

Psoriatic patients have an increased risk of polycystic ovary syndrome: results of a cross-sectional analysis

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Objective: To define the prevalence and the features of polycystic ovary syndrome (PCOS) in patients with psoriasis. To our knowledge, the association between PCOS and psoriasis has not been explored in previous studies. Psoriasis is linked with metabolic syndrome, insulin resistance, and non-alcoholic fatty liver disease, which are features often associated with PCOS.

Design: A cross-sectional analysis was performed between January 2010 and April 2012.

Setting: Unit of human reproductive pathophysiology, Catholic University Hospital.

Patient(s): We prospectively analyzed 51 patients with psoriasis and 102 healthy age- and body mass index (BMI)-matched controls.

Intervention(s): None.

Main Outcome Measure(s): The prevalence and characteristics of PCOS women of reproductive age with chronic plaque psoriasis.

Result(s): The prevalence of PCOS was greater in patients with psoriasis than in matched control subjects (47.05% and 11.76%, respectively; odds ratio, 6.66; 95% confidence interval 2.95–15.07). Among the women with psoriasis, the prevalence of Psoriasis Area and Severity Index ≥ 10 was higher in patients with PCOS than in subjects without PCOS (odds ratio, 3.5; 95% confidence interval 1.04–11.72).

Conclusion(s): The prevalence of PCOS in women with psoriasis is remarkably greater than in age- and BMI-matched control women. (Fertil Steril® 2013;99:936–42. ©2013 by American Society for Reproductive Medicine.)

Key Words: Polycystic ovary syndrome, psoriasis, prevalence

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Psoriasis is a chronic, relapsing inflammatory skin disorder affecting 2%–3% of the world population and characterized by erythematous and scaly plaques, typically at the extensor surfaces of the extremities, which may be stable for a long time, or progress to involve larger areas of the body. Histopathologically, these lesions are characterized by epi-

dermal hyperproliferation, increased dermal angiogenesis, and an inflammatory dermal infiltrate, consisting predominantly of T cells (1). Although its etiology is unknown, evolving data suggest that psoriasis is a complex disorder caused by the interaction between multiple predisposing genes, the immune system, and environmental factors.

Evidence showed an association between psoriasis and obesity, insulin resistance (IR) (2) and metabolic syndrome (MS) (3–7). In particular, compared with healthy subjects, women with psoriasis show a higher prevalence of IR and an increased incidence of cardiovascular disease (CVD) (2, 8). Recent data also linked psoriasis to non-alcoholic fatty liver disease (9, 10), hepatic disorder including a spectrum of conditions ranging from simple steatosis to steatohepatitis and cirrhosis (11).

Non-alcoholic fatty liver disease, IR, and MS are also associated with polycystic ovary syndrome (PCOS), one of the most common endocrine disorders affecting 6%–15% of reproductive-age women (12). In this regard, our recent data demonstrated

Received July 17, 2012; revised October 18, 2012; accepted October 21, 2012; published online November 16, 2012.

F.M. has nothing to disclose. C.D.S. has nothing to disclose. A.M. has nothing to disclose. A.T. has nothing to disclose. F.S. has nothing to disclose. C.P. has nothing to disclose. E.S. has nothing to disclose. A.T. has nothing to disclose. G.C. has nothing to disclose. M.D. has nothing to disclose. S.M. has nothing to disclose. A.L. has nothing to disclose. R.A. has nothing to disclose.

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Fertility and Sterility® Vol. 99, No. 3, March 1, 2013 0015-0282/\$36.00

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that non-alcoholic fatty liver disease was diagnosed in 58% of overweight hyperinsulinemic patients with PCOS and 32% of them also showed MS (13).

