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Research Article

Cervical Squamous Intraepithelial Lesions in Women with Polycystic Ovary Syndrome: A Descriptive Series of 9 Cases

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Abstract

Introduction: Few studies have explored the vulnerability of women with polycystic ovary syndrome (PCOS) to developing cervical cancer and its precursor lesions. Objective: To describe the clinical, metabolic, and endocrine characteristics of a series of women with cervical squamous intraepithelial lesions and polycystic ovary syndrome, aiming to generate hypotheses regarding the potential pathophysiological mechanisms linking both conditions.

Methods: A descriptive and hypothesis-generating study was conducted. The series consisted of nine women with a cytological diagnosis of cervical squamous intraepithelial lesions (SIL) who also met the Rotterdam criteria for PCOS. Characterization included age, sexual behavior, toxic habits, history of hypertension and diabetes mellitus, body mass index (BMI) and waist-to-hip ratio (WHR), metabolic parameters (insulin resistance, dyslipidemia), serum hormone levels (testosterone, prolactin, estradiol), human papillomavirus (HPV) 16/18 infection, grade of the lesion, and PCOS phenotypes.

Results: The mean age was 37,11 ± 12,8 years. Abdominal obesity was detected in 55,5% and insulin resistance in 44,4% of cases. Hyperprolactinemia was present in 33,3%. HPV 16/18 infection was identified in 77,7% of cases. Most patients presented high-grade squamous intraepithelial lesions (HSIL) and PCOS phenotype D.

Conclusion: The presence of HSIL in more than a third of the women in this case series is compatible with the hypothesis that PCOS, particularly those with insulin resistance, abdominal obesity, or hyperprolactinemia, may act as a multifactorial risk factor for cervical lesions, either independently or synergistically through metabolic and hormonal pathways that interact with HPV. These findings should be interpreted as preliminary observations that warrant confirmation in larger, controlled studies.

More Information

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Keywords: Cervical squamous intraepithelial lesions; Human Papillomavirus (HPV); Polycystic Ovary Syndrome (PCOS); Multifactorial risk factor

Introduction

Polycystic ovary syndrome (PCOS) is an endocrinopathy that occurs in women of reproductive age. It has broad health implications for those affected, either intrinsically or through associated comorbidities. It may present with familial heritability patterns and can show progressive worsening of its clinical manifestations [1]. It is characterized by the presence of at least two of the following criteria: clinical and/or biochemical hyperandrogenism, oligo-anovulation—manifested by menstrual disorders or infertility—and polycystic ovarian morphology on ultrasound (PCOM) [2,3]. The global prevalence of PCOS ranges from 4% to 21%, depending on the classification used [4]. The Rotterdam diagnostic criteria identify four phenotypes with distinct clinical presentations. Phenotype A includes hyperandrogenism, oligo-anovulation, and PCOM. Phenotype B lacks PCOM; phenotype C presents without ovulatory

dysfunction; and phenotype D is normoandrogenic [5]. The frequency of these phenotypes varies according to geographic, ethnic, and racial factors [6].

The etiology of PCOS is complex and not yet fully understood. Hyperandrogenism is known to result from alterations in the hypothalamic–pituitary–ovarian/adrenal axis. Risk factors for the development of PCOS include physical inactivity, diabetes mellitus, obesity, and family history. Alterations in reproductive and metabolic hormones observed in women with this syndrome increase the risk of obesity, insulin resistance, diabetes, dyslipidemia, hypertension, osteoporosis, psychiatric disorders, and cancer [7].

Recent evidence links PCOS with an increased risk of gynecological cancers. Women with this syndrome are three times more likely to be diagnosed with endometrial carcinoma compared to those without it. However, there remains a knowledge gap regarding which medical conditions in women with PCOS contribute to this type of cancer and what confounding factors are present [8]. The association with ovarian, breast, vaginal, vulvar, and cervical cancers has not been conclusively demonstrated, highlighting the need for further research in this area [9,10].

Although sufficient scientific evidence linking PCOS and cervical squamous intraepithelial lesions (SIL) is lacking, both conditions share certain risk factors. Given the endocrine–metabolic alterations present in PCOS, it is hypothesized that women with PCOS may exhibit a higher probability of developing SIL compared to the general population, so this syndrome could act as a multifactorial risk factor for cervical lesions. The objective of this study is to describe the clinical, metabolic, and endocrine characteristics of a series of women with cervical squamous intraepithelial lesions and polycystic ovary syndrome, aiming to generate hypotheses regarding the potential pathophysiological mechanisms linking both conditions.

