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High prevalence of subclinical atherosclerosis in psoriatic arthritis patients: a study based on carotid ultrasound

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Abstract Analyse the presence of subclinical atherosclerosis in psoriatic arthritis patients (PsA). A cross-sectional study of 53 patients with PsA and 53 controls matched for age and sex was designed. Carotid intima-media thickness (IMT) and the presence of carotid plaques (CP) were assessed with carotid ultrasound. Data on cardiovascular (CV) risk factors were collected. Patients with PsA had a higher prevalence rate of obesity and tobacco smoking. CP were detected more frequently in patients with PsA than in controls with an OR of 4.15, 95% CI 1.4–12.1, which adjusted for smoking and those with history of CV disease gave an OR of 3.9, 95% CI 1.2–12.7, $p = 0.026$. Carotid IMT was significantly higher in patients with PsA adjusted for age and tobacco smoking. According to ultrasound data, 30.2% of patients with PsA had carotid atherosclerosis (presence of CP and/or carotid IMT > 0.90 mm) compared with 9.4% of controls. The SCORE index (Systematic Coronary Risk Evaluation) underestimated the CV risk in these patients: most patients with CP had an intermediate CV risk. According to carotid ultrasound data, PsA patients have a high prevalence of subclinical atherosclerosis. These results support the importance of screening for CV risk and to include carotid ultrasound in CV prevention strategies in these patients.

Keywords Psoriatic arthritis · Cardiovascular disease · Carotid ultrasound

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Introduction

The association between inflammation and atherosclerosis is widely known. An increase in morbidity and mortality due to cardiovascular (CV) disease in inflammatory rheumatic diseases has been proved [1–4]. Rheumatoid arthritis (RA) has the greatest CV impact. Scientific societies and expert groups have developed recommendations for preventing cardiovascular risk in these patients [5, 6]. It has also been observed an increased CV risk and a greater morbidity in other inflammatory rheumatic diseases such as PsA [1, 7, 8]. It is known that patients with psoriasis or with PsA have a high prevalence of CV risk factors [9, 10], specially those in the metabolic syndrome group. However, the increased CV morbidity and mortality in these patients is not completely explained by the increased frequency of these risk factors. One study [8] showed that psoriatic arthritis patients without risk factors and without cardiovascular disease clinically evident had a high prevalence of macrovascular disease, which was seen as increased carotid artery. Probably the inflammatory burden associated with the disease added to classic CV risk factors is responsible for the increased cardiovascular risk in PsA [1].

CV prevention strategies in patients with rheumatic diseases have focused on the early diagnosis of atherosclerosis. Several studies have shown the existence of early arterial endothelial disorders that can be detected by carotid ultrasound and arterial elasticity techniques that can detect atherosclerosis at a subclinical stage. The presence of carotid plaques and increased carotid intima-media thickness (IMT) greater than 0.90 mm are considered subclinical atherosclerosis [3, 8, 11]. Several case–control studies and a meta-analysis [3, 8, 11–15] have shown that patients with PsA have carotid plaques more frequently and greater carotid intima-media thickness (IMT) than control patients.

The purpose of this study is to compare the presence of subclinical atherosclerosis based on the results of carotid ultrasound in a group of patients with PsA and a control group.

Method

Study design and data collection

A cross-sectional study with PsA patients and age- and sex-matched controls was designed. PsA patients were enrolled among those receiving outpatient rheumatology care services between July and December 2015. Controls were selected among patients from rheumatology and other specialties outpatient clinics without any inflammatory disease or psoriasis. A difference of 5 years between case and control was allowed when matching by age. All patients and controls were asked for their informed consent.