A PCOS diagnosis is performed in the presence of hyperandrogenism, morphologic changes in the ovary (polycystic), and chronic anovulation, according to Rotterdam criteria (14, 15). This syndrome is an heterogeneous condition characterized by several symptoms and clinical signs related to reproductive, metabolic, and cardiovascular disorders (15, 16). In particular, IR with accompanying compensatory hyperinsulinemia seems to play a pathophysiological role in the development of this syndrome (17). It is estimated that a significant proportion of patients with PCOS (50%–60%) also present with IR (18, 19) and that the prevalence of MS in women with PCOS is about 24% (20). Considering that IR, MS, and non-alcoholic fatty liver disease are common features of psoriasis as well as PCOS, it is likely that both entities may coexist within the same patient. Thus far, the possible link between psoriasis and PCOS has not been explored.

Starting from these observations, we conducted a cross-sectional analysis to determine the prevalence and the features of PCOS in patients with psoriasis.

MATERIALS AND METHODS

Study Design

The present study was performed in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology guidelines (21). This protocol of this study was approved by the institutional review board of our Institute at the Policlinico Gemelli, Rome. Informed consent was obtained from all participants before entering into the study.

Study Population

To determine the prevalence and characteristics of PCOS in women with psoriasis, we recruited 62 consecutive reproductive aged patients with chronic plaque psoriasis who attended to the Dermatology Outpatient Clinic between January 2010 and February 2011. To compare the prevalence of PCOS in patients without psoriasis, we prospectively selected 107 consecutive healthy age- and body mass index (BMI)-matched controls from the Gynaecological Outpatient Clinic (for Human Papillomavirus Screening and found with negative viral screening results) between March 2011 and April 2012.

All women enrolled in this study were examined by two dermatologists, who confirmed the diagnosis of chronic plaque psoriasis and of psoriatic arthritis, in accordance with Caspar criteria (22), in the dermatologic patients. The same dermatologists also examined gynecological patients with the aim to rule out subjects with a diagnosis of psoriasis. In case of disagreement between the two dermatologists about the diagnosis of psoriasis and psoriatic arthritis, a skin sample and a rheumatologic referral were performed to obtain a definite diagnosis. The dermatologists also independently calculated the patient's Psoriasis Area and Severity Index (PASI) as well as the Physician's Global Assessment (PGA). The average of the two scores by the dermatologists was used for analysis.

The PASI is a composite score ranging from 0–72 and reflects the extent, erythema, induration, and scaliness of the lesions in four body areas (head, trunk, arms, and legs) (23). A PASI score ≥ 10 was correlated with common indicators of severe disease (e.g., need of systemic therapy, hospitalization) (24). The PGA score is a seven-point ordinal scale used to assess the global severity of disease on the body as a whole. The following categories were used: clear, almost clear, mild, mild-to-moderate, moderate, moderate-to-severe, severe. For statistical analysis, the scale was assigned scores of 0–6, from clear to severe (25).

Polycystic ovary syndrome was diagnosed on the basis of the presence of chronic oligomenorrhea, biochemical hyperandrogenism/clinical hirsutism, and polycystic ovary (PCO) on ultrasound according to Rotterdam criteria (14).

The exclusion criteria were: age <18 or >40 years, diabetes mellitus, other hormonal dysfunction (hypothalamic, pituitary, thyroidal, or adrenal causes), treatment with clomiphene citrate (CC), oral contraceptives (OC), antiandrogens, drugs to control appetite or insulin-sensitizing drugs (metformin, pioglitazone, and rosiglitazone) during the past 6 months before our evaluation.

In case of systemic treatment for psoriasis at the time of enrolment, patients were evaluated after a drug wash-out period of 2 months. Topical steroids were admitted at the time of the evaluation only if applied to less than 10% of skin surface and for a limited period time (3–4 weeks).

Anthropometric, Clinical, and Biochemical Variables

Body mass index (in kilograms per meter squared [18]), waist-to-hip ratio, hirsutism, acne, oligomenorrhea, blood pressure, smoking habit, and family history of early coronary artery disease were analyzed in all patients. These anthropometric parameters were evaluated at day 3 of a spontaneous menstrual cycle or after an amenorrhea of >60 days (with a proven anovulation: plasma P <1.5 ng/mL), in the Gynaecological Outpatient Clinic.

