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BRIEF REPORT

Smoking is associated with a worse self-reported health status in patients with psoriatic arthritis: data from a Swedish population-based cohort

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Abstract The aim was to study possible associations between smoking habits and self-reported clinical features in a large population-based cohort of patients with psoriatic arthritis (PsA). All subjects with PsA who had sought health care in the period 2003–2007 were identified using a regional health-care register. In 2009, all those identified who were 18 years of age or more (n=2,003) were sent a questionnaire with questions on smoking, health-related quality of life [EuroQol five-dimension (EQ-5D)questionnaire], function [Health Assessment Questionnaire (HAQ)], pain, fatigue, and global health. We performed age- and sex-adjusted regression analysis to compare health status outcomes in never and ever smokers. Altogether, 1,185 subjects (59 %) returned the questionnaire. Mean age was 57 years (SD 13.5), and 58 % were women; 38 % were never smokers and 62 % were ever smokers. Mean age at disease onset was 38.2 years (SD 13.2) and 41.2 years (SD 13.6), respectively (p=0.001). In age- and sex-adjusted data, ever smokers reported worse EQ-5D (p=0.009); worse reports of global health (p=0.01), pain (p=0.01), and fatigue (p=0.04); and a higher number of painful body regions (p=0.04) compared to never smokers. In this population-based PsA cohort, patients who were ever smokers reported worse health status than never smokers. Besides being a possible result of a worse PsA in ever smokers, impaired health status could also be an effect of unstudied comorbidities. Further longitudinal studies are needed to gain a better understanding of cause and effect. However, smoking cessation should be recommended because of general health considerations as well as disease-specific issues.

Keywords Function · Pain · Psoriatic arthritis · Quality of life · Smoking

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Introduction

Smoking is an environmental factor that is known to have a negative effect on the disease course and treatment of rheumatic diseases such as rheumatoid arthritis [1] and ankylosing spondylitis [2]. Furthermore, in patients with axial spondylarthritis (SpA), smokers have earlier onset of disease, higher levels of disease activity, poorer physical functioning, and worse health-related quality of life (HRQoL) than patients who do not smoke [3]. Whether or not these findings can be extrapolated to other SpA subgroups such as psoriatic arthritis (PsA) is unclear, and research findings are conflicting [4–6].

The effects of smoking on disease development might be different in psoriasis and PsA; smoking is a risk factor for psoriasis [7], and an inverse relationship between smoking and development of PsA in patients with psoriasis was found by Eder et al. [5]. However, contradictory information has also been published in a study on women with psoriasis in the USA [8]. Regarding the disease course, clinically, tobacco smoking is well known to worsen the skin condition in patients with psoriasis [7, 9]. Another recent study found that smoking was associated with a worse outcome in physical function in patients with established PsA [6]. Patients with PsA have an increased risk of developing cardiovascular comorbidities [10] and features connected with the metabolic syndrome [11], both of which are well-known to be affected by smoking and/or a sedentary lifestyle in the general population. Smoking has also been reported to be associated with musculoskeletal pain in people with no known rheumatic disorder [12].

The purpose of this study was to investigate possible associations between smoking habits and self-reported health, function, pain, and fatigue in a large population-based cohort of patients with a diagnosis of PsA.

Patients and methods

Study population

The cross-sectional data in this study were based on a postal questionnaire-based survey performed in 2009 in the southernmost county of Sweden. Patients with PsA who would be eligible for the postal survey were identified from the Skane Health Care Register (SHCR); they are a subgroup of the SpA Scania cohort, which has been described in detail elsewhere [13].