The following medical history data were collected: age, sex, diagnosis of high blood pressure (HBP), diabetes mellitus (DM), dyslipidemia, obesity if body mass index (BMI) more than 30, systolic blood pressure, current or previous smoking habits, personal or family history of CV disease (ischaemic heart disease or stroke), C-reactive protein (CRP), total cholesterol, high-density lipoprotein (HDL), lipid-lowering therapy, antihypertensive steroids, disease modifying antirheumatic drugs (DMARDs) or biologic therapy and MHAQ (modified health assessment questionnaire). The SCORE index (Systematic Coronary Risk Evaluation) was calculated with the HDL/total cholesterol ratio [16]. Hyperlipidemia was defined if total cholesterol was over 200 mg/dl at the time of the study.

Carotid ultrasound

A carotid ultrasound using MyLab Class C ultrasound machine by Esaote was done.

Both carotid arteries were studied in the longitudinal and transverse axis searching for carotid plaques. The IMT was measured using the IMT tools software. Carotid plaque was defined by the Manheim consensus [17] as a focal structure that encroaches into the arterial lumen of at least 0.5 mm or 50% of the surrounding IMT value or demonstrates a thickness of $\geq$1.5 mm as measured from the media–adventitia interface to the intima–lumen interface.

Statistical analysis

The description of quantitative variables was performed by means and standard deviations (SD) or medians and interquartile ranges (IQR), depending on the nature of the variables. The description of categorical variables was made by frequencies and percentages. Comparison of these characteristics between groups was performed using the Student's t test or the Mann–Whitney test for the quantitative statistics and the Chi-square or the Fisher's test for qualitative variables, according to compliance with the requirements in each case. The magnitude of the association between the presence of carotid plaques and PsA was quantified by binary logistic regression models including CV risk factors that provided the estimate of the odds ratios (OR), both crude and adjusted. Additionally, the magnitude of the relationship between increased carotid IMT and PsA was quantified by comparing means and the general linear model to adjust for CV risk factors. The correlation between IMT and age or SCORE index was done with Spearman's rho test. Analyses were performed in SPSS version 20.

Results

Baseline characteristics

Fifty-three PsA patients and 53 controls matched for age and sex were included. Table 1 summarises the patients' characteristics and carotid ultrasound results.

Carotid ultrasound

Carotid plaques

Carotid plaques were detected in 16/53 patients with PsA and 5/53 of controls, which provided a significant association ($p = 0.011$) between the presence of carotid plaques and PsA, OR 4.15 (95% CI 1.4–12.1). Additionally, the presence of carotid plaques was significantly associated with active or past smoking habits, compared with those who had never smoked, and with the presence of past CV event (Table 2). The risk of carotid plaques for PsA, adjusted for CV risk factors that showed to be significant in the univariate analysis (smoking and history of CV disease), was OR 3.9, 95% CI 1.2–12.7; $p = 0.026$.

Carotid IMT

The carotid IMT was higher in patients with PsA compared to controls (Table 1). The association of carotid IMT with different CV risk factors is shown in Table 3. Multivariate analysis showed a significantly greater age and smoking-adjusted carotid IMT in PsA (Table 3). A significant increase in carotid IMT was found with age ($p = 0.001$),

