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Analysis of blood parameters and clinical features in women with pilonidal sinus disease: Is there a link between the disease and polycystic ovary syndrome?

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ABSTRACT

OBJECTIVE: Pilonidal sinus disease (PSD) is a common disorder in the sacrococcygeal region and has a lower incidence in female as compared with male patients. The aim of this study is to evaluate clinical, hematological, biochemical, and hormonal parameters in women with PSD, and to determine whether the disease plays a major role in abnormalities of clinical and laboratory findings. This study also brings to the forefront the issue of the association between PSD and polycystic ovary syndrome (PCOS).

METHODS: The prospective single-center study included women with PSD, and an equal number of healthy women enrolled in the control group (50 women in each arm of the study). Medical history was taken from every patient, and blood tests were performed on all participants. Ultrasound imaging was performed to evaluate the ovaries.

RESULTS: Both groups were matched for age (p=0.124). The prevalence of obesity and dyslipidemia was significantly higher in women with PSD compared to controls (p=0.046, p=0.008, respectively). The right ovary volume was significantly higher in the study group than the control group (p=0.028). The study group had also significantly higher mean levels of neutrophil, C-peptide, and thyroid stimulating hormone (p=0.047, p=0.031, and p=0.048, respectively). The prevalence of PCOS was higher in patients with PSD, but the difference failed to reach statistical significance (32 vs. 22%, p=0.26).

CONCLUSION: Based on the findings of our study, some clinical and blood parameters differed significantly between women with and without PSD. Although the present study revealed that the prevalence of PCOS was not significantly different in women with or without PSD, more comprehensive and prospective studies are required.

Keywords: Hormones; hyperandrogenism; obesity; pilonidal sinus; polycystic ovary syndrome.

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Pilonidal sinus disease (PSD) is an inflammatory condition that most often involves the sacrococcygeal region. It may present in the forms of asymptomatic, acute pilonidal abscess, chronic fistula, or recurrent disease [1]. This disease was first described by Herbert Mayo in 1833 as a "hair-containing sinus" in the sacrococcygeal area of a young woman. In 1880, Hodges coined the term "pilonidal," which is derived from the Latin words "pilus" (hair) and "nidus" (nest) [2]. PSD mainly affects young and active people aged between 15 and 30 years. The average age at diagnosis is 21 in men and 19 in women [3, 4]. Demonstrating that the onset of the disease can be observed before puberty and after the age of 60 years has brought up the role of endocrine hormones in the patho-

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genesis of the disease. The effect of sex hormones on pilosebaceous glands is considered to be one of the reasons why the incidence of disease is most likely to peak at puberty [5]. Other predisposing factors include prolonged sitting, obesity, deep natal cleft, and lack of hygiene [3, 4].

Although the exact etiology of PSD is not yet fully understood, several hypotheses such as being congenital or acquired have been proposed. The disease was believed to be congenital disorder due the emergence of new theories on the studies about the human embryology in the second half of the 19th century [6]. In 1946, Patey and Scarff stated that PSD can be localized in the clefts between the fingers of barbers. They revealed that the disease is caused by the penetration of short, stiff hairs into the subcutaneous tissues, and the development of a foreign body reaction [7]. The theory of Patey and Scarff strongly supported an acquired basis of etiology. In addition, the acquired origin has been further strengthened by the fact that the disease is more common among people with excessive body hair, relapses after excision, and most commonly appears after puberty. According to the acquired theory of Karydakis, PSD occurs as a result of hair density, increased pressure on hair follicles, and continuous microtrauma of the intergluteal sulcus [8]. Nowadays, the most widely accepted understanding is that PSD is acquired.

There is a noted lack of studies in the literature that extensively analyze clinical, hematological, biochemical, and hormonal features of PSD in female patients. Excessive body hair is the main risk factor for the PSD and is common in patients with both polycystic ovary syndrome (PCOS) or PSD, suggesting a possible connection. In addition, the peak incidence of both diseases is during reproductive years [9]. The present study was conducted to assess whether there is a difference in blood parameters and the prevalence of PCOS between women with and without PSD. To the best if our knowledge, there is only one study in the literature that previously reported the relationship between PCOS and PSD. The study by Ugurlu et al. [10] investigated the frequency of PSD in 40 PCOS patients compared to healthy controls, and found a statistically significant increase. On the contrary, our study examined the frequency of PCOS in patients with PSD. The aim of our study is to determine the clinical values and quantitative effects of these important risk factors. Thus, it is aimed to help establish evidence-based counseling for the prevention of the disorders accompanied by the disease, and to prove that PSD is not only a surgical or local disease, but also a systemic disease.