The SpA Scania cohort was established in 2008 by identifying all subjects in the SHCR who, at any time during five calendar years (2003–2007), had been given a diagnosis of SpA—as identified by International Classification of Diseases (ICD)-10 codes. The SpA diagnoses had a high level of accuracy when compared with clinical diagnoses retrieved from medical records [13]. In Sweden, all health-care visits (public and private, both inpatient and outpatient) are registered in regional healthcare registries such as the SHCR using a unique personal identification number with information on health-care provider, date of visit, and ICD-10 diagnosis as given by physicians in primary or secondary care. The data in this study were obtained from a questionnaire sent in 2009 to SpA Scania cohort patients who were 18 years of age or more. All the patients with a PsA diagnosis (L40.5, M07.0, M07.1, M07.2, M07.3) who were eligible for inclusion in this study were required to be registered in the SHCR at least once by a rheumatologist or intern or at least on two separate occasions by any other physicians in primary or secondary care. Altogether, 2,003 patients with PsA who were identified from the SpA Scania cohort received the questionnaire in 2009.

The postal survey

The questionnaire included several validated patient-reported outcome measures (PROMs). The composite questionnaire was also tested for face and content validity. Age at disease onset, smoking habits, physical function, pain, fatigue, global health, and HRQoL were the self-reported data retrieved from the questionnaire, while patient characteristics such as age, sex, and ICD-10 diagnosis were retrieved from the SHCR.

Smoking habits were reported as never, past, or present smoker. HRQoL was assessed with the EuroQol five-dimension (EQ-5D) questionnaire with a summary score ranging from 0 (no health) to 1 (full health) [14]. Physical function was measured with the Health Assessment Questionnaire (HAQ), with scores ranging from 0 to 3 (best to worst) [15]. Global health, fatigue, and pain were measured with Numerical Rating Scale (NRS) ranging from 0 to 10 (best to worst). Pain distribution was measured as a number of painful regions with a pain mannequin divided into 18 regions, yielding a score of 0–18 (with 0 = no painful regions) [16]. The patients also reported whether psoriasis was present (yes/no).

Statistical analysis

Linear regression analysis was performed with EQ-5D, HAQ, NRS pain, NRS fatigue, and NRS global health and with a number of painful regions as dependent outcome variables. Smoking was introduced as an independent variable, with smoking groups collapsed into ever and never smokers, but analyses were also performed by splitting the ever smoker group into current and past smokers. All data were controlled for age and sex and presented with parameter estimates (B) and 95 % confidence interval (CI). Differences between groups were analyzed with chi-square tests or t tests where appropriate. Data analyses were performed using SPSS for Windows v. 20.

Ethics

Informed consent was obtained from all patients according to the Declaration of Helsinki, and the Central Ethical Review Board in Lund, Sweden, approved the study (301/2007, 406/2008).

Results

Seventy-seven percent of the 2,003 patients with PsA responded to the survey; 18 % declined participation, and 1,185 patients (59 %) returned the questionnaire. Of the latter patients, mean age was 57.1 years (SD 13.5), and 681 (58 %) were women; 1,173 (99 %) responded to the smoking question (Table 1). Analysis of nonresponders showed that the higher age was associated with a better response rate in both men and women, while gender was not.

Of the 1,173 patients (672 women and 501 men) who responded to the questionnaire, 448 (38 %) were never smokers and 725 (62 %) were ever smokers. Of the ever smokers, 212 (18 %) were current smokers and 513 (44 %) had stopped smoking (past smokers). In the female group, 35 % were never smokers, 24 % current smokers, and 41 % past smokers vs. 43, 10, and 47 %, respectively, in the male group. Never smokers were slightly younger at the self-reported disease onset and at the time of diagnosis given by the physician (p=0.001 and p=0.000, respectively), but they all had similar disease duration (p=0.5) at the time of the survey (Table 1). Reports of the presence of skin psoriasis were reported by 78 % of never smokers, 82 % of current smokers, and 75 % of past smokers.

