



Epidemiology of Psoriatic Arthritis

Is the epidemiology of psoriatic arthritis (PsA) truly changing? Is the incidence indeed rising? If so, what are the possible reasons? Is it because psoriasis is becoming more prevalent? Clearly, genetic factors do not change over a few decades; therefore, how are environmental factors influencing the disease? These are the questions that spring to mind on reading the article by Wilson, *et al* in this issue of *The Journal*¹.

PsA is a form of seronegative spondyloarthritis associated with psoriasis². Although the occurrence of arthritis associated with psoriasis was probably recognized as early as 1818, it was as recently as 1964 that PsA was recognized by the American Rheumatism Association (American College of Rheumatology) as a distinct clinical entity³. And it was as late as 1996 that studies on prevalence and incidence of PsA were published⁴. A recent review of studies undertaken to December 2006 has shown widely varying estimates of incidence and prevalence⁴. While estimates obtained from studies conducted within Europe and North America vary significantly, the most striking difference is between Europe and Japan. The incidence in Europe and North America ranged between 3 and 23.1 cases/10⁵, whereas that in Japan was only 0.1 cases/10⁵. Similarly, the prevalence in Europe and North America ranged between 20 and 420 cases/10⁵, but in Japan it was only 1/10⁵. This large difference is most likely due to differences in ethnicity, since low prevalence of other spondyloarthropathies in Japan has also been reported⁵.

The challenges in conducting epidemiological studies in PsA neatly elucidated in 1994 by O'Neill and Silman are still relevant³. The most important problem identified was lack of validated classification criteria. It should be noted, however, that although a number of proposed classification criteria were available, until now most epidemiological studies have used the co-occurrence of psoriasis and arthritis or the European Spondylarthropathy Study Group (ESSG) criteria to identify cases of PsA^{4,6}. Use of these criteria may

not be appropriate because even if patients with inflammatory arthritis were correctly identified, not all patients with psoriasis and inflammatory arthritis have PsA. Moreover, the ESSG criteria have poor sensitivity⁷. The original criteria for PsA proposed by Moll and Wright in 1973 were meant to be diagnostic rather than classification criteria⁸. A number of classification criteria have since been proposed but none have been universally accepted⁷. Recently, the CIASsification criteria for Psoriatic ARthritis (CASPAR) Study Group compared the performance characteristics of these criteria and developed a new set⁷. These new classification criteria were developed in patients with long-standing disease and in the original study had specificity of 98.7% and sensitivity of 91.4%. Subsequently, the criteria were shown to have excellent sensitivity in both early and late disease⁹. The CASPAR criteria were found to have been developed using sound measurement principles although they remain to be fully validated¹⁰. In fact, modifications of these criteria for epidemiologic studies have already been proposed¹¹. One major issue yet unresolved is the definition of "inflammatory musculoskeletal disease." The CASPAR criteria can only be applied to those fulfilling this mandatory criterion. Identifying inflammatory musculoskeletal disease is usually not difficult for rheumatologists, but may be a stumbling block for wider application of criteria by physicians, whether in the community or in dermatology practices. In spite of this drawback, CASPAR criteria have been recognized to be simple and easy to apply to data collected retrospectively^{9,12}. Moreover, using these criteria it is possible to classify patients as having PsA even when they do not have current, past, or family history of psoriasis.

In this issue of *The Journal*, using the resources of the Rochester Epidemiology Project and applying the CASPAR classification criteria, Wilson and colleagues have shown that the incidence of PsA has been rising over 30 years in males and females in Rochester, Minnesota, USA¹. The fol-