For the waist-to-hip ratio measurement, waist circumference was determined as the minimum value between the iliac crest and the lateral costal margin, and the hip circumference was calculated as the maximum value over the buttocks.

Hirsutism was evaluated by the Ferriman-Gallwey map scoring system (hirsutism, >8) (26), and clinical acne was defined by a history of persistent acne (presence of acne on most days for at least 3 years) and presence of more than 10 inflammatory acne lesions (27). Oligomenorrhea was defined by <8 spontaneous menstrual cycles per year for at least 3 years before the first examination.

Hypertension was diagnosed in presence of systolic blood pressure >130 mm Hg and/or diastolic blood pressure >85 mm Hg in three different measures. The family history of early coronary artery disease was defined as a first-degree relative with a history of myocardial infarction at <60 years.

At day 3 of a spontaneous menstrual cycle or after an amenorrhea of >60 days (with a proven anovulation: plasma P <1.5 ng/mL), all patients underwent transvaginal ultrasonography. Ovarian volume was calculated by the following formula: $V = (\pi/6) \times D_{length} \times D_{width} \times D_{thickness}$, where

D denotes the dimension. The PCO morphology was defined by the presence of 12 or more follicles measuring 2–9 mm in diameter in each ovary and/or increased ovarian volume (>10 cm) (14).

On the same day, blood samples were obtained at 8:00 AM after an overnight fast to measure the levels of total T, sex hormone-binding globulin (SHBG), androstenedione (A), 17 α -hydroxyprogesterone (17-OHP), DHEAS, triglycerides, total cholesterol, high-density and low-density lipoprotein cholesterol (HDL and LDL cholesterol), fasting plasma glucose, and fasting plasma insulin.

The free androgen index was calculated by following formula: $T \times 100/\text{SHBG}$. The IR was determined by calculating the homeostasis model assessment score as $\text{fasting plasma glucose (in milligrams per deciliter)} \times \text{fasting plasma insulin (in microunits per milliliter)}/405$.

All patients also performed an oral glucose tolerance test (OGTT; 75 g of glucose); glycemia and insulinemia were assayed basally and every 30 minutes for the 2 following hours. The OGTT data were analyzed as the insulinemic area under the curve calculated by the trapezoidal rule (28).

Metabolic syndrome was diagnosed by the presence of three or more of the following five criteria, according to the US National Cholesterol Education Program Adult Panel Treatment III (NCEP-ATP III) definition: [1] waist circumference ≥ 88 cm, [2] hyperglycemia: fasting plasma glucose ≥ 110 mg/dL or medication for hyperglycemia, [3] plasma triglycerides ≥ 150 mg/dL, [4] HDL cholesterol < 50 mg/dL, and [5] blood pressure $\geq 130/85$ mm Hg or use of medication for hypertension (29).

Assays

All hormones were measured in our laboratory. Levels of A (normal range of our laboratory: 0.40–1.3 ng/mL), T (0.20–0.60 ng/mL), SHBG (25–100 nmol/L), DHEAS (800–3,000 ng/mL), 17-OHP (0.2–1.2 ng/mL) were measured in duplicate by RIA methods using a commercial kit (Radim). The intra-assay and interassay coefficients of variation (CV) for all the mentioned hormones were less than 7% and less than 12%, respectively. Insulin (basal, 5.0–20.0 $\mu\text{UI}/\text{mL}$) was determined by an immunoradiometric assay (DiaSorin) and the intra-assay and interassay CV were 2.1%–2.6% and 2.9%–4.7%, respectively.