Methods

In this study, from patient recruitment to data analysis, the condition under investigation was universally known as Polycystic Ovary Syndrome (PCOS). Coinciding with the completion of this work, an international consensus proposed a new term, Polyendocrine Metabolic Ovarian Syndrome (PCOS), to better represent its complex pathophysiology. However, it established a three-year transition period until 2028 for its full adoption [11]. In this publication, the previous nomenclature (PCOS) is retained, as it was the term used throughout the research process.

Study design and sample size

A descriptive and hypothesis-generating study was conducted. All women with cervical SIL who reported having a previous diagnosis of PCOS (n = 9) were selected when attending the gynecology classification consultation of the Institute of Oncology and Radiobiology (INOR) within the framework of a study previously described by Frontela, et al. [12]. All patients had been previously diagnosed with PCOS using the 2003 Rotterdam criteria (presence of at least two of the following: oligo-anovulation, clinical or biochemical hyperandrogenism, and PCOM [2]. These diagnoses were verified upon referral by a specialist in Endocrinology, who reviewed the available medical records, including hormonal profiles and ultrasound reports from the referring institutions. In cases where documentation was incomplete, complementary tests were ordered to confirm the diagnosis.

Sample collection

Patients who agreed to participate in the study underwent an interview, anthropometric measurements (weight, height, waist circumference, and hip circumference) were taken, and blood samples were obtained after an overnight fast, during the early follicular phase (days 3–5 of the menstrual cycle) or on any day in amenorrheic patients, for biochemical and hormonal analysis.

Regarding the cervical pathology, exfoliated cervical cells or colposcopy-directed biopsies were obtained for cytohistological studies, considering low grade (LSIL) when cervical intraepithelial neoplasia type 1 (CIN 1) was present and high grade (HSIL) when CIN2/CIN3/carcinoma i*n situ,* according to the Bethesda System [13].

Biochemical and hormonal analysis

Fasting blood glucose, triglycerides, cholesterol and HDL-c (cholesterol united to high-density lipoprotein) levels were determined. In addition, estradiol (E2), testosterone (T), and insulin (INS) determinations were performed by radioimmunoassay (RIA). Additionally, prolactin (Prl) was determined by immunoradiometric analysis (IRMA) using reagent kits from the Isotope Institute of Budapest and the company DIA source Immuno Assays S.A.

DNA isolation and purification

Additional exfoliated cervical cell samples were obtained and stored at - 20 °C in a transport solution (Digene Inc., Gaithersburg, MD) for subsequent use in DNA isolation and purification using the saline extraction method [14]. DNA quantification was performed by spectrophotometry in the ultraviolet range using a Colibri microvolume spectrophotometer (TITERTEK, Germany). The concentration was calculated using the following formula: DNA concentration (ng/µL) = Optical density 260 nm x 100 (dilution) x 50 (conversion constant). The ratios (OD 260 nm/OD 280 nm and OD 260/OD 230) were determined to verify the purity obtained. Values around 1,8 were indicative of purity. Furthermore, the integrity of the molecule was analyzed through electrophoresis in a 0,8% agarose gel and staining with ethidium bromide 10 mg/mL [14].

HPV detection by end-point Polymerase Chain Reaction (PCR)

The purified DNA was used for HPV detection by amplification of a region of the viral L1 gene, which encodes the structural protein L1, using the GP5+ and 5′-biotinylated GP6+ primers synthesized by Sigma-Aldrich (Genosys), according to Schmitt et al., 2008 [15]. The 5′-3′ nucleotide sequences used were: TTT GTT ACT GTG GTA GAT ACT AC and GAA AAA TAA ACT GTA AAT CAT ATT C, respectively. The size of the amplified product was 150 bp. The amplification program consisted of denaturation at 94 °C for 4 min, followed by 40 amplification cycles. Each cycle included a denaturation step at 94 °C for 1 min, annealing at 40 °C for 2 min, and elongation at 72 °C for 1 min. The final elongation step was carried out at 72 °C for 4 min.