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lowing are key findings of their studies: (1) overall age- and sex-adjusted annual incidence of PsA per 100,000 was 7.2, with a higher incidence in males than females; (2) the age- and sex-adjusted incidence of PsA per 100,000 increased significantly from 3.6 between 1970 and 1979 to 9.8 between 1990 to 2000; (3) the point prevalence per 100,000 was 158 in 2000, with a higher prevalence in males than females; and (4) at incidence, most PsA subjects had oligoarticular involvement with enthesopathy. This is remarkable, since using the same resources the same research group has shown that the incidence and prevalence of rheumatoid arthritis is decreasing¹³. Interestingly, using the same resources, Shbeeb and colleagues showed in 2000 that the overall incidence of PsA was 6.59 per 100,000 between 1982 and 1991, and the point prevalence on January 1, 1992, was 1 per 1000¹⁴, which is similar to the estimate in the current study¹ for that decade. The current study differs from the 2000 study in that patients were classified as having PsA based on the CASPAR criteria, and thus 30 additional subjects were identified who did not have cutaneous psoriasis. The criteria were applied to data collected from charts using a standardized data abstraction form. However, applying the criteria on data collected from charts has major limitations. The CASPAR criteria can only be applied to patients with “inflammatory articular disease (joint, spine, enthesal)”. As mentioned above, inflammatory articular disease might be difficult for general practitioners and dermatologists to recognize. In fact, the CASPAR study group opined that it is not possible to apply the results of their study to the general population or to other clinical populations such as dermatology clinic populations, and the criteria have to be validated in these populations⁷. In addition, in the current study¹, the radiographic criterion was applied rather loosely, as “radiographic evidence of psoriatic bone changes of the hand or foot such as joint space narrowing, juxtaarticular new bone formation, ankylosis, spondylitis, joint erosions, periostitis, osteolysis, and sacroiliitis on plain films at time of PsA diagnosis” rather than “radiographic evidence of juxtaarticular new bone formation, appearing as ill-defined ossification near joint margins (but excluding osteophyte formation) on plain radiographs of the hand or foot,” which was the only radiographic feature found to have value in discriminating PsA from other inflammatory arthritides⁷. Most included cases satisfied criteria of current psoriasis and negative rheumatoid factor; however, the impact of misclassification based on radiographic criterion is not known but is likely to be low. The results also support the notion that early PsA presents as enthesitis and oligoarthritis and progresses over time to polyarticular disease.

Thus, if PsA incidence is truly increasing over the last 3 decades, what are the reasons? One obvious reason is better recognition of the condition by physicians in more recent years. Genetic or ethnicity related factors are unlikely to be

responsible for the observed change since there has not been any significant immigration or emigration from this “captive” population. It would be interesting to study how environmental factors have changed over the years to give us an explanation. However, such studies are best done prospectively. Corticosteroid use, pregnancy, trauma, and rubella vaccination have been shown to be most important^{15,16}.

What is the relationship between PsA and psoriasis? The estimated prevalence of PsA in patients with psoriasis ranges from 6% to 42%². To my knowledge, there are no published studies on the incidence of PsA in patients with psoriasis. Since most patients with PsA have psoriasis, if the prevalence of PsA is increasing, it is likely that the prevalence of psoriasis is also increasing. Wilson and colleagues have investigated this and have found similar increasing prevalence of psoriasis in Rochester¹⁷. They have also investigated predictors for development of PsA in psoriasis, and it will be interesting to see what the results are¹⁷. Thus, the prevalence of both psoriasis and PsA is increasing, and environmental rather than genetic factors are probably responsible.

In contrast to reports from a Canadian referral-clinic population¹⁸, excess mortality in the Rochester cohort could not be demonstrated. The authors suggest that subjects with severe PsA are probably under-represented in this cohort compared to the Canadian cohort. Interestingly, an update from the Canadian cohort showed that the mortality risk has improved in recent decades. This could be due to earlier diagnosis and more aggressive treatment in the more recent followup period¹⁸. It should be noted, however, that in the current study¹, 32% had radiographic erosions and 13% had osteolysis, indicating severe disease. The results are consistent with a similar population-based study using the Norfolk Arthritis Register¹⁹, and a hospital-based early arthritis clinic²⁰, where erosions were detected at baseline in 36.7% and 27% of available radiographs, respectively.

Thus, this population-based study on PsA in a well-defined geographic location has provided us with some very interesting results that need to be replicated in other comparable cohorts. This knowledge, coupled with knowledge from future genetic and environmental studies, will help us better understand the epidemiology, genetics, and genetic epidemiology of both psoriasis and PsA.

VINOD CHANDRAN, DM,

Division of Rheumatology,
Department of Medicine,
University of Toronto,
Clinical Research Fellow, University of Toronto,
Toronto, Ontario, Canada

*Address reprint requests to Dr. Chandran, Centre for Prognosis Studies in the Rheumatic Diseases, Toronto Western Hospital, 1E 412, 399 Bathurst Street, Toronto, ON M5T 2S8, Canada.
E-mail: vinod.chandran@uhnres.utoronto.ca*

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