Plasma glucose (basal, 65–110 mg/dL) was determined by the glucose oxidase method. Glucose plasma concentrations were determined by the glucose oxidase technique with a glucose analyzer (Beckam). Total cholesterol (130–200 mg/dL) and triglyceride (20–150 mg/dL) concentrations were determined by an enzymatic assay (Bristol). The HDL (> 50 mg/dL) concentrations were determined after precipitation of chylomicrons, very low-density lipoprotein, and LDL (Boehringer). A magnesium chloride/phosphotungstic acid technique was used to precipitate LDL (< 130 mg/dL) from the bottom fraction after ultracentrifugation.

Sample Size

Our sample size was based on the primary outcome.

A sample size of 27 patients (psoriatic group) and 50 patients (control group) would guarantee a power of at least 80%, if assuming a delta of 30% in the prevalence of PCOS between the psoriatic and the control group. The statistical test used was the two-sided Fisher's exact test and the significance level of the test was targeted at 0.05. We aimed to recruit 62 psoriatic patients and 107 controls, 169 in total to compensate for possible missing data or drop-outs.

Statistical Analysis

Data distribution were assessed by the Kolmogorov-Smirnov test. Continuous variables were expressed as mean \pm SD or median and interquartile range, as appropriate; dichotomous variables were expressed as percentages. Continuous variables among the groups in the study population were compared with unpaired *t*-test or Mann-Whitney *U* test as appropriate; categorical variables were compared using the Fisher's exact test. The results were expressed, when appropriate, as odds ratio (OR) and 95% confidence intervals (CI). The prevalence of PCOS in women with psoriasis was compared with that in controls stratifying data by MS, hypertension, and family history of early coronary disease, according to the Mantel-Haenszel method. All analyses were performed by use of SPSS, v.16.0 software.

In addition, Cohen kappa statistic was calculated to determine agreement between the two observers in defining the PASI and PGA score. The *P* and κ values that approximated 1 denoted perfect agreement, whereas values that approximated 0 denoted agreement no better than that by chance. Guidelines for evaluating level of agreement were: > 0.80 good, 0.60–0.80 moderate/fair, < 0.60 poor (30).

Outcomes

The primary outcome of the present study was to estimate the prevalence of PCOS in patients with psoriasis. The secondary outcome was the evaluation of the clinical-biochemical features (waist-to-hip ratio, hypertension, MS, lipid profile, homeostasis model assessment of insulin resistance, insulinemic area under the curve, androgenic levels, severity index of psoriasis) among the group of women with psoriasis with and without PCOS.