Amplification of the β-globin gene was used as an internal control for the reaction. For this, the MS3 and MS10 5' biotinylated primers, synthesized by Sigma-Aldrich (Genosys), were used. The 5'-3' nucleotide sequences used were: AAA ATA TGT GTG CTT ATT TG and AGA TTA GGG AAA GTA TTA GA, respectively. The amplicon obtained from the β-globin gene was 200 bp [16].The amplification reaction and detection were performed under the same conditions previously described for the HPV L1 gene.

All end-time amplification reactions were performed in a conventional TC-3000 thermocycler (TECHNE). The amplified products were visualized by electrophoresis on a 2% agarose gel and stained with 10 mg/mL ethidium bromide [14].

HPV 16/18 genotyping by real-time PCR

HPV 16/18 genotyping was performed using real-time PCR with the SUMASIGNAL HPV 16/18 diagnostic kit (SANSURE BIOTECH INC.), which is based on the amplification of the HPV L1 gene, marketed by the Center for Immunoassay (CIE) and validated by Soto, et al. (2022) at the Pedro Kourí Institute of Tropical Medicine (IPK), Havana, Cuba. The SUMASIGNAL HPV 16/18 kit showed excellent performance indicators (> 95%), 96% concordance, and a kappa index of 0,93, compared to the commercial HPV 16/18 Real-TM Quant kit (Sacace Biotechnologies, Italy), certified by the European Community, the Food and Drug Administration Agency, and the World Health Organization for use in *in vitro* HPV diagnosis [17].

The reactions were performed in 0,2 mL PCR tubes containing, for each sample, 38 µL of PCR mix, 2 µL of enzyme mix (Uracyl-N-Glycosylase and Reference Marker), and 0,25 µL of internal control. Five microliters of each sample under study, four quantitative references (A, B, C, D), and the positive and negative controls were added. Reactions were carried out in the SLAN-96P thermocycler (SANSURE BIOTECH INC.) using the following parameters: 50 °C for 2 min for the UNG enzyme reaction; 94 °C for 5 min for Taq polymerase enzyme activation; and 45 cycles of hybridization, extension, and fluorescence reading at 94 °C for 15 s and 57 °C for 30 s. followed by a cooling cycle at 25 °C for 10 s. The results were interpreted from the Cycle threshold (Ct), so that samples that were detected with a Ct value ≤ 39 and showed an S-shaped curve were considered positive for HPV 16/18.

Variables and statistical analysis

The dependent variable, squamous intraepithelial lesions (SIL), was classified as low grade (LSIL) or high grade (HSIL), as previously explained [13]. Through an interview, information was obtained on age, number of sexual partners, age of first sexual relations, toxic habits (alcohol consumption and smoking), parity, use of oral contraceptives, history of sexually transmitted infections, menopausal status, personal pathological history of diabetes mellitus, and high blood pressure.

Body mass index (BMI = [weight (kg)/(height (m))^2] and waist-to-hip ratio (WHR = [waist circumference (cm)/ hip circumference (cm)]) were calculated [18]. The BMI was used for the classification of patients according to WHO, [19] in the following categories: normal weight (18,5 - 24,9 Kg/m^2), overweight (25-29,9 kg/m^2), and obese (30 kg/m^2). Furthermore, abdominal obesity was defined when WHR was ≥ 0.85 [20].

Moreover, prediabetes was defined when fasting blood glucose was altered (5,6 – 6,9 mmol/L), [21] according to the American Diabetes Association, and insulin resistance (IR) was determined by the homeostatic model of Mathews (HOMA-IR) calculated by the following formula: fasting insulin uU/mL x fasting glucose mmol/L/22.5. For adult women, IR was defined when the HOMA-IR value was ≥ 2,6 [22,23]. The presence of dyslipidemia was defined when triglyceride levels were ≥ 1,7 mmol/L, cholesterol levels were ≥ 5,2 mmol/L, or HDL-c levels were < 1,03 mmol/L [24,25].

Given the small sample size and the descriptive, hypothesis-generating nature of this case series, only descriptive statistics were therefore used to summarize the characteristics of the cohort. Mean, standard deviation, and range of age were calculated. Absolute and relative frequencies of variables were determined. Statistical Package for the Social Sciences (SPSS program, version 21) was used for statistical processing.

Ethical considerations

This study adhered to the ethical principles for human research outlined in the Declaration of Helsinki of the World Medical Association [26]. It also complied with the regulations of the Ethics Committee of the Institute of Oncology and Radiobiology. All participants provided informed consent.

