Review Article

Psoriatic arthritis epidemiology

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Abstract. Psoriatic arthritis (PsA) is a chronic inflammatory arthritis that can cause severe joint deformities and disability. The expected prevalence of inflammatory arthritis among patients with psoriasis has varied from 7% to 40%. Early diagnosis and treatment of PsA is recommended to discourage from disease associated disability and breakdown of work place efficiency. The epidemiological studies are quite limited because of small and selective study populations, limited follow-up, and absence of validated PsA classification criteria. The epidemiology of psoriatic arthritis has been reviewed in this manuscript by summarizing incidence and prevalence estimates up to date and epidemiological studies indicating the degree of genetic load of PsA. Potential risk factors like demographic features, characteristics of psoriasis lesions and the effect of environmental and hormonal factors in epidemiological spectrum of psoriatic arthritis are analyzed.

Key words: Psoriatic, arthritis, epidemiology

1. Introduction

Psoriatic arthritis (PsA) occurs in approximately 7–40% of patients with psoriasis and detected as generally seronegative (1-3). Patients with PsA still practice considerable morbidity, including progressive joint destruction, functional disability, and amplified health care costs in spite of treatment advances with newly developed immune modulating agents (4-7). Consequently, early diagnosis and treatment of PsA is suggested to keep away from disease associated disability and failure of work place productivity (1,8).

Nevertheless studies up to now regarding the incidence and risk factors for PsA have been limited by small cross-sectional studies, discerning study populations, limited follow-up, and PsA classification criteria lacking diagnostic sensitivity. Numerous classification criteria with extensively variable sensitivity and specificity have been used, but none have been accepted across the world. Therefore, the comparability of the published incidence and prevalence estimates of PsA is challenging.

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For a long time the classification of PsA has been based on the Moll and Wright criteria (9). According to these criteria PsA can be classified when a patient with psoriasis has an inflammatory form of arthritis, negative rheumatoid factor, and one of five distinct clinical subsets (Table 1). Other criteria have since been proposed (10,11). More recently, the Classification of Psoriatic Arthritis (CASPAR) group developed classification criteria for PsA with a sensitivity of 91.4% and a specificity of 98.7% which were based on observational clinical data from 588 cases and 536 controls (Table 2) (12,13). Application of the new, validated diagnostic criteria recommends an exceptional occasion to document the incidence and factors predictive for the development of PsA in patients with psoriasis and assess time trends in incidence and prevalence rates of PsA using the same criteria consistently over time (13). CASPAR Study Group criteria for PsA is quite sensitive and specific and also very suitable for retrospectively composed records (13,14). It is also feasible to classify the patients for PsA, even if there is no history of psoriasis in family.

Prevalence of psoriatic arthritis
Since there are some difficulties in performing epidemiological studies in PsA, the number of the studies is limited. Deficiency of current classification criteria develops the main issue. Up to now for identifying cases of PsA the European Spondyloarthropathy Study Group (ESSG) criteria is mainly used (15). Although patients

with inflammatory arthritis were correctly identified, all patients with psoriasis and inflammatory arthritis don't have PsA. Therefore these criteria are not suitable in all circumstances. Furthermore, the ESSG criteria have reduced sensitivity for PsA (13). The original criteria for PsA proposed by Moll and Wright in 1973 were meant to be diagnostic rather than classification criteria (9). A number of classification criteria have since been proposed but none have been universally accepted (13). Recently developed CASPAR Study Group criteria for PsA has been recognized to be sensitive in both early and late disease and specific (13,14). Although PsA is found primarily in those with the skin disease psoriasis, PsA sine psoriasis is well described and diagnosed by rheumatologists (16) and the CASPAR criteria do not require skin disease psoriasis to be present for the diagnosis to be made (10). Therefore PsA should not be required only among patients with the diagnosis of psoriasis as cases without psoriasis will be

missed, as well as cases where the skin disease is mild and has not been diagnosed or treated separately. In fact, modifications of these criteria for epidemiologic studies have already been proposed (17). The definition of inflammatory musculoskeletal disease is the main problem. The CASPAR criteria can only be applied to those fulfilling this essential criterion. Although identifying inflammatory musculoskeletal disease is usually not difficult for rheumatologists, it might be difficult for general practitioners and dermatologists to recognize. However, CASPAR criteria are quite advantaged since it is suitable for the retrospective data in spite of this mentioned disadvantage (14,18).