Table 1 Characteristics of patients and controls

	All ($N = 106$)	PsA ($N = 53$)	Controls ($N = 53$)	p value
Age, mean (SD) (years)	56.3 (13.2)	56.2 (13.5)	56.4 (13.1)	ns
Sex, male, *n* (%)	64 (60.4)	64 (60.4)	64 (60.4)	ns
Carotid plaques, *n* (%)	21 (19.8)	16 (30.2)	5 (9.4)	0.013
Carotid IMT, mean (SD) (μm)	662.3 (152.7)	696.0 (149.9)	628.6 (149.3)	0.022
SCORE median (IQR)	2 (0–3)	2 (0–3)	2 (0–2.5)	ns
SBP, median (IQR) (mmHg)	130 (110–138)	130 (120–140)	130 (110–135)	ns
Hyperlipemia, *n* (%)	53 (50)	26 (49.5)	27 (50.9)	ns
Total cholesterol, mean (SD) (mg/dl)	202.6 (42.9)	197.2 (42.1)	207.9 (43.4)	ns
HDL/total cholesterol ratio, median (IQR)	3.8 (3.3–4.9)	4 (3.2–5.0)	3.8 (3.3–4.7)	ns
Uricemia, median (IQR) (mg/dl)	5 (4.1–6.2)	5.5 (4.2–6.3)	4.9 (3.8–5.8)	ns
CRP, median (IQR) (mg/l)	1.3 (0.8–4.2)	3.3 (1.1–6.5)	1 (0.5–1.75)	0.001
Hypertension, *n* (%)	42 (39.6)	24 (45.3)	18 (34.0)	ns
Diabetes, *n* (%)	7 (6)	5 (9.4)	2 (3.8)	ns
Obesity, *n* (%)	33 (31.1)	24 (45.3)	9 (17.0)	0.002
Current smokers, *n* (%)	22 (20.8)	14 (26.4)	8 (15.1)	ns
Former smokers, *n* (%)	40 (37.7)	24 (45.3)	16 (30.2)	ns
Never smoker, *n* (%)	44 (47.5)	15 (28.3)	29 (54.7)	0.021
Current/former smoker, *n* (%)	62 (58.5)	38 (71.7)	24 (45.3)	0.005
Lipid-lowering agents, *n* (%)	22 (20.8)	11 (20.8)	11 (20.8)	ns
Corticosteroids, *n* (%)	22 (20.8)	20 (37.7)	2 (3.8)	<0.001
NSAIDS, *n* (%)	41 (38.7)	31 (58.5)	10 (18.9)	<0.001
M-HAQ, median (IQR)	0 (0–0.2)	0.13 (0–0.4)	0 (0–0.1)	0.005
History of CV events, *n* (%)	11 (10.4)	6 (11.3)	5 (9.4)	ns
Family history of CV events, *n* (%)	43 (40.6)	19 (35.8)	24 (45.3)	ns

Hyperlipidemia was defined as total cholesterol >200 mg/dl at the time of study

PsA psoriatic arthritis, *IMT* intima-media thickness, *SCORE* Systematic Coronary Risk Evaluation algorithm, *SBP* systolic blood pressure, *HDL* high-density lipoprotein, *CRP* C-reactive protein, *NSAIDs* non-steroidal anti-inflammatory drugs, *M-HAQ* Modified Health Assessment Questionnaire, *CV* cardiovascular, *IQR* interquartile range, *SD* standard deviation

this increase being higher in patients with PsA compared to controls ($p = 0.010$).

The correlation between age and carotid IMT was good in the control group (Spearman's rho 0.572) and moderate in patients with PsA (Spearman's rho 0.380), $p = 0.005$.

There was good correlation between SCORE and carotid IMT (Spearman's rho 0.480; $p = 0.001$). The increase in carotid IMT according to the SCORE was higher in PsA patients than in controls ($p = 0.004$).

Carotid IMT greater than 0.90 mm was detected in six subjects: four cases and two controls. Carotid plaques were detected in three of the four cases and none of the controls ($p = 0.09$).

According to carotid ultrasound, the 30.2% of patients with PsA had atherosclerosis (presence of carotid plaques and/or carotid IMT greater than 0.9 mm) compared to 9.4% of controls $p = 0.007$. Excluding subjects with past CV events, 22.6% of cases and 5.6% of controls had subclinical atherosclerosis, $p = 0.02$.

SCORE index

The SCORE index was zero (no CV risk in 10 years) in 18.9% of cases and 24.5% of controls. It was between 1 and 5 (intermediate risk) in 62.3% of patients and 56.7% of controls, between 5 and 9 (high risk) in 18.9% of cases and 7.5% of controls. SCORE index was greater than 10 (very high risk) in seven subjects (7.4%), all of them were controls, one of which had carotid plaques. Among the 21 patients with carotid plaques, 17 had a SCORE between 1 and 5, three between 5 and 9 and in one subject the SCORE was above 10.