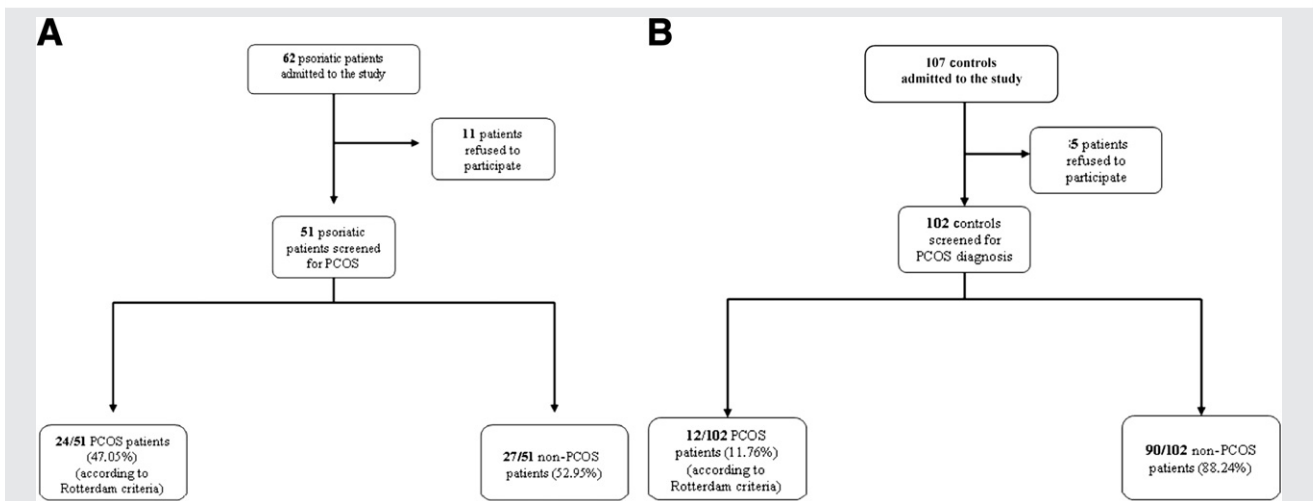
RESULTS

Prevalence of PCOS in Psoriatic Patients and Control Group

During the enrolment period, 201 women were evaluated. Ninety-nine patients were ruled out because of out of the age range, 17 because they were affected by diabetes, 11 because of thyroidal dysfunction, and 12 because of taking hormonal contraceptives. Therefore, 62 women with psoriasis were within the inclusion and exclusion criteria and were seen in the Dermatology Outpatient Clinic and of those 51 agreed to participate in the study (Fig. 1A). In addition, we admitted 107 age- and BMI-matched controls from our Gynaecological Outpatient Clinic, and 102 of them accepted to be screened for PCOS (Fig. 1B).

The prevalence of PCOS was more in patients with psoriasis than in matched control subjects. In particular, PCOS

FIGURE 1



Study flow chart. (A) Flow chart showing the progress of psoriatic patients through the various stages of the cross-sectional cohort study. (B) Flow chart showing age- and body mass index (BMI)-matched controls without psoriasis progress through the various stages of the cross-sectional cohort study. PCOS = polycystic ovary syndrome.

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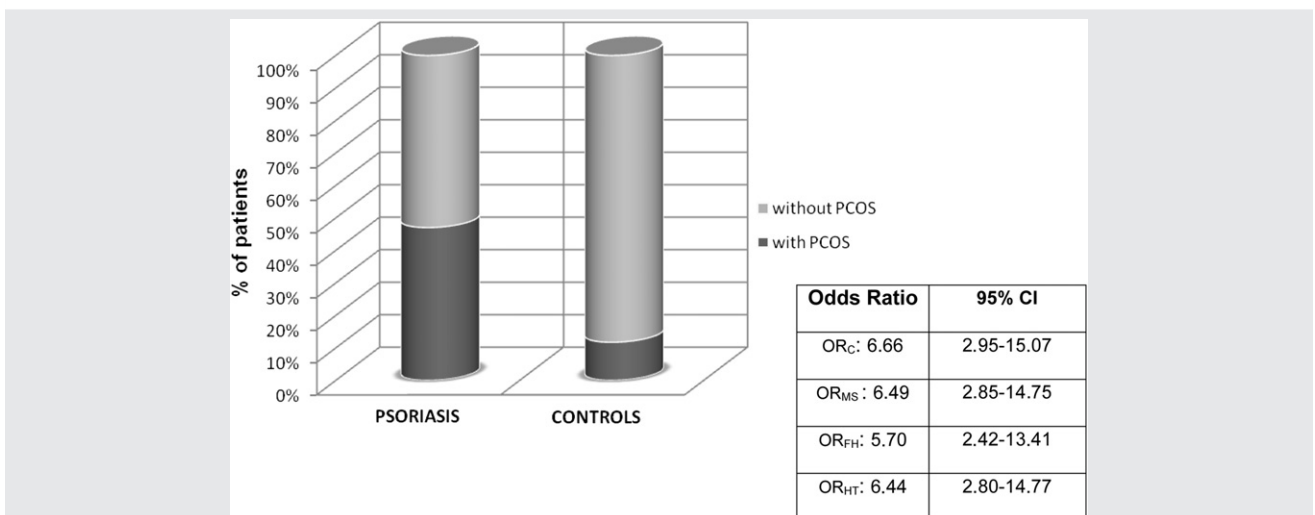
has been diagnosed in 24 patients (47.05%) of the psoriatic group and in 12 patients (11.76%) of the control group (OR, 6.66; 95% CI 2.95–15.07; $P=.000$) (Fig. 2). Polycystic ovary syndrome was not yet diagnosed in the 24 patients with both psoriasis and PCOS. After stratifying for MS, family history of early coronary disease, and hypertension, adjusted ORs were similar to crude OR (OR, 6.49, 95% CI 2.85–14.75; OR, 5.70, 95% CI 2.42–13.41; OR, 6.44, 95% CI 2.80–14.77, respectively) (Fig. 2).

