Original article

Differences in body mass index among individuals with PsA, psoriasis, RA and the general population

Vidula M. Bhole¹, Hyon K. Choi^{1,2}, Lindsay C. Burns^{1,3}, Cristián Vera Kellet⁴, Diane V. Lacaille 1,5, Dafna D. Gladman and Jan P. Dutz 3

Abstract

Objectives. To compare obesity among individuals with PsA, psoriasis (PsO), RA and the general population (n), and identify correlates of obesity among individuals with PsO and PsA.

Methods. We compared the BMI of patients with PsA (n = 644). PsO (n = 448). RA (n = 350) and the general population using age- and sex-adjusted linear and logistic regression analyses. We conducted multivariate analyses limited to PsO and PsA to determine correlates of BMI and obesity.

Results. The mean BMI (kilogram per square metre) for individuals with PsA, PsO, RA and the general population were 29.6, 27.9, 27.3 and 26.1, respectively. The proportion with obesity was 37, 29, 27 and 18% for individuals with PsA, PsO, RA and the general population, respectively. The differences in BMI were significant between all categories (P < 0.05) except between PsO and RA. Ageand sex-adjusted linear and logistic regression confirmed that these differences were significant. In multivariate logistic regression analyses adjusted for age, sex, smoking, PsO duration, psoriasis area severity index score, use of DMARDs, glucocorticoids and biologics, the odds of obesity were 61% higher for PsA patients than PsO patients (95% CI 1.10, 2.37). When we additionally adjusted for the physical component summary of the short form-36, the association was attenuated and became insignificant.

Conclusions. Individuals with PsA have a higher mean BMI than those with PsO, RA or the general population. The BMI difference between PsA and PsO correlates with physical health.

Key words: psoriasis, psoriatic arthritis, rheumatoid arthritis, body mass index, obesity.

Introduction

Psoriasis (PsO) is a chronic, inflammatory, autoimmune disease of the skin that affects 2-3% of the population

> linked with an increased risk of PsO (inflammatory skin condition) [3] and RA (inflammatory arthritis) [4]. The underlying hypothesis is that the increased inflammation [5] associated with obesity could contribute to the risk and severity of these inflammatory conditions. If this hypothesis holds true, one could expect the link with PsA to be even stronger, as the condition has inflamma-

uals with PsO and PsA.

[1]. Up to 40% of individuals with PsO develop PsA [2], which adds substantially to the disease burden for PsO

patients, with frequent joint destruction associated

with cartilage erosions, bone damage and joint fusion. Of several purported risk factors, obesity has been

tory components of both the skin and joints. A recent

study reported that PsA was associated with BMI at the

age of 18 years, but not with current BMI [6]. To further

address this hypothesis, we compared obesity among in-

dividuals with PsA, PsO, RA and the general population

(n), and identified the correlates of obesity among individ-

¹Arthritis Research Centre of Canada, Vancouver, BC, Canada, ²Section of Rheumatology and the Clinical Epidemiology Unit, Boston University of School of Medicine, Boston, MA, USA, ³Department of Dermatology and Skin Science, University of British Columbia, Vancouver, BC, Canada, ⁴Department of Dermatology, Pontificia Universidad Católica de Chile, Santiago, Chile, ⁵Department of Medicine, Division of Rheumatology, University of British Columbia, Vancouver, BC and ⁶Department of Medicine, Division of Rheumatology, University of Toronto, Toronto Western Hospital, Toronto, ON, Canada.

Submitted 29 June 2011; revised version accepted 14 September 2011.

Correspondence to: Jan P. Dutz, Skin Care Centre, 835 West 10th Avenue, Vancouver, BC V5Z 4E8, Canada.

