



# Psoriasis and polycystic ovary syndrome: a new link in different phenotypes



Francesca Moro<sup>a,\*</sup>, Anna Tropea<sup>a</sup>, Elisa Scarinci<sup>a</sup>, Alex Federico<sup>a</sup>, Clara De Simone<sup>b</sup>, Giacomo Caldarola<sup>b</sup>, Emanuele Leoncini<sup>c</sup>, Stefania Boccia<sup>c</sup>, Antonio Lanzone<sup>a,1</sup>, Rosanna Apa<sup>a,1</sup>

<sup>a</sup> Institute of Obstetrics and Gynecology, Università Cattolica del Sacro Cuore, Rome, Italy

<sup>b</sup> Institute of Dermatology, Università Cattolica del Sacro Cuore, Rome, Italy

<sup>c</sup> Institute of Public Health, Section of Hygiene, Università Cattolica del Sacro Cuore, Rome, Italy

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## ABSTRACT

**Objective:** Women affected by PCOS and psoriasis are more likely to have insulin-resistance, hyperinsulinemia, reduced HDL cholesterol levels and a more severe degree of skin disease than those with psoriasis alone. The mechanism underlying this association between PCOS and psoriasis is currently unknown. The aim of the present study was to evaluate the features of psoriasis and the psoriasis severity scores in the different PCOS phenotypes and in age and body mass index (BMI)-matched psoriatic control patients.

**Study design:** A cross-sectional study was performed on 150 psoriatic patients: 94 PCOS and 56 age- and BMI-matched controls. PCOS patients were diagnosed and divided into four phenotypes according to Rotterdam criteria: A – patients with complete phenotype with hyperandrogenism (H) *plus* oligoamenorrhea (O) *plus* polycystic ovary (PCO) on ultrasound examination; B – patients with H *plus* O (without PCO); C – patients with H *plus* PCO (ovulatory phenotype); D – patients with O *plus* PCO (without H).

The patient's Psoriasis Area and Severity Index (PASI) as well as the Physician's Global Assessment (PGA) were calculated. A PASI score  $\geq 10$  was correlated with common indicator of severe disease. A PGA  $\geq 4$  was considered as a condition of moderate to severe disease.

**Results:** Among the four phenotypes investigated, the group with complete phenotype (H *plus* O *plus* PCO) had a higher prevalence of patients with patient's PASI  $\geq 10$  compared to controls (Odds Ratio (OR) 4.71, 95% confidence intervals (CI) 1.59–13.95). The group with O *plus* PCO had a higher prevalence of patients with PGA  $\geq 4$  compared to controls (OR 26.79, 95% CI 3.40–211.02) while the ovulatory group had a lower prevalence of patients with PGA  $\geq 4$  (OR 0.06, 95% CI 0.01–0.51).

**Conclusions:** The ovulatory phenotype displays a milder psoriasis form than other phenotypes while the phenotypes with oligoamenorrhea presented higher severity scores of disease than other phenotypes and control group.

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## Introduction

Polycystic ovary syndrome (PCOS) is a heterogeneous condition characterized by several symptoms and clinical signs related to reproductive, cardiometabolic and psychological disorders

[1,2]. In general population, the PCOS prevalence has been estimated to be in the 6–10% range according to National Institutes of Health (NIH) criteria and becoming about 15% when the broader Rotterdam criteria are applied [3]. According to the Rotterdam criteria, PCOS is defined by the presence of two of the following three features (excluding other aetiologies): biochemical and/or clinical hyperandrogenism (H), oligo or anovulation (O) and polycystic ovary (PCO) on ultrasound [4]. Thus, the Rotterdam Consensus has generated 4 different phenotypes, highlighting the clinical heterogeneity of this syndrome [1,5]: complete phenotype with all features (H + O + PCO), phenotype with H + O,

\* Corresponding author at: Institute of Obstetrics and Gynecology, Università Cattolica del Sacro Cuore, Largo A. Gemelli 8, 00168 Rome, Italy.