Results and discussion

PCOS is associated with metabolic and hormonal alterations that may influence cancer development. Although

evidence for gynecological cancers—except endometrial cancer—is not well established, shared risk factors and biological mechanisms may link PCOS to the development and progression of SIL to cervical cancer. Cervical cancer and its precursor lesions have a multifactorial origin; therefore, risk factor analysis requires a comprehensive approach. Although HPV infection is the main etiological factor, it is insufficient; additional risk factors and comorbidities must contribute to cervical carcinogenesis.

The mean age of patients in the series was 37, 11 ± 12,8 years (range: 21–56 years). Regarding sexual behavior, one patient reported having had more than five sexual partners, another initiated sexual activity before the age of 15, and all reported inconsistent condom use. Those are risky sexual behavior which facilitates the acquisition of sexually transmitted infections such as HPV [27]. Toxic habits such as smoking and alcohol consumption were present in 66,7% (6/9) of patients. A history of hypertension was found in 22,2% (2/9). At the time of the study, two patients (cases 4 and 9) were postmenopausal. None had a prior diagnosis of diabetes mellitus; however, 44,4% (4/9) had elevated fasting glucose levels consistent with prediabetes. Overweight was present in 33,3% (3/9), general obesity in 22,2% (2/9), and abdominal obesity in 55,5% (5/9) (Table 1).

A high proportion of patients reported toxic habits, which are significantly associated with SIL. Smoking is an independent risk factor for cervical cancer, with a meta-analysis reporting a significant association (OR 3,05, 95% CI 1,73–5,38) [28]. Smoking also increases the persistence of high-risk HPV infection [29]. Hypertension was found in only two patients; however, a moderate proportion of women showed prediabetes, a condition frequently observed in women with PCOS. Three of them had HSIL, which, is according to results obtained in a recent research published by Zhang, et al. (2025), who demonstrated that diabetes and prediabetes are associated with high-risk HPV combined with HSIL [30].

Obesity was the most frequent comorbidity. Elevated BMI and WHR, an indicators of abdominal obesity, were observed in more than half of the cases. This aligns with previous findings, as obesity affects over 80% of women with PCOS and exacerbates metabolic disturbances [31]. Abdominal obesity is associated with hypertension, altered glucose metabolism, insulin resistance, and dyslipidemia. It also contributes to chronic low-grade systemic inflammation, creating a tumor-promoting environment [32]. Although evidence is inconclusive, obesity has been associated with SIL development in some populations [33,34].

Table 1: Characteristics of the patients in the study series.

Case	Age (years)	Toxic habits	Hypertension	Prediabetes	BMI	WHR
1	24	No	Yes	No	28,5	0,85
2	21	No	No	No	18,2	0,81
3	31	Yes	No	Yes	30,6	0,92
4	56	Yes	Yes	Yes	21,2	0,93
5	31	Yes	No	No	24,6	0,75
6	46	Yes	No	Yes	25,8	0,85
7	29	No	No	No	36,9	0,95
8	42	Yes	No	No	26,2	0,87
9	54	Yes	No	Yes	23,6	0,91

Legend: BMI: Body Mass Index [normal weight (18,5-24,9 kg/m^2), overweight (25,0-29,9 kg/m^2), and obese (30,0 kg/m^2)]; WHR: Waist-to-hip ratio (abdominal obesity ≥ 0.85)

Table 2 shows serum concentrations of estradiol, testosterone, prolactin, and insulin, as well as HOMA-IR values and dyslipidemia status. Seven patients (77,7%) were normoandrogenic with oligovulation and PCOS, corresponding to phenotype D. Case 1 showed clinical hyperandrogenism (hirsutism), and case 3 showed biochemical hyperandrogenism, both classified as phenotype A.

All patients reported pregnancies or childbirth; only case 7 reported infertility. Two patients had used oral contraceptives for more than five years. Some participants showed elevated prolactin levels (3/9; 33,3%). Hyperinsulinemia and insulin resistance were diagnosed in 55,5% (5/9) and 44,4% (4/9) of cases, respectively, while dyslipidemia was detected in 22,2% (2/9).