Highlight key points

- The present study is one of the few that investigate the possibility of an interaction between PCOS and PSD.
- The high prevalence of obesity and dyslipidemia was found among women with pilonidal sinus disease.
- It is important to delineate clinical, hematological, biochemical, and hormonal abnormalities accompanying pilonidal sinus disease in order to prevent the disorders accompanied by the disease, provide superior therapeutic strategies, and guarantee improved quality of life for women with the disease.

MATERIALS AND METHODS

The study was performed in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the local ethics committee for this prospective study with the decision number 217 dated February 14, 2022. Informed consent was obtained from all individual participants included in the study. A total of 100 women, 50 patients with PSD and 50 controls, who applied to the general surgery outpatient clinic between February 15, 2022, and April 20, 2022, were prospectively analyzed. The study group comprised of women with PSD between the ages of 18 and 45. The control group was selected from healthy volunteers matched for age and gender. Patients with PSD and healthy controls were classified as Group 1 and Group 2, respectively. Congenital adrenal hyperplasia, Cushing's syndrome, diseases that cause hyperprolactinemia, known thyroid abnormality, diabetes mellitus, metabolic syndromes, medications affecting lipid, and hormone metabolism (fibrates, statins, glucocorticoids, retinoids, immunosuppressive drugs, etc.), pregnancy, alcohol use, and drug addiction were determined as exclusion criteria for the cases and controls.

Demographic data, additional comorbidities, body mass index (BMI), menstrual irregularity, clinical signs of hyperandrogenism (unusual acne, hirsutism or androgenetic alopecia), tobacco use, radiological, and laboratory data were recorded. Venous blood samples were drawn for measurement of hematological parameters, glucose, insulin, C-peptide, lipid profile (total cholesterol [TC], high density lipoprotein cholesterol [HDL-C], low density lipoprotein cholesterol [LDL-C], triglyceride [TG]), dehydroepiandrosterone sulfate [DHEAS], estradiol, progesterone, testosterone, cortisol, follicle-stimulating hormone [FSH], luteinizing hormone [LH], prolactin [PRL], thyroid function tests (thyroid stimulating hormone [TSH], free thyroxine [fT4], and free tri-iodothyronine [fT3]) in the morning time between 8.00 and

Table 1. Characteristics of study population

	Groups		p
	Group 1 (n=50)	Group 2 (n=50)	
Total number of patients	n	50	50
Age (year)			0.124
Mean±SD	25.36±7.07	26.42±5.83	
Median (Min–Max)	23 (18–45)	26 (18–40)	
Height (cm)			**0.035**
Mean±SD	164.24±4.66	162.5±5.63	
Median (Min–Max)	163.5 (154–175)	162 (150–183)	
Weight (kg)			**<0.001**
Mean±SD	67.14±13.04	58.24±10.14	
Median (Min–Max)	65.5 (42–95)	57.5 (40–95)	
BMI (kg/m^2)			**0.001**
Mean±SD	24.8±4.2	22.05±3.7	
Median (Min–Max)	24.95 (16.4–33.7)	21.4 (16–37.1)	
Obesity (%)			**0.046**
Yes	16	4	
No	84	96	
Smoking (%)			0.806
Yes	22	20	
No	78	80	
Treatment (%)			
Surgery	18		
Phenol application	62		
Conservative treatment	20		

SD: Standard deviation; Min: Minimum; Max: Maximum; BMI: Body mass index.

9.00 am after an overnight fasting for at least 8 h. Weight and height measurements were taken, and BMI was calculated as the ratio of weight/height^2 (kg/m^2). Obesity was defined as a BMI of 30 kg/m^2 or higher [11]. homeostatic model assessment for insulin resistance (HOMA-IR) is a method used to quantify insulin resistance and beta-cell function. The HOMA-IR index was calculated from the formula: Fasting insulin (ulU/mL)×Fasting glucose (mg/dL)/405. The threshold value of insulin resistance was accepted as HOMA-IR ≥2.1 in non-diabetic individuals [12]. Dyslipidemia was identified as serum levels of TG above 150 mg/dL, LDL-C above 100 mg/dL, and HDL-C of <50 mg/dL in females according to American diabetes association guidelines [13]. Subclinical hypothyroidism was defined as a normal serum fT4 and fT3 concentrations in the presence of an elevated serum TSH concentration [14].