Tel.: +39 06 30156770; fax: +39 06 3057794.

E-mail address: [morofrancy@gmail.com](mailto:morofrancy@gmail.com) (F. Moro).

<sup>1</sup> These authors shared senior authorship.

phenotype with H and PCO without O and phenotype with O and PCO without H.

Interestingly, short time ago we demonstrated that PCOS is a common condition in psoriatic women affecting nearly half of them [6].

Similarly to PCOS, psoriasis is often associated with pathological conditions, such as insulin resistance (IR), type 2 diabetes (DM2), obesity and it has potentially increased risk for cardiovascular disease (CVD) [7–12]. We previously demonstrated that 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 [6]. The mechanism underlying this association between PCOS and psoriasis is currently unknown, even though the chronic inflammatory status characterizing both conditions [13,14], probably feeds the common features of the two diseases.

Based on these observations, we conducted a cross-sectional study to determine the features of psoriasis and the psoriasis severity scores in the different PCOS phenotypes and in age and body mass index (BMI)-matched psoriatic control patients.

## Materials and methods

This study is a cross-sectional study evaluating 242 reproductive aged patients with chronic plaque psoriasis who attended to the Dermatology Outpatient Clinic between January 2010 and November 2014. Of these women, 92 were excluded due to the exclusion criteria: 37 women had an autoimmune disease, 22 patients took an oral contraceptive, 29 were taking insulin-sensitizing drugs or systemic treatment for psoriasis at the time of evaluation and 4 patients were excluded for other exclusion criteria. Therefore, 150 were included in the study for final analysis: 94 PCOS women and 56 age- and BMI-matched controls.

All women enrolled in this study were examined by specialist dermatologists, who diagnosed chronic plaque psoriasis on clinical grounds and psoriatic arthritis in accordance with Caspar criteria [15]. The patient's Psoriasis Area and Severity Index (PASI) as well as the Physician's Global Assessment (PGA) were calculated. The PASI is a composite score ranging from 0 to 72 and reflects the extent, erythema, induration and scaliness of the lesions in 4 body areas (head, trunk, arms and legs) [16]. A PASI score (PASIs)  $\geq 10$  was correlated with common indicators of severe disease (e.g. need of systemic therapy, hospitalization) [17]. The PGA score (PGAs) is a descriptive assessment using a seven-point ordinal scale to assess the global severity of disease over the body as a whole. This assessment is made by the physician's judgment and does not take into account the different districts concerned. The following categories were used: 'clear' = no signs of psoriasis (postinflammatory hyperpigmentation may be present); 'almost clear' = intermediate between mild and clear; 'mild' = slight plaque elevation, scaling, and/or erythema; 'mild to moderate' = intermediate between moderate and mild; 'moderate' = moderate plaque elevation, scaling, and/or erythema; 'moderate to severe' = marked plaque elevation, scaling, and/or erythema; 'severe' = very marked plaque elevation, scaling, and/or erythema. For statistical analysis, the scale was assigned scores of 0 to 6, from 'clear' to 'severe', respectively [18].

All PCOS patients were diagnosed and divided into four phenotypes according to Rotterdam criteria [4]: patients with complete phenotype with H + O + PCO (A phenotype); patients with H + O (B phenotype); patients with H + PCO (C phenotype) and those with O + PCO (D phenotype).

The exclusion criteria were: neoplasm, autoimmune disease, treatment with clomiphene citrate, oral contraceptives, antiandrogens, drugs to control their appetite or insulin-sensitizing drugs (metformin, pioglitazone and rosiglitazone) during the last

6 months prior to our evaluation, DM2, major surgery in the last 3 months, or other hormonal dysfunction (hypothalamic, pituitary, thyroidal, or adrenal causes). 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 steroid were admitted at the time of the evaluation, only if applied on less 10% of skin surface and for limited periods time (3–4 weeks). To our knowledge, none of the Rotterdam criteria was influenced by the drugs commonly used in psoriasis treatment.