We can examine the prevalence of PsA in two ways that one of them is estimating the prevalence of PsA in the general population. From Europe, the first reported population-based study in which association between psoriasis and rheumatoid arthritis was demonstrated was

Table 1. Five subtypes of psoriatic arthritis as described by Moll and Wright

1) oligoarticular (<5 tender and swollen joints) asymmetric arthritis
2) polyarticular arthritis
3) distal interphalangeal (DIP) joint predominant
4) spondylitis predominant
5) arthritis mutilans

Table 2. The CASPAR criteria

Inflammatory articular disease (joint, spine, orentheseal) with 3 points from the following:
1. Evidence of Psoriasis
(a)Current psoriasis: psoriatic skin or scalp disease present today as judged by a rheumatologist or dermatologist†
(b)History of psoriasis: A history of psoriasis that may be obtained from a patient, family physician, dermatologist, rheumatologist, or other qualified healthcare provider
(c)Family history of psoriasis : A history of psoriasis in a first- or second-degree relative according to patient report
2. Nail dystrophy
Typical psoriatic nail dystrophy including onycholysis, pitting, and hyperkeratosis observed on current physical examination
3. A negative test for RF
By any method except latex but preferably by enzyme-linked immunosorbent assay or nephelometry, according to the local laboratory reference range
4. Dactylitis
(a)Current dactylitis: Swelling of an entire digit
(b)History of dactylitis: recorded by a rheumatologist
5. Radiographic evidence of juxtaarticular new bone formation
Ill-defined ossification near joint margins (but excluding osteophyte formation) on plain radiographs of the hand or foot
† Current psoriasis is assigned a score of 2; all other features are assigned a score of 1.

performed by Hellgren in Sweden; the prevalence was 0.02% (19). Next study from Netherlands reported a prevalence of 0.05% (20). In a survey from France using a telephone questionnaire followed by physical examination parallel conjecture was obtained (21). The prevalence of PsA in Greece was reported as 0.06% between 1982 and 2001 (22). Trontzas et al. detected the prevalence of PsA as 0.17% (23). While 87.5% of the cases were accurately diagnosed by rheumatologists; just 7.7% of the cases were detected by non-rheumatologists. On the light of these findings challenges can be easily noticed in non-rheumatologists' diagnose of PsA. A prevalence of 0.195% from Western Norway (24), 0.164% from Iceland (25) and 0.42% from Italy was detected (26).

As a result of these surveys, distinctions in the estimated prevalence are realized in different parts of Europe. Ethnic deviation, different techniques and diagnosing ways for the disease may be the reasons for this situation.

In the United States, Shbeeb et al. (27) reported a population prevalence of 0.101% in Rochester, Minnesota. A prevalence of 0.158% demonstrated by Wilson et al. (28) in Olmsted County, Minnesota, using newly developed CASPAR in 2000. A telephone survey study revealed a prevalence of 0.25% in US (29).

The prevalence is reported to be 0.00001% in Japan (30), 0.01 - 0.1% in China (31) and 0.47% in an Australian Aboriginal community (32). The prevalence in various populations has been summarized in Table 3.

Table 3. Prevalence of psoriatic arthritis

Population-based prevalence of PsA		
Country	Study	Prevalence
Europe		
Sweden	Hellgren, 1969	0.02%
Netherlands	Van Romunde, 1984	0.05%
Greece	Alamanos, 2003	0.06%
Greece	Trontzas, 2005	0.17%
France	Saraux, 2005	0.19%
Italy	Salaffi, 2005	0.42 %
Norway	Madland, 2005	0.195%
America		
USA	Shbeeb, 2000	0.1%
USA	Gelfand, 2005	0.25%
Asia		
Japan	Hukuda, 2001	0.00001%
China	Zeng, 2008	0.01–0.1%

The other trend for the prevalence is to detect the prevalence of PsA in patients with psoriasis, since psoriasis precedes arthritis or arises simultaneously in most of the patients with PsA (33). From England a prevalence of 13.8% was reported (34) whereas Gelfand et al. (29) detected the prevalence of PsA as 11% in patients with psoriasis in USA. Wilson et al. (35) demonstrated that <10% of psoriasis patients develop PsA over a 30-year period (Table 4).