Discussion

The main finding of this study is the increased risk of carotid plaques and greater carotid IMT in patients with PsA compared with controls. Our findings are similar to

Table 2 Risk of carotid plaque. Univariate and multivariate logistic regression (below) for PsA and other cardiovascular risk factors

	OR (95% CI)	*p* value
Variable group		
PsA	4.15 (1.39–12.37)	0.011
Socio-demographic variables		
Age	1.02 (0.98–1.06)	0.251
Sex (M compared with F)	2.47 (0.83–7.35)	0.105
CV risk factors		
Tobacco	3.78 (1.17–12.17)	0.026
Hyperlipidemia	2.36 (0.86–6.43)	0.093
Obesity	1.48 (0.54–4.00)	0.442
Hypertension	1.51 (0.57–3.94)	0.405
Diabetes	3.37 (0.69–16.41)	0.138
History of CV events	6.40 (1.73–23.7)	0.005
Family history of CV events	1.82 (0.67–4.76)	0.227
Corticosteroids	2.33 (0.80–6.77)	0.136
NSAIDS	1.58 (0.60–4.15)	0.454
SCORE	1.09 (0.96–1.27)	0.174
Multivariate model result		
PsA	3.87 (1.18–12.74)	0.026
History of CV events	6.30 (1.51–26.23)	0.011
Tobacco	2.44 (0.70–8.48)	0.162

PsA psoriatic arthritis, *SCORE* Systematic Coronary Risk Evaluation algorithm, *NSAIDs* nonsteroidal anti-inflammatory drugs, *CV* cardiovascular, *M* male, *F* female, *OR* odds ratio, *CI* confidence interval

Table 3 Univariate and multivariate linear regression (below) of the carotid IMT for PsA and other cardiovascular risk factors

	β^{a} (95% CI)	*p* value
Variable group		
PsA	67.4 (9.8 to 125.0)	0.022
Socio-demographic variables		
Age	15.1 (3.1 to 7.1)	<0.001
Sex (M compared with F)	57.1 (−2.2 to 116.5)	0.059
CV risk factors		
Tobacco	90.6 (33.3 to 147.9)	<0.001
Hyperlipemia	9.96 (−49.1 to 69.0)	0.739
Obesity	59.9 (−2.8 to 122)	0.061
Hypertension	73.5 (14.8 to 132.2)	0.015
Diabetes	179 (66.2 to 193.6)	0.002
History of CV	97.1 (2.1 to 192.1)	0.045
Family history of CV	23.5 (−36.5 to 80.5)	0.438
Corticosteroids	27.4 (−45.3 to 100.0)	0.457
NSAIDS	22.5 (−37.9 to 83.1)	0.462
SCORE	15.9 (7.4 to 24.3)	<0.001
Multivariate model result		
PsA	53.1 (0.4 to 105.6)	0.048
Age	4.8 (2.9 to 6.8)	<0.001
Tobacco	58.5 (4.6 to 112.4)	0.034

PsA psoriatic arthritis, *IMT* intima-media thickness, *M* male, *F* female, *SCORE* Systematic Coronary Risk Evaluation algorithm, *NSAIDs* nonsteroidal anti-inflammatory drugs, *CV* cardiovascular, *CI* interval of confidence

[a] The estimated β value represents difference in the mean IMT, expressed in μm, for patients with and without the various characteristics with confidence intervals and, in the multivariate model, the adjusted result

those made by other authors supporting the evidence of PsA as an independent CV risk factor.

It was found an increased prevalence of obesity and smoking among patients with PsA, which has been previously reported [18, 19].

There was an increased risk of carotid plaques in PsA patients. The magnitude of this risk (OR 3.8, 95% CI 1.2–12.5) is similar to the one described in a meta-analysis [12] which includes 898 PsA patients and 1140 controls (OR 3.12, 95% CI 1.03–9.39).