E-mail: dutz@interchange.ubc.ca

Methods

Study population

Four hundred and forty-eight PsO and 644 PsA patients were selected from the Toronto and Vancouver sites of the International Psoriasis and Arthritis Research Team (IPART) database. All diagnoses were made by dermatologists and rheumatologists. PsA diagnoses were further confirmed by the CIASsification of Psoriatic ARthritis (CASPAR) criteria [7]. RA data were collected from 350 physician-diagnosed patients enrolled in a longitudinal survey of RA care, identified from administrative health databases of all residents of British Columbia, Canada. Comparison data for the general population were obtained from the 2005 Canadian Community Health Survey (CCHS). Cycle 3.1. The CCHS represents a cyclic, telephone-administered survey conducted by Statistics Canada every 2 years [8]. Analyses were limited to respondents who were ≥18 years of age. Ethical approval for this study was obtained from the University of British Columbia Behavioural Research Ethics Board.

Assessment of variables

BMI was calculated as the subject's weight in kilograms divided by height in metres, squared. BMI was further dichotomized into non-obese ($<30 \, \text{kg/m}^2$) and obese ($>30 \, \text{kg/m}^2$) categories [9]. Data on age, sex and other variables were obtained from the respective databases.

Statistical analysis

We compared BMI among the four groups (PsA, PsO, RA and n) using analysis of variance (ANOVA) and performed all pairwise comparisons using Tukey's test. We performed age- and sex-adjusted linear regression analyses using BMI as a continuous outcome, and logistic regression analyses using obesity as a dichotomous outcome. We conducted multivariate linear as well as logistic regression analyses limited to PsO and PsA groups adjusting for age, sex, smoking, PsO duration, psoriasis

area severity index (PASI) score and use of DMARDs, glucocorticoids and biologics to determine the independent correlates of increased obesity in these patients. To further investigate factors mediating differences in BMI, we evaluated the impact of adding the mental component summary (MCS) and physical component summary (PCS) of the short form-36 (SF-36) guestionnaire, a health-related quality of life measure, into our final model. The MCS is comprised of four psychological components, including vitality, social functioning, emotional role and mental health, whereas the PCS is comprised of four physical health components, including physical functioning, physical role, bodily pain and general health. Higher MCS and PCS values suggest better mental and physical health, respectively. For all point estimates, we calculated 95% Cls. All P-values are two sided. Statistical analyses were conducted using SAS software, version 9.2 (SAS Institute, Cary, NC, USA).

Results

The mean BMIs (kilogram per square metre) for individuals with PsA, PsO, RA and the general population were 29.6, 27.9, 27.3 and 26.1, respectively. The demographics and BMI of the study population according to group are shown in Table 1. The differences in BMI were significant between all categories (ANOVA, P < 0.0001, Tukey's test P < 0.05) except between PsO and RA. Table 2 shows the age- and sex-adjusted differences in BMI and obesity in the PsO, PsA and RA groups compared with the general population. All three groups had significantly higher BMIs and odds of obesity than the general population. Table 3 displays the results of the multivariate logistic regression analysis limited to PsO and PsA patients with complete data. After adjustments for listed co-variables (Model 1), the odds of obesity were 61% higher in PsA than in PsO (95% CI 1.10, 2.37). We further evaluated correlations of the MCS and PCS of the SF-36 questionnaire with BMI in our models. The addition of MCS into our multivariate logistic regression model (Model 2), did not impact the

TABLE 1 Demographics and BMI of the study population

Characteristics	PsA (<i>n</i> = 644)	PsO (n = 448)	RA (<i>n</i> = 350)	General population (<i>n</i> = 115787)
Age, mean (s.b.), years	50 (13)	47 (13)	65 (13)	50 (19)
Sex: female, %	41	42	68	53
BMI, mean (s.d.), kg/m ²	29.6 (7.3)	27.9 (5.7)	27.3 (6.0)	26.1 (5.0)
Obese (BMI \geq 30 kg/m ²), %	37	29	27	18
Smoking (ever), %	44	48	60	-
Arthritis duration, mean (s.d.), years	13.0 (10.0)	-	17.1 (12.8)	-
PsO duration, mean (s.d.)	21.7 (13.5)	16.8 (15.0)	-	-
PASI, mean, median (interquartile range)	5.2, 2.7 (0.9-5.4)	5.9, 4.1 (2.4-6.6)	-	-
PCS, mean (s.p.)	39.1 (12.1)	49.7 (9.6)	-	-
MCS, mean (s.d.)	45.6 (12.1)	47.2 (10.1)	_	-
Biologics, %	19	3	-	-
DMARD use, %	60	12	-	-
Steroid use, %	5	1	_	-