Hormonal alterations in PCOS, including hyperandrogenism, are influenced by BMI and WHR. Insulin resistance and hyperinsulinemia associated with abdominal obesity may serve as a metabolic link between PCOS and SIL risk. Insulin stimulates ovarian androgen production via the insulin receptor and IGF-1 receptor in theca cells, while reducing hepatic synthesis of sex hormone-binding globulin, thereby increasing free testosterone. Furthermore, acts as a mitogenic agent in cervical epithelium and, together with IGF-1, may enhance the oncogenic effects of high-risk HPV through activation of the PI3K/Akt/mTOR pathway [35,36].

Hyperandrogenism, a core feature of PCOS in phenotypes A, B, and C, has been implicated in the pathogenesis of cervical cancer through multiple mechanisms. A recent review documents that cervical tissue expresses androgen receptors and that elevated levels of endogenous androgens

Table 2: Results of hormonal analyses, insulin resistance, and dyslipidemia in patients with polycystic ovary syndrome.

Caso	E_2 (pg/mL)	T (nmol/L)	Prl (ng/mL)	Ins (µU/mL)	HOMA-IR	Dyslipidemia
1	28,5	2,9	456,0	30,8	6,0	Yes
2	63,7	2,4	930,0	12,4	2,3	Yes
3	20,0	9,7	225,0	13,8	3,7	No
4	ND	2,9	99,2	11,0	3,0	No
5	60,7	2,4	278,0	6,9	1,7	No
6	141,0	1,1	211,0	9,8	2,5	No
7	98,3	4,0	434,0	15,9	3,7	No
8	91,7	2,1	332,0	13,4	3,2	No
9	ND	0,3	257,0	8,3	2,2	No

Legend: E2: estradiol (Normal Values (NV): premenopausal 51-376 pg/mL, postmenopausal 6-53 pg/mL); T: testosterone (NV: 0,9-4,5 nmol/L); Prl: prolactin (NV: premenopausal 63-425 µIU/mL, postmenopausal 42-319 µIU/mL); Ins: insulin (NV: ≤ 12 µIU/mL); HOMA-IR: Homeostatic Index of Insulin Resistance (NV: < 2,6); ND: Not Detected

have been linked to an increased risk of cervical cancer, affecting proliferation, apoptosis, differentiation, and cellular transformation. Furthermore, the viral oncoproteins E6 and E7 of HPV can directly interact with the androgen receptor, potentiating oncogenic signaling even in the absence of elevated androgen ligands [37].

Prolonged oral contraceptive use may also modulate cervical cellular responses to HPV infection. Although only two patients reported use for more than five years, this practice is common in PCOS management and may increase the risk of progression to cancer in HPV-positive women [38]. Hyperandrogenism itself, or the conversion of androgens into estrogens, exerts a synergistic effect with the virus to promote the progression of lesions in patients with PCOS [39].

The fact that PCOS is a highly heterogeneous entity creates uncertainty in the analysis of the results. Most of the patients in the series presented the normoandrogenic phenotype, which is controversial because it can lead to overdiagnosis of the syndrome or classifying women with other conditions [4]. However, excluding this phenotype can lead to an underestimation of PCOS, especially in its initial stages when hyperandrogenism may be intraovarian and, therefore, not detectable in blood or in its clinical expression [40]. Phenotype D has been associated in recent studies with subtle but significant metabolic alterations, including some degree of insulin resistance and unfavorable lipid profiles [41]. The presence of cervical lesions in this subgroup suggests that even non-hyperandrogenic forms of PCOS may not be exempt from cervical oncogenic risk, possibly mediated by other metabolic factors. In Cuba, according to the study carried out by González, et al. in 2018, different frequencies were described for phenotypes A (28,9%), B (15,8%), and D (55,3%) [42]. In the study conducted by Frontela, et al. in 2022, it was described that total testosterone concentration values above 2 nmol/L are associated with the presence of LSIL, which indicates that such high testosterone concentration values are not required for the initiation of cervical carcinogenesis [10].

Hyperprolactinemia was observed in a subset of patients, consistent with previous reports (~ 30% of PCOS cases). Various pathological conditions, physiological changes, and medication use, among other factors, can cause this increase. Furthermore, 29% of hyperprolactinemia cases in women with PCOS are of idiopathic origin [43]. A 2023 review highlights that Prl not only affects the reproductive axis but also modulates metabolism through its interaction with the pancreas, liver, hypothalamus, and adipose tissue, contributing to the adverse metabolic profile of PCOS [44].