In the univariate analysis for carotid IMT the factor that had the greatest weight was DM, followed by previous CV event, smoking and HBP (Table 3).

The presence of PsA increased carotid IMT by 64 μm on average, which, adjusted for age and smoking, provided an average increase in carotid IMT of 53.1 μm (0.4–105.6) $p = 0.048$. A study [20] found that a difference of 100 μm in carotid IMT increased the risk of a significant CV event by 13–18% and the presence of nonstenosing carotid plaques by 10–61%.

In our study, current or previous smoking habits were associated with the presence of carotid plaques. It is known that smoking is an independent CV risk factor and that is more prevalent in patients with PsA. In addition, PsA patients who smoke get worse and have poorer response to anti-TNF and lower adherence to treatment [19]; therefore, a greater inflammatory burden is maintained. Smoking could be the most important predictor of atherosclerosis in patients with PsA. However, in our study a higher frequency of carotid plaques and a greater carotid IMT in patients with PsA adjusted for smoking and age supports that inflammatory disease is an independent CV risk factor.

DM is the most significant CV risk factor known in the general population. In this study carotid IMT in diabetics was significantly higher. These results agree with other studies [18, 21] that found the main predictors of CV events in PsA patients were diabetes, the number of dactylitis, polyarticular onset and ESR.

CV disease is associated with age. We found an increase in carotid IMT with age that was higher in patients with PsA compared to controls. This finding supports that there are additional CV risk factors in these patients, probably the inflammation.

Despite the increased frequency of carotid atheroma in patients with PsA, the prevalence of past CV events was the same in both groups. This may be because 45% of patients were on biological treatment, which is known to reduce cardiovascular risk probably by decreasing the inflammatory burden.

We found a significant association of carotid IMT with the SCORE. This supports the SCORE's usefulness as a screening for atherosclerosis in patients with PsA. However, it has been reported [9, 22, 23] an underestimation of cardiovascular events in patients with PsA predicted by the modified SCORE recommended by EULAR for patients with RA and by the Framingham SCORE. In a study [24] that evaluated CV risk in PsA patients without risk factors or atherosclerosis, also detected an underestimation of risk with the SCORE and suggested a modification including disease duration and CRP. However, another study showed that the greater IMT in psoriatic arthritis patients was independently correlated with parameters of disease activity and conventional risk factors for atherosclerosis [25].

Our study supports the underestimation of CV risk by the SCORE index. Most patients with carotid plaques had an intermediate risk (SCORE between 1 and 5%).

A modification of the SCORE would probably be necessary. It could include a multiplier factor, as in the case of RA, or to consider other risk factors not included in the SCORE, such as obesity or inflammation (number of swollen joints, number of dactylitis, CRP or ESR).

In clinical practice, CV risk screening should be carried out with a SCORE adapted to patients with PsA and, for those with an intermediate risk, a carotid ultrasound study should be performed. In this study, the 30.2% of patients had atherosclerosis compared with 9.4% of controls $p = 0.007$, just with the ultrasound study. This finding alone would support the usefulness of carotid ultrasound to screen for CV disease in PsA patients.

The main limitation of our study is its sample size which is not enough to provide statistical power to adjust for all the known CV risk factors. Its strength is to support the evidence of subclinical atherosclerosis in patients with PsA and the importance of screening for CV disease in these patients.

In conclusion, this study supports the evidence of increased subclinical atherosclerosis in patients with PsA compared to people without inflammatory disease. It also suggests an underestimation of CV disease with the SCORE index and proves the usefulness of carotid ultrasound as a screening method for cardiovascular disease in PsA patients.

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Compliance with ethical standards

Conflict of interest Rosario Ibáñez has received speak fees from Abvi, BMS and funding for congresses assistance from Roche and Pfizer. Eduardo Loza has received speak fees from Roche, Abvi, and BMS and funding for congresses from MSD and Pfizer. The rest of authors declared no conflicts of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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