The presence of at least two of the three criteria, including oligo-anovulation, clinical and/or biochemical hyperandrogenism, and polycystic ovaries on ultrasound (US) examination is sufficient for the diagnosis of PCOS according to the Rotterdam criteria [15]. Other possible causes that mimic clinical features of PCOS were excluded before the diagnosis was established. The Ferriman-Gallwey scale was used to assess clinical hyperandrogenism, and a total score of 8 or more was considered diagnostic of hirsutism [16]. Biochemical hyperandrogenism was defined as a total testosterone level >80 ng/dL or DHEAS level >350 ng/dL [16]. The detailed assessment of ovarian morphology and volume was based on transabdominal US examination performed between days 6 and 8 of the menstrual cycle by an experienced radiologist. Radiological characteristics of polycystic ovaries included either 12 or more peripheral follicles in an ovary with a diameter of 2–9 mm and/or increased ovarian volume [17].

Table 2. Comparison of ovarian volume and diagnostic criteria for polycystic ovary syndrome between the groups

	Groups		p
	Group 1 (n=50)	Group 2 (n=50)	
Menstrual irregularity (%)			1.000
Yes	40	40	
No	60	60	
Polycystic morphology (%)			0.617
Yes	22	18	
No	78	82	
Right ovary volume, (cm^3)			**0.028**
Mean±SD	11.63±11.11	7.9±3.68	
Median (Min–Max)	8.1 (3.4–62)	7.1 (2.5–17)	
Left ovary volume, (cm^3)			0.057
Mean±SD	9.93±4.48	8.82±5.39	
Median (Min–Max)	9.25 (2.4–24)	7.65 (2.4–36)	
Clinical hyperandrogenism (%)			0.829
Yes	32	30	
No	68	70	
Biochemical hyperandrogenism (%)			0.617
Yes	22	18	
No	78	82	
Polycystic ovary syndrome (%)			0.260
Yes	32	22	
No	68	78	

SD: Standard deviation; Min: Minimum; Max: Maximum.

Statistical Analysis

We used the SPSS program (SPSS version 25.0, SPSS, Inc. Chicago, IL, USA) for statistical assessment of the data. We applied descriptive statistical techniques (mean, standard deviation, median, frequency, percentage, minimum, and maximum) for the evaluation of the data. The normal distribution of the variables was assessed with Kolmogorov–Smirnov test. Pearson's Chi-square test was used to compare the categorical variables. The Mann–Whitney U-test was used to compare two groups of data that were not normally distributed. Statistical significance was accepted as $p<0.05$.

RESULTS

Total of 50 subjects diagnosed with PSD and 50 controls were assessed in the recent study. Table 1 presents the baseline characteristics of study groups. The mean age of the subjects was 25.36±7.07 (18–45) years in Group 1, and 26.42±5.83 (18–40) years in Group 2. The rate of tobacco use was 22% in Group 1, and 20% in Group 2. There were no significant differences in age and tobacco use between the groups ($p=0.124$ and $p=0.806$, respectively). The mean value of BMI was 24.8±4.2 kg/m^2, and 16% of the participants were obese in group 1. The mean value of BMI was 22.05±3.7 kg/m^2, and 4% of the participants were obese in Group 2. Our findings revealed that Group 1 had significantly higher BMI, and obesity prevalence than Group 2 ($p=0.001$ and $p=0.046$, respectively).

Both groups were compared with each other in terms of oligo-anovulation, clinical and/or biochemical hyperandrogenism, and polycystic ovary morphology, which are the criteria for diagnosis of PCOS (Table 2). There was no statistically significant difference regarding the prevalence of menstrual disorders between the groups ($p=1.000$). Both groups suffered equally from menstrual disorders, such as infrequent, irregular, or

Table 3. Hematological parameters of the study group compared with the control group using Mann–Whitney U-test