risk of obesity associated with PsA (OR = 1.60, 95% CI 1.09, 2.36). However, when we added PCS to our model (Model 3), the excess risk of obesity associated with PsA was lost [odds ratio (OR) = 1.13, 95% CI 0.74, 1.72] indicating that the PCS was associated with obesity risk (OR = 0.96, 95% CI 0.95, 0.98). Only age and sex remained as significant predictors of obesity. Similar results were obtained with multivariate linear regression analyses.

When we conducted additional analyses using the four individual components of the PCS (physical functioning, role physical, bodily pain and general health) in Model 3

TABLE 2 Age- and sex-adjusted differences in BMI/ obesity in people with RA, PsO and PsA in comparison with the general population

	Age- and sex-adjusted differences (β coefficient) in BMI by linear regression (95% CI)	Age- and sex-adjusted OR of obesity by logistic regression (95% CI)	
General population	0 (Referent)	1.00 (Referent)	
RA	1.03 (0.51, 1.55)	1.62 (1.28, 2.05)	
PsO	1.80 (1.34, 2.26)	1.84 (1.50, 2.26)	
PsA	3.39 (3.00, 3.77)	2.71 (2.31, 3.18)	

instead of the PCS, our results did not change materially. Similarly, the results did not change in separate models using the four MCS scales (vitality, social functioning, emotional role and mental health) instead of the MCS in Model 3.

Discussion

We observed that individuals with PsO, PsA and RA were at greater risk of obesity than the general population, and that individuals with PsA were at markedly increased risk in particular. Individuals with PsA have higher BMIs than those with PsO after adjusting for age, sex, smoking, PsO duration, PASI score, DMARDs, glucocorticoids, biologics and the MCS of the SF-36 health-related quality of life questionnaire. Further adjustment for the PCS of the SF-36 revealed that the PCS correlated with the increased obesity in PsA. Considering the substantially increased risk of cardiovascular disease (CVD) in PsA, obesity and physical activity represent potentially important modifiable risk factors.

In this study, the mean BMI for individuals with PsA was $1.80\,\mathrm{kg/m^2}$ (95% CI 0.61, $2.99\,\mathrm{kg/m^2}$) higher than that for those with PsO, and the odds of obesity were increased by 61% among individuals with PsA compared with PsO (95% CI 1.10, 2.37) after adjustments for co-variables. Previous evidence from cross-sectional [10], case-control [11] and cohort studies [3] suggests that obesity is a significant risk factor for PsO. Recently, a large PsO cohort

Table 3 Multivariate logistic regression analyses of correlates of obesity in people with PsO and PsA

	Multivariate ^a OR of obesity (95% CI)	Multivariate ^b OR of obesity (95% CI)	Multivariate ^c OR of obesity (95% CI)
Diagnosis			
PsO	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
PsA	1.61 (1.10, 2.37)	1.60 (1.09, 2.39)	1.13 (0.74, 1.72)
Sex			
Female	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
Male	0.63 (0.45, 0.88)	0.63 (0.45, 0.89)	0.70 (0.50, 0.99)
Age	1.02 (1.01, 1.04)	1.02 (1.01, 1.04)	1.02 (1.00, 1.03)
Smoking			
No	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
Yes	1.44 (1.04, 2.01)	1.42 (1.02, 1.98)	1.26 (0.89, 1.78)
PsO duration	0.99 (0.98, 1.00)	0.99 (0.97, 1.00)	0.99 (0.98, 1.00)
PASI score	1.03 (1.01, 1.05)	1.03 (1.01, 1.05)	1.02 (0.99, 1.05)
DMARD use			
No	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
Yes	0.99 (0.67, 1.45)	0.98 (0.67, 1.44)	0.92 (0.62, 1.36)
Glucocorticoid use	e		
No	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
Yes	1.26 (0.51, 3.11)	1.21 (0.49, 3.00)	0.99 (0.39, 2.51)
Biologics use			
No	1.00 (Referent)	1.00 (Referent)	1.00 (Referent)
Yes	1.14 (0.69, 1.89)	1.13 (0.68, 1.87)	1.11 (0.66, 1.86)
MCS		0.99 (0.98, 1.01)	0.99 (0.98, 1.02)
PCS			0.96 (0.95, 0.98)