Table 4. Prevalence of PsA In psoriasis patients

Country	Study	Prevalence
United Kingdom	Ibrahim, 2009	13.8%
USA	Gelfand, 2005	11%
USA	Wilson 2009	<10%

PsA prevalence in psoriasis patients has been reported as between 6 to 42% by means of clinic-based studies (2).The prevalence ranges from 7.7 to 36% in Italian patients with psoriasis (36-38). In a study by Alenius et al. (38) from Sweden the prevalence of PsA was 48% and in the current studies from Germany prevalence was reported as 19–20.6% (40,41). It was shown that the prevalence increased to 20.5% with time by means of the most current international study involving clinics from the UK, Italy, France, Spain and Germany (42). The prevalence of arthritis in psoriasis patients from Croatia was reported as 15.3% and from Iran 9.1% and Japan 1% (43-45).

Therefore it gives the impression that the prevalence of PsA is higher in the patients of dermatology practices according to the data from the population.

Incidence of psoriatic arthritis
Studies to date concerning the incidence and risk factors for PsA have been limited by small and discriminating study populations.

The overall incidence of PsA was reported as 6.59 /100,000 between 1982 and 1991 by Shbeeb et al. (27) and later the overall annual incidence of PsA in Rochester, Minnesota was detected as 7.2/100,000 by Wilson et al. (28) by using the CASPAR classification criteria. Amusingly, the incidence increased considerably from 3.6 in 1970–79 to 9.8 in 1990–2000 (28). This was the first report on the time trends in the incidence and prevalence of PsA using the CASPAR criteria over a 30-year period in a geographically-defined population. The incidence of PsA in various populations has been summarized in Table 5.

Wilson et al. (35) reported incidence and clinical predictors of psoriatic arthritis in patients

with psoriasis in another population-based epidemiologic research facilitated in Olmsted County. An increasing incidence of 1.7%, 3.1%, and 5.1% at 5, 10, and 20 years following psoriasis incidence was determined. In this population-based data demonstrated that <10% of psoriasis patients develop PsA over a 30-year period and scalp psoriasis, nail dystrophy, and intergluteal / perianal psoriasis were significantly associated with an increased PsA risk and the risk of PsA increases in subjects with ≥3 psoriasis sites, suggesting that the risk of PsA is higher in psoriasis subjects with more extensive disease (35).

Table 5. Incidence of psoriatic arthritis

Country	Study	Incidence
Finland	Kaipiainen-Seppanen, 1996	6.1
Sweden	Soderlin, 2002	8
Greece	Alamanos, 2003	3.0
Finland	Savolainen, 2003	23.1
USA	Shbeeb, 2000	6.6
USA	Wilson,2009	7,2
Japan	Hukuda, 2001	0.1

Eder et al. (46) performed a prospective study to determine the incidence of PsA among psoriasis patients. It was the first study that has prospectively assessed the incidence of PsA among psoriasis patients. After 4 years of follow up, the annual incidence rate was 1.87 cases among 100 psoriasis patients. It was suggested that the incidence rate of PsA may be higher than previously reported, especially among patients with moderate to severe psoriasis.

An international clinic-based study from Europe reported the incidence of PsA among psoriasis patients remained constant (74 per 1000 person-years) (42).

Genetic epidemiology psoriatic arthritis

The genetic basis of psoriasis and PsA is supported by family based investigations, population based epidemiological studies, association studies with human leukocyte antigens (HLAs), genome-wide linkage scans, and candidate gene studies within and outside the major histocompatibility complex (MHC) region. Epidemiological studies indicating the degree of genetic load of PsA are dense. Moll and Wright (47) reported the most vigorous study estimating the strong familial clustering of PsA in 1973. First and second degree relatives of 88 patients with PsA were evaluated consequently the general prevalence of PsA among first degree

relatives was 5.5%. Since the premeditated prevalence of PsA in the UK population is 0.1%, the risk for affected first degree relatives is 55, significantly higher than those obtained for psoriasis. This study also renowned a 19-fold increase in prevalence of psoriasis among first degree relatives of probands with PsA evaluated with the general population.

It is now admitted that psoriasis and PsA are consistent with a multifactorial pattern of inheritance across the world (48,49). For psoriasis and PsA, a non-mendelian mode of transmission submitted as genomic imprinting (50,51). Genomic imprinting refers to an epigenetic effect that causes differential expression of a gene depending on the sex of the transmitting parent (52). A recent linkage study in PsA distinguished significant linkage only when assessing the transmission of alleles of paternal origin (53). Consequently the existence of this epigenetic phenomenon should be well thought-out for integration in the genetic model for linkage studies, as its inclusion may manipulate the confirmation for a linkage.