	Groups		p
	Group 1 (n=50)	Group 2 (n=50)	
White blood cells, $\times 10^9$/L			0.106
Mean±SD	7.43±1.85	6.83±1.91	
Median (Min–Max)	7.15 (3.5–11.9)	6.85 (3.6–12.4)	
Neutrophils, $\times 10^9$/L			**0.047**
Mean±SD	4.72±1.71	4.2±1.61	
Median (Min–Max)	4.35 (1.7–9.7)	3.99 (1.83–9.2)	
Lymphocytes, $\times 10^9$/L			0.852
Mean±SD	1.86±0.76	1.82±0.69	
Median (Min–Max)	1.9 (0.32–4.2)	2 (0.3–3.2)	
Monocytes, $\times 10^9$/L			0.500
Mean±SD	0.67±0.34	0.63±0.34	
Median (Min–Max)	0.57 (0.24–2.38)	0.6 (0.29–2.5)	
Eosinophils, $\times 10^9$/L			0.303
Mean±SD	0.11±0.11	0.12±0.08	
Median (Min–Max)	0.1 (0–0.7)	0.1 (0–0.4)	
Basophils, $\times 10^9$/L			0.704
Mean±SD	0.05±0.06	0.04±0.05	
Median (Min–Max)	0.04 (0–0.4)	0.03 (0–0.3)	
Red blood cells, $\times 10^{12}$/L			0.912
Mean±SD	4.67±0.39	4.62±0.3	
Median (Min–Max)	4.61 (4–6.45)	4.59 (4.06–5.19)	
Hemoglobin, g/dL			0.855
Mean±SD	13.29±1.1	13.23±1.28	
Median (Min–Max)	13.4 (9.8–15.1)	13.4 (9.7–15.3)	
Hematocrit, %			0.702
Mean±SD	39.5±2.79	39.21±3.22	
Median (Min–Max)	39.75 (30.9–44.9)	39.55 (30.2–45.1)	
MCV, fL			0.877
Mean±SD	84.92±6.59	84.83±6.06	
Median (Min–Max)	85.8 (54.1–94.5)	86 (70.4–97.7)	
MCH, pg			0.785
Mean±SD	28.59±2.66	28.63±2.53	
Median (Min–Max)	29.15 (16.7–32.6)	29.4 (23.1–33.7)	
MCHC, g/dL			0.801
Mean±SD	33.63±0.96	33.71±0.81	
Median (Min–Max)	33.6 (30.9–35.4)	33.8 (31.9–35.3)	
Red cell distribution width, %			0.788
Mean±SD	14.16±2.11	13.89±1.14	
Median (Min–Max)	13.6 (12.3–24.3)	13.65 (12–16.6)	
Platelet count, 10^9/L			0.118
Mean±SD	288.56±61.33	272.56±67.96	
Median (Min–Max)	278 (174–445)	253.5 (155–476)	
Mean platelet volume, fL			0.452
Mean±SD	8.48±0.88	8.55±0.77	
Median (Min–Max)	8.35 (7–11.4)	8.55 (6.7–9.9)	
Platelet distribution width, %			0.621
Mean±SD	16.67±1.04	16.77±0.43	
Median (Min–Max)	16.7 (10.3–18.2)	16.8 (15.9–17.8)	

SD: Standard deviation; Min: Minimum; Max: Maximum; MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; MCHC: Mean corpuscular hemoglobin concentration.

Table 4. Plasma glucose, insulin, and C-peptide parameters of groups

	Groups		p
	Group 1 (n=50)	Group 2 (n=50)	
Fasting blood glucose level (mg/dL)			0.320
Mean±SD	90.78±15.91	87.14±9.47	
Median (Min–Max)	88.5 (62–157)	85 (67–116)	
Insulin (mIU/L)			0.149
Mean±SD	12.65±10.19	10.07± 8.02	
Median (Min–Max)	8.83 (2.37–54.78)	7.61 (2.09–40.9)	
C–peptide (ng/mL)			**0.031**
Mean±SD	2.62±1.62	2.07±1.22	
Median (Min–Max)	2.2 (0.84–9.36)	1.6 (0.71–6.59)	
HOMA–IR			0.107
Mean±SD	2.93±2.59	2.27± 2.08	
Median (Min–Max)	1.83 (0.42–12.44)	1.54 (0.39–9.69)	
Insulin resistance (%)			0.300
Yes	42	32	
No	58	68	

HOMA-IR: Homeostatic model assessment for insulin resistance; SD: Standard deviation; Min: Minimum; Max: Maximum.