The following data were obtained from our database: age, BMI ( $\text{kg}/\text{m}^2$ ), waist-hip ratio (WHR), hirsutism, acne, O, IR and PCO on ultrasound. Hirsutism was evaluated with Ferriman-Gallwey (FG) map scoring system (hirsutism was diagnosed if  $\text{FG} > 8$ ) [19], and clinical acne was defined by a history of persistent acne (presence of acne on most days for at least 3 years), recent acne treatment and presence of more than 10 inflammatory acne lesions [20]. Oligoamenorrhea was defined by  $< 8$  spontaneous menstrual cycles per year for at least 3 years before the first examination. IR was determined by calculation of the homeostasis model assessment (HOMA) score as  $\text{fasting plasma glucose (mg/dL)} \times \text{fasting plasma insulin (}\mu\text{U/mL)} / 405$ . For the diagnosis of PCO morphology, all women underwent transvaginal ultrasonography during the early follicular phase. Ovarian volume was calculated by following formula:  $V = (\pi/6) \times D_{\text{length}} \times D_{\text{width}} \times D_{\text{thickness}}$ , where  $D$  denotes the dimension. The presence of PCO was diagnosed by the presence of 12 or more follicles measuring 2–9 mm in diameter in each ovary and/or increased ovarian volume ( $> 10 \text{ cm}^3$ ) [4].

At day 3 of a spontaneous menstrual cycle or after an amenorrhea  $> 60$  days (with a proven anovulation: plasma progesterone  $< 1.5 \text{ ng/mL}$ ), all patients were tested for plasma levels of androstenedione (A), sex hormone-binding globulin (SHBG), 17-hydroxyprogesterone (17-OHP), dehydroepiandrosterone sulphate (DHEAS), triglycerides, total cholesterol, and high-density lipoprotein cholesterol (HDL). The free androgen index (FAI) was calculated by following formula:  $\text{Testosterone} \times 100 / \text{SHBG}$ .

All patients also performed an oral glucose tolerance test (OGTT; 75 g of glucose), glycaemia and insulinaemia were assayed basally and every 30 min for the two following hours.

The OGTT data were analyzed as the insulinaemic area under the curve calculated by the trapezoidal rule ( $\text{AUC}_{\text{0-2h}}$ ) [21].

All hormones were measured in our laboratory. Levels of A (normal range of our laboratory:  $0.40\text{--}13 \text{ ng/mL}$ ), SHBG ( $25\text{--}100 \text{ nmol/L}$ ), DHEAS ( $800\text{--}3000 \text{ ng/mL}$ ), 17OHP ( $0.2\text{--}1.2 \text{ ng/mL}$ ), were measured in duplicate by radioimmunoassay methods using a commercial kit (Radim, Pomezia, Italy). The intra- and interassay coefficients of variation for all the above mentioned hormones were less than 7% and less than 12%, respectively.

Insulin (basal:  $5.0\text{--}20.0 \mu\text{U/mL}$ ) was determined by an immunoradiometric assay (DiaSorin, Vercelli, Italy) and the intra-assay and interassay coefficients of variation were 2.1–2.6% and 2.9–4.7%, respectively.

Plasma glucose (basal:  $65\text{--}110 \text{ mg/dL}$ ) was determined by the glucose oxidase method. Glucose plasma concentrations were determined by the glucose oxidase technique with a glucose analyzer (Beckam, Fullerton, CA, USA). Total cholesterol ( $130\text{--}200 \text{ mg/dL}$ ) and triglyceride ( $20\text{--}150 \text{ mg/dL}$ ) concentrations were determined by an enzymatic assay (Bristol, Paris, France). HDL ( $> 50 \text{ mg/dL}$ ) concentrations were determined after precipitation of chylomicrons, VLDL and LDL (Boehringer, Mannheim, Germany).

Continuous variables were expressed as mean  $\pm$  standard deviation (SD) or median and interquartile range as appropriate; dichotomous variables were expressed as percentages. Continuous variables among the four groups of subjects in the study population were compared with analysis of variance or Kruskal–Wallis test as

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