PsA patients with early onset psoriasis were more probable to have a family history of psoriasis or PsA (60% in early onset, 30% in late onset; p=0.001). In the early onset group considerable differences in other clinical features such as skin lesions preceding joint lesions, a lower number of actively inflamed joints at presentation, a higher frequency of spondyloarthropathy, and differential expression of HLA antigens (HLA-B17, HLA-Cw6) were also renowned (54).

In a another study of PsA, it was reported that if there was already one affected child in the family, the corresponding risk for another affected sibling was 0.10, 0.22, or 0.31 if no parents was affected, the mother was affected, or the father was affected, respectively (55). So far only one genome-wide scan has been fulfilled in PsA, and this study localized a candidate region on chromosome 16q (53).

HLA-Cw*0602 is increased among those with PsA and is also associated with an earlier age of onset of psoriasis (56,57). HLA-B27, HLA-B38, and HLA-B39 have also constantly been noted to have an amplified frequency in PsA cases compared with controls (58,59). With respect to disease expression, HLA-B27 is associated with back involvement, while HLA-B38 and HLAB39 arisen more often among patients with peripheral polyarthritis. (58,59). In addition, connections with IL12B and IL23R and TNF-α -238 polymorphism have recently been established (60,61).

Briefly, there is a clear genetic donation to psoriasis and PsA. Epidemiological and immunological evidence proposes that some genetic determinants are likely to be allocated between these two diseases even though there are some separate genetic differences.

Demographic features

Nearly all of the cases arise between 45 to 64 years old (22,27,46,62). In contrast to RA which has a female predominance, PsA was regarded as an equigender disease. Four retrospective (22,27,30,46) and two prospective studies (47,48) found an extensive disparity in the male/female ratio, from 0.4 to 1.3. The study Wilson et al. (28) performed showed that the incidence of PsA in women is less than the incidence in men until the sixth decade of life. Consequently an achievable hormonal influence in the onset of PsA should be well thought-out.

The effect of hormones

PsA improves during pregnancy, but disease flares are common during the post-partum period when estrogen levels are in flux (28). In addition patients treated with estrogen-modifying drugs were less likely to develop PsA (63). Briefly, the potential role of sex hormones in the etiology of PsA should be considered on the light of these findings. To illuminate the composite role of sex hormones in the pathogenesis of PsA in men and women additional research should be performed.

Environmental factors

Infectious agents (viral and bacterial) and physical trauma have been recommended as probable risk factors for the onset of PsA. Psoriatic lesions arising from traumatic areas, known as the Koebner phenomenon, afford the hypothesis of deep Koebner phenomena as an explanation for posttraumatic PsA (63) and a history of injury requiring medical consultation had an odds ratio of 2.53.

Rubella vaccination, recurrent oral ulcers and HIV infection (63,64) are other factors that were associated with development of inflammatory arthritis in patients with psoriasis.

In addition there is rising verification that PsA patients show an enhanced reaction to gram positive infection by magnifying the inflammatory cascade (65,66). Elevated T cell reactivity levels to bacteria in serum and enhanced proliferative response to staphylococcal super antigens occur in patients with PsA.

Smoking is a risk factor for the development of psoriasis and there is a dose–response remarkable correlation (67). Smoking prior to psoriasis onset diminishes time to onset of PsA, but smoking after psoriasis onset raises time to development of PsA (68).

Characteristics of psoriasis lesions

The usual setting seen in 75 to 85% of patients demonstrates that psoriasis leads the onset of arthritis by an average of 10 years; in 5-10%, psoriasis may occur concurrently or follow the onset of arthritis (1,2,5) Psoriasis vulgaris is the major form of psoriasis associated with PsA, but other variants include pustular, guttate, flexural and erythrodermic psoriasis.

Nail lesions, most commonly nail pitting (but sometimes onycholysis), take place more frequently in patients with PsA than patients with uncomplicated psoriasis (58).

Wilson et al. (35) reported that scalp psoriasis, nail dystrophy, and intergluteal/perianal psoriasis were significantly associated with an increased PsA risk and the risk of PsA increases in subjects with ≥3 psoriasis sites, suggesting that the risk of PsA is higher in psoriasis subjects with more extensive disease.

Consequently, the epidemiology of psoriatic arthritis is affected from many factors; genetic, environmental risk factors, different ethnicities and geographic regions. Lack of standard classification of PsA for studies is also challenging. However, by the help of newly organized and extended further studies it will be better to realize pathogenesis, recognize drug targets, forecast disease route and treatment response in patients with PsA.

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