Characteristics of the Study Population

The clinical and biochemical characteristics of psoriatic and control group are summarized in Table 1.

No differences were observed in terms of age, BMI, and waist-to-hip ratio between PCOS and non-PCOS groups. Regarding to the cardiovascular risk factors, the two groups were similar in terms of smoking status, hypertension, obesity (BMI >30), family history of early coronary artery disease, and MS (Table 1).

FIGURE 2



Prevalence histogram of polycystic ovary syndrome (PCOS) between psoriasis and control groups. OR_C = crude odds ratio; OR_{MS} = odds ratio adjusted for metabolic syndrome; OR_{FH} = odds ratio adjusted for family history of early coronary artery disease; OR_{HT} = odds ratio adjusted for hypertension; 95% CI = 95% confidence interval. $P=.000$.

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TABLE 1

Clinical and biochemical features of the study population.

Parameter	Psoriasis with PCOS (n = 24)	Psoriasis without PCOS (n = 27)	Controls (n = 102)	P value
Age (y), mean ± SD	31 ± 4	32 ± 5	33 ± 4	NS
BMI (kg/m ²), median (range)	26.8 (21–29.2)	26.6 (20–30.3)	27.2 (22–38.7)	NS
BMI>30, n (%)	8 (33)	8 (29)	32 (31.7)	NS
WHR, median (range)	0.87 (0.6–0.98)	0.86 (0.7–0.95)	0.81 (0.68–0.91)	NS
Smoking, n (%)	8 (33.3)	8 (29.6)	30 (29.4)	NS
Family history, n (%)	8 (33.3)	4 (14.8)	9 (8.8)	b,c
Hypertension, n (%)	4 (16.6)	5 (18.5)	15 (14.7)	b
Metabolic syndrome, n (%)	6 (25)	3 (11.1)	6 (6.1)	b,c
Total cholesterol (mg/dL), mean ± SD	184.3 ± 25.3	164.5 ± 19.4	159.0 ± 18.0	a,c
LDL cholesterol (mg/dL), mean ± SD	109.7 ± 17.0	93.7 ± 10.4	67.0 ± 7.1	b,c
HDL cholesterol (mg/dL), mean ± SD	49.5 ± 12.1	58.4 ± 9.0	89.1 ± 13.0	a,b,c
Triglycerides (mg/dL), mean ± SD	116.0 ± 28.4	99.6 ± 34.1	73.0 ± 18.0	b,c
Glycemia (mg/dL), mean ± SD	85.7 ± 10.3	79.5 ± 15.5	71.5 ± 8.0	b,c
Insulin (μIU/mL), mean ± SD	15.5 ± 4.9	7.8 ± 2.6	5.8 ± 1.4	a,b,c
Insulinemic area (μIU/mL × 120), mean ± SD	9,353.0 ± 3,998	5,111.0 ± 2,984	5,748.0 ± 2,543	a,c
HOMA-IR, median (range)	2.94 (1.7–3.7)	1.49 (0.8–2.4)	1.2 (0.9–2.1)	a,c
T (ng/mL), median (range)	0.66 (0.27–0.8)	0.33 (0.24–0.55)	0.35 (0.3–0.5)	a,c
FAI, mean ± SD	7.5 ± 1.6	1.6 ± 0.7	1.4 ± 0.6	a,c
SHBG (nmol/L), median (range)	33.3 (25.3–54.0)	61.0 (28.0–67.4)	42.0 (31.0–58.0)	a,b,c
A (ng/mL), median (range)	3.5 (1.5–4.2)	1.8 (1.6–2.5)	1.7 (1.4–2.4)	a,c
DHEAS (ng/mL), median (range)	3,295.0 (1,646.0–2,790.0)	2,004.0 (1,558.0–2,785.0)	1,987.0 (1,457.0–2,893.0)	a,c
17-OHP (ng/mL), median (range)	0.7 (0.5–0.7)	0.6 (0.4–0.7)	0.5 (0.3–0.8)	NS
Ovarian volume (cm ³), median (range)	11.5 (9.1–13.8)	6.43 (5.6–9.8)	6.12 (5.4–8.9)	a,c