Ever smokers reported worse HRQoL (EQ-5D), global health, pain, and fatigue, and a higher number of painful areas than never smokers (based on linear regression analysis). The parameter estimates (B) and 95 % CI for ever smokers for EQ-5D were −0.04 (−0.073; −0.011, p=0.009), for HAQ 0.043 (−0.024; 0.11, p=0.21), for global health 0.36 (0.087; 0.637, p=0.01), for pain 0.38 (0.092; 0.674, p=0.01), for fatigue 0.34 (0.018; 0.661, p=0.04), and for painful regions 0.54 (0.016; 1.072, p=0.04) (Table 2). Splitting of the ever smoker group into current and former smokers showed that current smokers reported a statistically significantly worse outcome in EQ-5D and in a number of painful regions than former smokers (p=0.02 and 0.04, respectively). All analyses were controlled for age and sex.

Discussion

In this population-based study with a large number of PsA patients responding to a questionnaire survey, ever smokers reported worse health status than never smokers. These findings are in accordance with earlier findings from other phenotypes in SpA [2, 3] and on patients with RA [1].

In this study, reports of disease onset and age at doctor's diagnosis were slightly but statistically significantly higher in ever smokers than in never smokers. There is conflicting evidence in the literature concerning the effect of tobacco on disease development and severity in PsA. Some studies have found that tobacco can delay disease onset in PsA [5], while others have noted an earlier development of PsA due to smoking [8]. The latter has also been found in subjects with axial SpA [3]. Due to our cross-sectional design, we can neither support nor dismiss this possibility and longitudinal studies are warranted. It is possible that heredity may cause earlier disease onset in the never smoker group. In a post hoc analysis, we found that heredity was reported by 72 % of never smokers and by 67 % of ever smokers, with a statistical significance of p=0.06, suggesting that this might be a mediator for age at diagnosis.

Table 1 Breakdown of data on never and ever smokers in 1,173 subjects with PsA. If not otherwise stated, the data are presented as mean (SD)

	Never smokers	Ever smokers		
	n=448	Current and past smokers, n=725	Current smokers, n=212	Past smokers, n=513
Men/women, n (%)	215/233 (48/52)	286/439 (39/61)	49/163 (23/77)	237/276 (46/54)
Age at survey, years	54.31 (14.67)	58.72 (12.44)	54.54 (12.60)	60.44 (11.96)
Age at disease onset, years	38.23 (13.17)	41.15 (13.65)	39.23 (14.28)	41.96 (13.31)
Age at diagnosis, years	42.26 (13.38)	46.02 (13.15)	43.37 (13.63)	47.09 (12.81)
EQ-5D (0–1)[a]	0.68 (0.24)	0.63 (0.26)	0.58 (0.30)	0.65 (0.24)
HAQ (0–3)[b]	0.59 (0.58)	0.71 (0.61)	0.78 (0.64)	0.68 (0.59)
Global health (0–10)[c]	3.85 (2.36)	4.35 (2.28)	4.67 (2.33)	4.22 (2.25)
Pain (0–10)[c]	3.85 (2.43)	4.38 (2.47)	4.70 (2.52)	4.25 (2.43)
Fatigue (0–10)[c]	4.53 (2.76)	4.95 (2.68)	5.40 (2.68)	4.77 (2.66)
Pain areas (0–18)[d]	7.24 (3.98)	7.90 (4.27)	8.68 (4.46)	7.58 (4.16)

[a] 0–1, worst to best

[b] 0–3, best to worst

[c] 0–10, best to worst

[d] 0–18, best to worst

Table 2 Linear regression analysis with parameter estimates (*B*) and 95 % confidence interval (CI)

	EQ-5D[a], *n*=940 *B* (95 % CI)	HAQ[b], *n*=992 *B* (95 % CI)	Global[c], *n*=991 *B* (95 % CI)	Pain[c], *n*=993 *B* (95 % CI)	Fatigue[c], *n*=991 *B* (95 % CI)	Pain areas[d], *n*=864 *B* (95 % CI)
Men	0	0	0	0	0	0
Women	−0.6 (−0.091; −0.030)	0.34 (0.280; 0.411)	0.84 (0.570; 1.1.2)	0.99 (0.708; 1.271)	1.12 (0.805; 1.427)	1.68 (1.161; 2.194)
Age, years	0.00 (−0.002; −0.001)	0.10 (0.008; 0.012)	0.015 (0.005; 0.025)	0.013 (0.003; 0.024)	0.003 (−0.015; 0.008)	−0.002 (−0.021; 0.016)
NS	0	0	0	0	0	0
ES	−0.04 (−0.073; −0.011)	0.043 (−0.024; 0.11)	0.36 (0.087; 0.637)	0.38 (0.092; 0.674)	0.34 (0.018; 0.661)	0.54 (0.016; 1.072)