^aAdjusted for diagnosis, age, sex, smoking, PsO duration, PASI score, DMARD use, glucocorticoid use and biologics use. ^bAdjusted for MCS in addition to all of the above. ^cAdjusted for PCS in addition to all of the above.

study [6] indicated that obesity increases the risk of PsA as well. Our results support these observations. Obesity may increase the risk for PsA by modulating inflammation or may increase the risk of joint damage and subsequent inflammation due to increased joint loading. Alternatively, pain and inflammation can lead to reduced physical activity in arthritis patients [12], which in turn may increase the risk of obesity. Whether PsA specifically promotes obesity through decreased physical functioning or whether obesity promotes PsA (as suggested by prior cohort studies) needs further prospective investigations.

BMI increases with age in adulthood, until the age of 60-75 years, and the prevalence of obesity or extreme obesity is the highest in women below the age of 60 [13]. Our study was consistent with these findings as increasing age and female gender were observed risk factors for obesity in PsO and PsA patients.

Our study did not confirm previous reports of rheumatoid cachexia, or low body mass among RA patients [14], potentially due to improved treatment regimens in RA. Patients with PsA had higher rates of obesity than patients with RA, possibly pointing out metabolic differences between these diseases. The present finding that the mean BMI in PsA is $2.3\,\mathrm{kg/m^2}$ higher than in a comparative RA population (29.6 vs $27.3\,\mathrm{kg/m^2}$) is consistent with findings from a large US registry (32.1 vs $29.8\,\mathrm{kg/m^2}$, P < 0.001) [15].

Chronic inflammation, such as that noted in psoriatic conditions, has been recognized as a risk factor for CVD [16, 17]. For example, extent of skin disease (as determined by skin score) in patients with PsA correlates with cardiovascular risk [18]. Furthermore, obesity has also been associated with chronic inflammation and cardiovascular risk [5, 19, 20]. Given the substantially increased risk of CVD in PsA, obesity and physical activity represent potentially impactful modifiable risk factors.

The diagnostic accuracy and uniformity in data capture for the large sample of patients with PsO and PsA are unique strengths of this study. Physician-diagnosed RA cases were identified using diagnostic codes from province-wide administrative billing databases, and the definition was made further specific by including only those with two RA codes at least 2 months apart, to remove those with transient inflammatory arthritis [21]. The height and weight of PsO and PsA patients were ascertained objectively, avoiding recall bias. In lieu of raw physical or psychiatric assessment data, we have used PCS and MCS to measure physical and mental dimensions of health, respectively. With regard to disease-specific comparisons (RA vs PsA), we cannot rule out the possibility of selection bias, as the individuals with RA were identified from a different cohort than the PsA and PsO patients. Such a potential selection bias would not affect the comparisons between PsA and PsO.

In conclusion, the present study demonstrates that BMI is higher in PsA (skin and joint disease) than that in PsO (skin disease), RA (joint disease) or the general population. Furthermore, our results suggest that the BMI difference between PsA and PsO is independent of other risk factors

of obesity but correlates with the PCS of the SF-36. These findings highlight for clinicians the need to control obesity in PsA patients through regular non-weight-bearing physical exercises. Studies of the impact of physical activity on BMI and CVD among these populations are urgently needed.

Rheumatology key messages

- PsA patients have higher BMIs than those with PsO, RA or the general population.
- The BMI difference between PsA and PsO correlates with physical health.
- PsA patients should be urged to control obesity through regular non-weight-bearing physical exercises.

Acknowledgements

The authors thank all participants in the study. We would also thank Ms Renise Ayearst for her help in data extraction. J.P.D. was supported by a Senior Scientist Award of the Child and Family Research Institute and is a Senior Scholar of the Michael Smith Foundation.