prolonged menstrual periods; the prevalence of menstrual disturbances was 40%. The frequencies of clinical and biochemical hyperandrogenism were evaluated between the groups, and no significant difference was found (p=0.829, p=0.617, respectively). The presence of polycystic ovary appearance on US scan was detected in 11 cases in Group 1, and in nine cases in Group 2 (p=0.617). The mean volumes of the right and left ovaries for PSD patients were 11.63±11.11 and 9.93±4.48 cm^3, respectively, and the number of PCOS cases was 16. The mean volumes of the right and left ovaries for controls were 7.9±3.68 and 8.82±5.39 cm^3, respectively, and the number of PCOS cases was 11. Among these, only the difference between the groups for right ovarian volume was statistically significant (p=0.028). Although left ovary volume was found to be slightly higher in Group 1 compared to Group 2, the difference failed to reach statistical significance (9.93±4.48 cm^3 vs. 8.82±5.39 cm^3, p=0.057).

Hematologic parameters of patients and controls are depicted in Table 3. Compared to group 2, the neutrophil count was significantly elevated in Group 1 (p=0.047). We noted no significant differences in other hematologic parameters tested.

Fasting plasma levels of glucose, insulin, and C-peptide were tested (Table 4). Fasting blood glucose and insulin levels were found to be higher in Group 1, which was not statistically significant (p=0.320, and p=0.149, respectively). Group 1 had significantly higher C-peptide levels than group 2 (p=0.031). Both groups did not differ significantly in terms of HOMA-IR levels and the prevalence of insulin resistance (p=0.107 and p=0.300, respectively).

In the analysis of lipid profile, no significant difference was observed between the groups according to TG, TC, and LDL-C parameters (p=0.123, p=0.128, and p=0.257, respectively). However, we found significantly lower mean concentrations of HDL-C in Group 1 (p=0.004). A condition of dyslipidemia was found in 72% of Group 1 versus 46% of Group 2 (p=0.008) (Table 5).

The results and statistical data of hormonal parameters are shown in Table 6. There were no significant differences in hormonal parameters concerning DHEAS, estradiol, progesterone, testosterone, cortisol, LH, FSH, LH/FSH ratio, and PRL. On the evaluation of thyroid profile, significantly higher TSH levels were found in PSD subjects compared to con-

Table 5. Descriptive statistics and mean comparisons of lipid profile for Groups 1 and 2

	Groups		p
	Group 1 (n=50)	Group 2 (n=50)	
Triglyceride (mg/dL)			0.123
Mean±SD	105.02±66.47	85.62±39.83	
Median (Min–Max)	90.5 (36–414)	75.5 (31–240)	
Total cholesterol (mg/dL)			0.128
Mean±SD	161.58±38.94	175±30.81	
Median (Min–Max)	162 (75–254)	171 (127–251)	
LDL–C (mg/dL)			0.257
Mean±SD	93.1±30.3	102.74±26.69	
Median (Min–Max)	95.5 (30–158)	104.5 (55–171)	
HDL–C (mg/dL)			**0.004**
Mean±SD	48.7±10.33	55.1±10.88	
Median (Min–Max)	47.5 (26–74)	55 (32–83)	
Dyslipidemia (%)			**0.008**
Yes	72	46	
No	28	54	

LDL–C: Low density lipoprotein cholesterol; HDL–C: High density lipoprotein cholesterol; SD: Standard deviation; Min: Minimum; Max: Maximum.

trols (p=0.048). No significant difference in fT4 and fT3 levels between the two groups was determined (p=0.553, p=0.317, respectively). None of the cases in both groups had subclinical hypothyroidism.

DISCUSSION

PSD is a chronic inflammatory disease usually located in the intergluteal fold. The disease predominantly affects young adults and is approximately 3–4 times more prevalent in males than females [18, 19]. Females commence puberty at a younger age than males, and therefore, they are more likely to develop PSD at an earlier age than males [20]. Body hair type, sedentary lifestyle, obesity, repetitive local trauma, and poor hygiene facilitate the development of the disease [3, 4]. Although a genetic susceptibility has not been determined, a family history of PSD predisposes to earlier onset of the disease, and higher rates of recurrence after surgical interventions [21]. Risk factors such as genetics, age, gender, or anatomical characteristics cannot be altered; however, if the predisposing factors and high-risk patient groups are known, it may be beneficial to change all modifiable risk factors or to apply the basic preventive measures before the disease process begins. Excessive body hair is the main predisposing agent in all theories that attempt to explain the pathogenesis of PSD, which may be the reason for the male predomination of the disease [19]. On the other hand, predisposing factors in female gender, where hirsutism is less common, are still a matter of debate. Therefore, the focus of the present study is clinical, hematological, biochemical, and hormonal features in female patients with PSD.