Hyperprolactinemia is relevant to this study because, firstly, it contributes to alterations in the hypothalamic-pituitary-gonadal axis, which exacerbates the reduction of ovarian follicles and the hormonal imbalance that leads to oligo-anovulation in women with PCOS [45]. Secondly, prolactin can exert a direct influence on cervical epithelial cells. In addition to the action of serum prolactin, a recent finding demonstrates that the prolactin receptor increases its expression as the degree of intraepithelial lesions increases. This, coupled with the fact that the transformed cervical cells themselves can produce a 60 kDa prolactin isoform, implies the creation of an autocrine and paracrine signaling circuit that functions independently [46,47].

Prolactin may influence cervical epithelial cells directly and contribute to carcinogenesis through increased receptor expression and autocrine/paracrine signaling loops. This signaling activates the JAK2/STAT pathway, promoting proliferation, inhibiting apoptosis, and enhancing migration and invasion [48]. Additionally, prolactin may interact with estradiol to increase expression of HPV oncogenes E6 and E7 [49,50].

Table 3 summarizes the results of the cytological analyses, as well as the presence of HPV infection. Positivity for HPV genotypes 16/18 is also described. The majority of cases (7/9; 77,7%) presented with HSIL. Among these, cases three and four were negative for viral infection.

Most patients tested positive for HPV 16/18, although two cases were negative. HPV-negative HSIL is rare but recognized. Sometimes, premalignant lesions or even invasive cancer are diagnosed without evidence of viral infection. Worldwide, HPV genotypes 16/18 are found in 3,9% of cytology-negative cases, 25,8% of LSIL cases, 51,9% of HSIL cases, and approximately 70% of cervical cancers [38]. HPV-negative HSIL is possibly due to low viral DNA levels, non-tested genotypes, or HPV-independent oncogenic pathways involving p53, Rb, PI3K/AKT/mTOR, and Wnt/β-catenin signaling [51]. The *"hit-and-run"* hypothesis suggests HPV may initiate transformation and later be cleared, leaving no detectable infection at diagnosis [52].

Recently, a large-scale population-based study using the U.S. National Inpatient Sample (2016-2019), which included over 15 million patients, found that PCOS was significantly associated with endometrial cancer (OR = 3,90), but not with cervical cancer (OR = 0,83; 95% CI: 0,62-1,11; p = 0,218) in multivariate analysis. This finding, seemingly contradictory to this series, warrants careful consideration. First, this work

Table 3: Results of cytological analyses and HPV infections in patients with polycystic ovary syndrome

Case	SIL	HPV	Genotypes
1	High grade	Positive	16/18
2	Low grade	Positive	16/18
3	High grade	Negative	-
4	High grade	Negative	-
5	High grade	Positive	16/18
6	High grade	Positive	16/18
7	High grade	Positive	16/18
8	High grade	Positive	16/18
9	Low grade	Positive	16/18

Legend: SIL: Squamous Intraepithelial Lesion; HPV: Human Papillomavirus.

describes precursor lesions (SIL), not invasive cancer, and therefore may be capturing an earlier stage of carcinogenesis that does not necessarily translate into established cancer. Second, the population-based study adjusts for multiple covariates that could mask specific effects in high-risk metabolic subgroups [53].

This work presents some limitations, such as the small sample size and the absence of a control group, which preclude causal inferences or robust statistical significance analyses. The diagnosis of PCOS, although verified by a specialist, was based on previous studies from other institutions. Likewise, HPV genotyping was limited to types 16/18, without distinction between them and excluding other high-risk genotypes (31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 68) that could be frequent in this series and account for some of the observed lesions.

These findings suggest that women with PCOS, especially those with abdominal obesity, insulin resistance, or hyperprolactinemia, might benefit from closer gynecological surveillance, including periodic cytology and, when indicated, extended HPV genotyping. Prospective studies with control groups and larger samples are needed to confirm these observations and elucidate the underlying molecular mechanisms. Future studies should also explore the potential protective role of metformin and evaluate hormonal and metabolic biomarkers as predictors of SIL in women with PCOS.

Conclusion

The presence of HSIL in more than a third of the women in this case series is compatible with the hypothesis that PCOS, particularly those with insulin resistance, abdominal obesity, or hyperprolactinemia, may act as a multifactorial risk factor for cervical lesions, either independently or synergistically through metabolic and hormonal pathways that interact with HPV. These findings should be interpreted as preliminary observations that warrant confirmation in larger, controlled studies.

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