Funding: This study was funded in part by a CIHR New Emerging Team Grant in Clinical Autoimmunity, awarded to D.D.G.

Disclosure statement: The authors have declared no conflicts of interest.

References

- 1 Nestle FO, Kaplan DH, Barker J. Psoriasis. N Engl J Med 2009;361:496–509.
- 2 Gelfand JM, Gladman DD, Mease PJ et al. Epidemiology of psoriatic arthritis in the population of the United States. J Am Acad Dermatol 2005;53:573-7.
- 3 Setty AR, Curhan G, Choi HK. Obesity, waist circumference, weight change, and the risk of psoriasis in women: nurses' Health Study II. Arch Intern Med 2007;167:1670-5.
- 4 Symmons DPM, Bankhead CR, Harrison BJ et al. Blood transfusion, smoking, and obesity as risk factors for the development of rheumatoid arthritis. Results from a primary care based incident case control study in Norfolk, England. Arthritis Rheum 1997;40:1955-61.
- 5 Hamminga EA, van der Lely AJ, Neumann HAM et al. Chronic inflammation in psoriasis and obesity: implications for therapy. Med Hypotheses 2006;67: 768-73.
- 6 Soltani-Arabshahi R, Wong B, Feng B-J et al. Obesity in early adulthood as a risk factor for psoriatic arthritis. Arch Dermatol 2010;146:721-6.
- 7 Taylor W, Gladman D, Helliwell P et al. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum 2006;54: 2665–73.

- 8 Statistics Canada. Canadian Community Health Survey (CCHS) Cycle 3.1 (2005) Public Use Microdata File (PUMF) User Guide. Ottawa: Statistics Canada, 2006.
- 9 Anonymous. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organization Technical Report Series 894. Geneva: WHO, 2000; i-xii.
- 10 Herron MD, Hinckley M, Hoffman MS et al. Impact of obesity and smoking on psoriasis presentation and management. Arch Dermatol 2005;141:1527–34.
- 11 Naldi L, Chatenoud L, Linder D et al. Cigarette smoking, body mass index, and stressful life events as risk factors for psoriasis: results from an Italian case-control study. J Investigat Dermatol 2005;125:61-7.
- 12 Hootman JM, Macera CA, Ham SA et al. Physical activity levels among the general US adult population and in adults with and without arthritis. Arthritis Care Res 2003; 49:129-35.
- 13 Wang Y, Beydoun MA. The obesity epidemic in the United States-gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. Epidemiol Rev 2007;29: 6-28.
- 14 Munro R, Capell H. Prevalence of low body mass in rheumatoid arthritis: association with the acute phase response. Ann Rheum Dis 1997;56:326-9.

- 15 Reddy SM, Anandarajah AP, Fisher MC et al. Comparative analysis of disease activity measures, use of biologic agents, body mass index, radiographic features, and bone density in psoriatic arthritis and rheumatoid arthritis patients followed in a large US disease registry. J Rheumatol 2010;37:2566-72.
- 16 Boehncke W-H, Boehncke S, Tobin A-M et al. The 'psoriatic march': a concept of how severe psoriasis may drive cardiovascular comorbidity. Exp Dermatol 2011;20: 303-7
- 17 Greenberg JD, Kremer JM, Curtis JR et al. Tumour necrosis factor antagonist use and associated risk reduction of cardiovascular events among patients with rheumatoid arthritis. Ann Rheum Dis 2011;70: 576–82.
- 18 Gladman DD, Ang M, Su L et al. Cardiovascular morbidity in psoriatic arthritis. Ann Rheum Dis 2009;68:1131–5.
- 19 Hubert HB, Feinleib M, McNamara PM *et al.* Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation 1983;67:968–77.
- 20 Wellen KE, Hotamisligil GS. Inflammation, stress, and diabetes. J Clin Invest 2005;115:1111-9.
- 21 Lacaille D, Anis AH, Guh DP *et al.* Gaps in care for rheumatoid arthritis: a population study. Arthritis Care Res 2005;53:241–8.