We examined 50 PSD and 50 control female subjects for various clinical, hematological, biochemical, hormonal, and radiological parameters. In this study, the mean age of PSD patients was 25.36±7.07 years, which is consistent with the literature [3, 18]. Obesity has been reported as a significant risk factor for PSD, and to be associated with higher rates of post-operative complications and recurrences compared to normal-weight individuals [22]. Weight gain may pave the way for the disease development by deepening of the gluteal sulcus, and increasing the trauma to the hair follicles. In our study, the mean BMI value of the subjects in the control group was 22.05±3.7 kg/m^2, which was significantly lower than the mean BMI value of the patient group (24.8±4.2 kg/m^2) (p=0.001). A Mann–Whitney U test revealed significant differences in obesity prevalence between the

Table 6. Comparative description of hormonal parameters between groups using Mann–Whitney U–test

	Groups		p
	Group 1 (n=50)	Group 2 (n=50)	
DHEAS (µg/dL)			0.250
Mean±SD	257.58±99.36	246.23±138.14	
Median (Min–Max)	246.1 (98.1–535.1)	225.7 (44.3–644.8)	
Estradiol (pg/mL)			0.152
Mean±SD	81.56±76.73	95.92±85.86	
Median (Min–Max)	61.27 (15–472.94)	70.84 (17.69–525.49)	
Progesterone (ng/mL)			0.312
Mean±SD	2.96±3.94	4.24±5.94	
Median (Min–Max)	0.98 (0.08–13.48)	1.17 (0.08–25.08)	
Testosterone (pg/mL)			0.603
Mean±SD	50.32±21.3	51.08±31.05	
Median (Min–Max)	46.5 (16–111)	44 (10–173)	
Cortisol (mcg/dL)			0.215
Mean±SD	12.03±5.01	10.81±4.56	
Median (Min–Max)	11.98 (5.32–24.45)	9.61 (3.43–23.65)	
FSH (mIU/mL)			0.863
Mean±SD	5.9±2.83	5.94±3.02	
Median (Min–Max)	5.82 (1.03–15.72)	5.81 (0.75–15.72)	
LH (mIU/mL)			0.100
Mean±SD	10.62±8.96	8.7±8.14	
Median (Min–Max)	7.68 (0.41–45.96)	6.02 (0.35–34.81)	
LH/FSH ratio			0.201
Mean±SD	1.91±1.59	1.49±1.04	
Median (Min–Max)	1.6 (0.37–8.38)	1.14 (0.16–4.51)	
PRL (ng/mL)			0.104
Mean±SD	17.87±15.1	15.85±15.14	
Median (Min–Max)	14.5 (5.48–84.09)	10.68 (2.62–83.51)	
TSH (mU/L)			0.048
Mean±SD	2.1±0.97	1.75±0.88	
Median (Min–Max)	2.07 (0.52–4.72)	1.52 (0.59–4.81)	
fT4 (ng/dL)			0.553
Mean±SD	0.87±0.12	0.85±0.09	
Median (Min–Max)	0.85 (0.64–1.31)	0.86 (0.58–1.07)	
fT3 (ng/dL)			0.317
Mean±SD	3.51±0.43	3.43±0.34	
Median (Min–Max)	3.47 (2.74–4.63)	3.4 (2.63–4.34)	

DHEAS: Dehydroepiandrosterone sulfate; FSH: Follicle–stimulating hormone; LH: Luteinizing hormone; PRL: Prolactin; TSH: Thyroid stimulating hormone; fT4: Free thyroxine; fT3: Free tri-iodothyronine; SD: Standard deviation; Min: Minimum; Max: Maximum.

groups (p=0.046). However, a retrospective analysis reported by Cubukcu et al. [23] showed no significant difference in BMI values between women with PSD and controls. Although there are many studies reporting that PSD may be associated with obesity, there are also studies reporting that there is no relationship [22, 23]. Despite the different results in the literature, obesity is generally accepted to be a prominent risk factor for PSD.