Note: χ^2 test or Fisher's exact test, as appropriate, has been used for categorical variables. For continuous variables, Student's *t* test or Mann-Whitney *U* test has been used, as appropriate. A *P* value of < .05 was considered significant. A = Δ 4-androstenedione; BMI = body mass index; FAI = free androgen index; Family history = family history of early coronary artery disease; HOMA-IR = homeostasis model assessment of insulin resistance; NS = no statistical differences between the groups; 17-OHP = 17 α -hydroxyprogesterone; WHR = waist-to-hip ratio.

^a Significant difference (*P* < .05) between psoriatic patients with and without PCOS.

^b Significant difference (*P* < .05) between psoriatic patients without PCOS and controls.

^c Significant difference (*P* < .05) between psoriatic patients with PCOS and controls.

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Concerning the lipid profile, patients with PCOS showed a significant increase of total cholesterol levels (*P* = .031) and reduced levels of HDL cholesterol (*P* = .026). Conversely, no differences were observed between the two groups in terms of LDL cholesterol and triglycerides (Table 1). In addition, fasting plasma insulin, insulinemic area under the curve, and homeostasis model assessment of insulin resistance in patients with PCOS were significantly higher than in women without PCOS (*P* = .001, *P* = .019, *P* = .042, respectively). As expected, the androgen levels and ovarian volume were higher in the PCOS group when compared to the non-PCOS group (Table 1).

All participants presented chronic plaque psoriasis. Psoriatic arthritis was diagnosed in 15 patients (29.4%), with a similar prevalence in the two groups. Interestingly, all

severity indexes of psoriasis were found to be increased in women affected by PCOS. In particular, the PCOS group showed a prevalence of PASI \geq 10 and PGA \geq 4 (from moderate to severe) remarkably greater than the non-PCOS group (*P* = .046; OR, 3.5, 95% CI 1.04–11.7; *P* = .045; OR, 3.75, 95% CI 1.13–12.39, respectively) (Table 2). Interobserver agreement about severity index of psoriasis between the two dermatologists was significantly good (PASI score \geq 10, κ = 0.87; *P* \geq .001 and PGA score \geq 4, κ = 0.84; *P* \geq .001). Regarding to the mean duration of psoriasis diagnosis, no differences were observed between the two groups (Table 2), even if the clinical history of previous systemic treatment was more frequent in the PCOS group than in the non-PCOS group (*P* = .045; OR, 3.75, 95% CI 1.13–12.39) (Table 2).

TABLE 2

Psoriatic severity index in psoriatic cohort.

Parameter	Psoriasis with PCOS (n = 24)	Psoriasis without PCOS (n = 27)	P value	OR (95% CI)
Psoriatic arthritis, n (%)	7 (29.1)	8 (29.6)	NS	
PASI score \geq 10, n (%)	12 (50.0)	6 (22.2)	.046	3.5 (1.04–11.7)
PGA score \geq 4, n (%)	18 (75.0)	12 (44.4)	.045	3.75 (1.13–12.39)
Previous systemic therapies, n (%)	18 (75.0)	12 (44.4)	.045	3.75 (1.13–12.39)
Duration of psoriasis diagnosis (mo), mean ± SD	12.0 ± 8.0	13.0 ± 9.0	NS	

Note: χ^2 test or Fisher's exact test, as appropriate, has been used for categorical variables. NS = no statistical differences between the groups; OR (95% CI) = odds ratio (95% confidence interval); PASI = Psoriasis Area and Severity Index (PASI score \geq 10 was considered an indicator of severe disease); PGA = Physician's Global Assessment (PGA score \geq 4 indicate a disease from moderate to severe).

