarthritis (SpA), a group of seronegative inflammatory rheumatic disorders, where HLA-B27 association is unique. In Asia, the highest SpA prevalence was measured in China, whereas it was infrequent among Japanese, Malay, Philippines and middle-east Arabians. Likewise, the most SpA prevalent European country is Norway, though least frequent among Hungarian. SpA is even rarer in Australia and African countries [16].

Regarding osteoarthritis (OA), African-American women have higher prevalence of radiographic knee OA than do Mexican-Americans and Caucasians. The reported prevalence of knee OA in India and Bangladesh was 5.78% and 10.20% respectively. In their study, Islam et al. reported point prevalence of knee arthritis among Bangladeshi tribal groups, like, Chakma (72.7%), Marma (15.1%), Tripura (6.5%) and Tanchyanga (5.7%) [17]. Comparative adult skeletons study in Japan, China and France revealed higher OA changes in Asian skeletons than did Caucasians' [18]. Difference in the prevalence of overall and regional pain syndromes between ethnic populations has also been documented [19].

Why this ethnic disparity in rheumatic disorders? As per literature review, genetics-environmental-social-behavioral factors nexus play substantial role in the concerned issue [1]. For example,

variations in genetic factors reportedly influence manifestations and outcome of SLE, systemic sclerosis among ethnic groups. HLA-DRB1, HLA-DPB1, and HLA-B locus are common with RA patients'. Though DRB1 (*0401) allele is frequent with RA, ethnic variations of DR allele also exist and find some examples as stated here, DR1 (Israeli Jews, Asian Indians), DR3 (Arab population), DR9 (Chileans), DR10 (Spanish) [20]. PADI4 (peptidyl arginine deminase) gene also contributes in RA pathogenesis and found more often in Asian and European descent ACPA positive RA [21]. Besides, the strongest genetic associations of myositis found with HLA-II locus (in Caucasians, HLA-DRB1*0301 and HLA-DQA1*0501 alleles are common; in Asians, HLA-B7 is frequent) [22]. Disparity regarding HLAB27 locus among various SpA groups also exist, for example, HLA-B*2704 is predominant among Chinese SpA, whereas, B*2705 is frequent with Mexicans and Brazilians [16].

SLE may be induced following exposure to ultraviolet rays, cosmetic products, demethylating drugs; drug-induced SLE affects white rather than black-skin. Similarly, In ACPA-positive RA, tobacco smoke, silica dust, exposure to mineral oil, coffee consumption, etc. contribute [22]. The outbreak of eosinophilia-myalgia syndrome (EMS) with manifestations suggestive of systemic sclerosis was documented following ingestion of dietary supplements contaminated with L-tryptophan. EMS occurs in different parts of the world, including the UK, US, France, Israel, Japan, Germany, and Canada and found prevalent in white people. In addition, seasonal variation influences incidence of biopsy proven giant cell arteritis as found in Sweden and Israel population: in Sweden the condition was seen late winter and autumn, though, in Israel it was more in late spring and early summer [23]. Cyclical fluctuations of GCA few years apart was also reported in Jerusalem, northwest Spain and Sweden [24]. Alongside various other factors, role of pathogenic virus, bacteria inducing inflammatory rheumatic disorders is well-established as well [21].

Other than disease causation-association, ethnic disparity also exists in health-care access, utilization of prevailing health-care services, and quality of health care sources for various medical ailments including musculoskeletal conditions. For an example, African-Americans, Hispanics, and Asian descent Europeans

appeared to have very limited access to advanced health-care facilities than do Caucasians. These ethnic groups seem to have less understanding capability of sophisticated/advanced medical procedures, because of poor educational status as well. African-Americans, South Asian patients' and Hispanics are more likely to use prayer, herbs, ointments and less likely to use traditional medical care for arthritis symptoms than are Caucasians [2].

In conclusion, ethnicity and ethnic disparity greatly affect the prevalence, clinical manifestations, disease severity, even drug response to rheumatic musculoskeletal disorders. So, making treatment approach for various rheumatic disorders meaningful, understanding and accounting of all these factors could be of great value.

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