Serum HDL-C levels tend to be lower in smokers than in non-smokers [24]. Although no significant difference was observed in current smoking prevalence among our study groups, HDL-C levels were found to be significantly lower in the PSD group than in the control group (p=0.004). PSD is a chronic skin infection that can cause irritation, infection, and abscess formation. Neutrophils are a type of white blood cells mostly target bacterial infections [25]. As expected, neutrophil levels were significantly higher in the group of women with PSD (p=0.047). Significant alterations in lipid metabolism and lipoprotein composition occur during inflammation and infection. HDL-C and LDL-C levels reduce while TG levels rise [26]. Significantly lower (p=0.004) levels of HDL-C in PSD subjects compared with controls were reported in our study. The study results also demonstrated that PSD subjects had higher levels of TG, and lower levels of LDL-C compared to controls, but statistical significance was not obtained (p=0.123, p=0.257, respectively). From this point of view, dyslipidemia might be correlated with inflammation or infection of an existing PSD.

Thyroid function tests of the study population were also assessed in the study. The levels of TSH were found to be higher in females with PSD. This notable finding may be due to the fact that the patient group had significantly higher BMI, and obesity prevalence than the control group (p=0.001 and p=0.046, respectively). Overweight and obesity result in alterations in thyroid function. Our findings support the previous reports that obese patients have slightly higher levels of TSH as compared to patients within the normal weight range [27, 28].

PCOS is an endocrine disorder affecting 12–21% of women of childbearing age, but the prevalence varies according to the diagnostic criteria used [29]. PCOS may be accompanied by several metabolic disorders, including obesity, insulin resistance, impaired glucose tolerance, diabetes mellitus, and dyslipidemia [30, 31]. The present study showed that women afflicted by PSD had slightly increased prevalence of PCOS, although the difference in outcomes did not reach statistical significance (p=0.26). The prevalence of insulin resistance tended to be higher in the patient group, but there was no significant difference between the two groups (p=0.3). Similarly, the mean values of fasting blood glucose, insulin, C-peptide, and HOMA-IR were higher in the patient group, but only C-peptide value reached statistical significance (p=0.031). These hormonal disturbances might be a result of the slightly increased frequency of PCOS in the patient group [9].

Many studies have investigated the size differences of ovaries [32]. Our results showed that the right ovarian volume was found to be significantly increased in women with PSD compared to control group (p=0.028). Although the patient group had higher mean volume of the left ovaries, but it was at the verge of statistical significance (p=0.057). The difference in ovarian volume between the groups is likely to be explained by the more common presence of PCOS in women with PSD, and the presence of more antral follicles on the right side [10, 32].

The quality of life of the patient is negatively affected by the side-effects that result both from the illness itself and as a consequence of treatment. The disease may lead to absenteeism at work, job losses, and long-term social disconnection [33]. Thus, preventive measures, non-operative, or minimally invasive methods have been more preferred in recent years. In general, minimally invasive surgery provides better quality of life for patients, and enables patients to return to a state close to healthy normal individuals in a short period of time. Asymptomatic pits of the gluteal cleft do not require any specific therapy in children and young people. It can be sufficient to comply with the rules of personal hygiene, and to remove hair regularly from the gluteal cleft. If there are signs and symptoms of an infection, antibiotics are used and a warm compress is recommended on a pilonidal cyst to support blood circulation [34, 35]. Surgery was performed in nine cases, phenol application in 31 cases, and conservative treatment (meticulous hair removal by natal cleft shaving, improved natal-cleft hygiene, weight loss, and drainage for abscess) in 10 cases in the study group. These results also show that minimally invasive techniques and conservative approaches are preferred more than surgical excision methods.

The main weakness of the present study is the small number of patients included in the study. Second, this study lacks analysis of other known risk factors playing a role in PSD, including prolonged sitting, natal cleft depth, sweating, poor hygiene, and genetic predisposition. On the other hand, selecting patients of the same age and gender, and excluding patients with diseases that may affect the hormonal and metabolic profile allow our analysis to be interpreted with confidence.

Conclusion

This study discussed only some from a possible large number of parameters differed significantly between women with and without PSD. The present study confirmed a significantly high prevalence of obesity and dys-

lipidemia in women with PSD. Although the difference was not significant, the incidence of PCOS appeared to be slightly higher among women with PSD. In light of the controversial considerations regarding the characteristics of the disease, further studies are needed to elucidate the intricacies of the associations between the disease and PCOS. Given these serious consequences, it is important to thoroughly understand clinical, hematological, biochemical, and hormonal abnormalities accompanies PSD to provide superior therapeutic strategies, and guarantee improved quality of life for women with the disease.

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