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Topical steroids were used in 45 patients with psoriasis at the time of evaluation, in particular, in 21 PCOS subjects and in 24 non-PCOS patients ($P > .05$). No evidence of iatrogenic Cushing's syndrome was found in the two groups.

DISCUSSION

To our knowledge this is the first study on the potential association between PCOS and psoriasis. In the general population, the PCOS prevalence has been estimated to be in the 6%–10% range according to National Institutes of Health criteria and about 15% when the broader Rotterdam criteria are applied (12). Similarly, in our control population, there were about 11% patients with PCOS.

The present study shows that in patients with psoriasis the prevalence of PCOS is remarkably greater (~47%) than in control women who were matched for age and BMI, independently of some potential confounders, such as MS, hypertension, and family history of early coronary disease. This strong association between PCOS and psoriasis could be consequent to the fact that both diseases have similar comorbidities such as IR and increased cardiovascular risk. Although the etiology of PCOS is unknown, evolving data suggest that IR could play an important pathophysiological role in the development of this syndrome and may also contribute to the PCOS-related increase of CVD risk (17, 31). In addition, risk factors for CVD, including dyslipidemia, hypertension, oxidative stress, inflammation, and increased frequency of CD4⁺ CD28null T lymphocytes, are associated with PCOS (32).

Similarly, compared with healthy subjects, women with psoriasis are more frequently affected by IR (2) and seem to present an increased incidence of CVD. In particular, the relative risks of myocardial infarction and stroke are higher in patients with psoriasis compared with the general population. These aspects are especially observed in younger patients with more severe disease, and they could contribute to the 3- to 4-year reduction in life expectancy among patients with severe psoriasis (8).

Psoriasis is a chronic inflammatory, immune-mediated skin disease characterized by marked proliferation of keratinocytes leading to pronounced epidermal hyperplasia elongation of rete ridges and hyperkeratosis. The most common form of psoriasis is the chronic plaque psoriasis and innate as well as adaptive immunity are crucial in the development and maintenance of the plaque. In particular, type 1 CD8⁺ and type 17 CD4⁺ T lymphocytes and their products, such as interferon- γ , interleukins, and tumor necrosis factor (TNF)- α are essential to the disease expression (33). Similarly, PCOS is a low-grade chronic inflammation condition; several factors, like TNF- α and polymerase chain reaction (PCR), are increased in this population (34). This occurrence could explain the higher psoriasis severity scores in patients having both psoriasis and PCOS.

In addition, the mentioned proinflammatory cytokines, as well as other several inflammatory markers, are overproduced in patients with PCOS and psoriasis and are strongly associated with IR and CVD risk (8, 34–36). These factors support our results showing a higher insulinemic area under the curve and homeostasis model assessment of insulin

resistance in women with PCOS compared with patients with psoriasis alone.

Our study presents some limitations. We are aware that of the low numbers patients screened and we are currently trying to increase our population to confirm our preliminary data. In conclusion, our findings could have important clinical implications, PCOS being a common condition in psoriatic women, affecting nearly half of these patients. Nevertheless, women with PCOS and psoriasis are more likely to have IR, hyperinsulinemia, reduced HDL cholesterol levels, and a more severe degree of skin disease than those with psoriasis alone. Therefore, these data suggest that women with psoriasis need a multidisciplinary approach, which should take in consideration the possibility of a gynecological and metabolic derangement.

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