NS never smokers, *ES* ever smokers

[a] 0–1, worst to best

[b] 0–3, best to worst

[c] 0–10, best to worst

[d] 0–18, best to worst

The presence of psoriasis at the time of the survey in the three smoking groups was based on a single question in the questionnaire. We do not have any information regarding a temporal association between PsA and psoriasis. This has been found to make a difference in some earlier studies, where smoking was found to be a risk factor for the development of psoriasis but to be a protective factor if PsA developed after the skin disease [5, 7].

Eighteen percent of the patients were current smokers in this study. Similar figures were found in a Swedish National Survey carried out in 2009, where 14 % of the responders admitted to being daily tobacco smokers in the Skane region, which is where our study sample was based. This can be compared to the figure of 12 % for the whole of Sweden with no gender differences. On a national level, 57 % of respondents reported that they had never smoked while 18 % had smoked in the past (http://www.fhi.se/Statistik-uppfoljning/Nationella-folkhalsoenkaten/Levnadsvanor/Tobaksvanor/, accessed 3 July 2013). In this study, the female group of current smokers was twice the size of the male group. One possible hypothesis is that there is an underreporting of smoking in men. Another more speculative hypothesis is that female smokers might be more prone to develop PsA compared with men, which however cannot be supported by earlier studies. Possible gender differences in current smokers found in this study need to be further studied.

Associations between worse physical functioning or exercise and smoking have also been found in patients with PsA [6]. Tillett et al. [6] found an elevated HAQ score in smokers with PsA, which was not clearly supported by findings in our study. An association between pain and smoking is commonly found in chronic musculoskeletal pain studies, but cause and effect is still unclear [12]. The mechanism behind a possible effect of smoking on pain is unclear, although nicotine in itself has been reported to have an analgesic effect on the central nervous system [17]. Tobacco smoke has been suggested to increase oxidative stress due to an antioxidant imbalance, resulting in a pro-aging effect that is more pronounced in current smokers than in never and past smokers [18, 19]. Effects of smoking on oxidative stress may also cause inflammation, which is associated with many chronic diseases. There is growing evidence for increase in pro-inflammatory cytokines and decrease in anti-inflammatory cytokines from smoking [20], supporting long-term results of a worse health status in ever smokers than in never smokers.

The strengths of the present study include the population-based approach and the relatively large sample size, which gives robust estimates. One limitation was the cross-sectional study design, so that the causes and effects of smoking could not be differentiated from each other. Another limitation was the lack of information concerning comorbidity, which would have enhanced our understanding if associations between smoking and comorbidity had been studied. Unfortunately,

the questionnaire used for information in this study did not include comorbidities why this issue needs to be further studied. Neither did it distinguish between cigarette, cigar, and pipe smoking which could further illuminate the understanding of smoking habits. Given the general health implications of smoking, referral to smoking cessation programs is important but health-care professionals also need more knowledge concerning disease-related facts and associated risks to support smoking cessation recommendations in the clinic.

In conclusion, patients with PsA who were ever smokers reported worse health status and more pain and fatigue than never smokers did. Besides being a possible result of a worse PsA in ever smokers, impaired health status could also be an effect of unstudied comorbidities. Longitudinal studies will be needed to gain a better understanding of cause and effect. However, smoking cessation should be recommended by health-care professionals not only for general health reasons but also because of disease-specific considerations.

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Conflict of interest The authors have no disclosures to declare.

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