Original paper

Selected hormone levels and lipid abnormalities in patients with *acne vulgaris*

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Abstract

Introduction: *Acne vulgaris* is one of the most common dermatological diseases. Hormonal imbalance affects the skin condition and results in the formation of *acne vulgaris* lesions.

Aim: To evaluate serum levels of testosterone, prolactin, luteinizing hormone (LH), follicle-stimulating hormone (FSH), triglycerides (TG), and high-density lipoprotein (HDL) in patients with *acne vulgaris* and compare them to healthy population.

Material and methods: Forty-one patients with *acne vulgaris* and 47 age- and body mass index (BMI)-matched controls were enrolled in the study.

Results: The mean ± SD testosterone serum level in the study group was 0.45 ±1.03 ng/ml in females and 4.24 ±0.68 in males and in the control group 0.73 ±2.03 ng/ml and 5.3 ±1.3 ng/ml in females and males, respectively. The prolactin serum level was 16.73 ±8.02 ng/ml in the study group and in the control group 13.74 ±8.71 ng/ml (p = 0.011). The FSH serum level was 12.17 ±16.93 mIU/ml and 6.2 ±7.3 mIU/ml in the study and control groups, respectively (p = 0.0001), whereas LH serum levels were 18.44 ±19.71 mIU/ml and 11.26 ±8 mIU/ml, respectively (p = 0.2659). The HDL serum level was 65.63 ±15.67 mg/dl in the study group and 61.53 ±15.89 mg/dl in the control group (p = 0.219), and TG levels were 175.29 ±82.15 mg/dl and 87.32 ±30.64 mg/dl, respectively (p < 0.00001).

Conclusions: Our study demonstrates, that hormonal and lipid imbalance could be linked to *acne vulgaris* formation. Evaluation of hormonal and lipid abnormalities could help in treatment decisions and could affect the occurrence of complications and the course of acne.

Key words: acne vulgaris, hormone profile, lipid profile.

Introduction

Acne vulgaris is one of the most common dermatological diseases in adolescent and adult populations [1]. Puberty is a period when the first symptoms of *acne* often appear [2]. The first acne lesions, like comedones, papules, pustules, or nodules [3], occur at the age of 11–12 and affect 85% of adolescents [4]. Puberty is a period of numerous physiological changes in the human organism, including not only psycho-social maturation but also multiple changes in the hormonal profile which lead to sexual maturation [2]. However, puberty is controlled not only by sex hormones – oestrogen, progesterone, and testosterone, but also by growth hormone, thyroid hormones, and many more [5, 6]. Currently, the first symptoms of acne are being observed more and more often in younger children (6–7 years old). This may be related to the acceleration of puberty [7].

Hormonal imbalance in both adolescent and adult people affects the skin condition and results in the formation of *acne vulgaris* lesions, which are associated with abnormalities of sebaceous glands. These glands are closely linked to hormonal biosynthesis. The conversion of 17-hydroxyprogesterone straight to dihydrotestosterone with the avoidance of testosterone has been demonstrated. What is more, in sebaceous glands both testosterone and dihydrotestosterone are inactivated by 17B-hydroxysteroid

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dehydrogenase [8, 9]. Androgens regulate the synthesis of sebum and influence inflammatory processes in sebaceous glands affected by *acne vulgaris* [9].

The sebaceous gland cells demonstrate the expression of receptors for sex hormones – not only androgens, but also for progesterone and oestrogen [10, 11]. Oestrogen application also reduces the severity of acne [12, 13]. Moreover, fluctuations in the severity of acne lesions during the menstruation cycle in girls and women indicate the significant role of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) in sebum production, skin immune activity, and pathogenesis of acne. Increased sebum production and vascularity during the second phase of the menstrual cycle are most probably correlated with a high serum level of progesterone and a decreased serum level of oestrogen [14–16].

In addition to the imbalance in hormone levels, abnormalities in the lipid profile also seem to be associated with the development of acne lesions. Metabolic disorders and lipid peroxidation may influence the inflammatory processes and immune response underlying the pathogenesis of *acne vulgaris.* Moreover, women with the irregular cycle and hyperandrogenaemia are at risk of metabolic disorders like elevated serum levels of triglycerides or the occurrence of insulin resistance, which indicates a relationship between hormonal and lipid imbalance [1].

Many correlations between *acne vulgaris* and activity of the endocrine system and metabolic disorders have been demonstrated, which indicates the influence of different hormones and lipid imbalances on the pathogenesis and the course of acne. However, there are few studies that present a disparity of hormone and lipid profiles in patients affected by *acne vulgaris*.

Aim

The aim of our study was to evaluate selected hormone serum levels: testosterone, prolactin, LH, FSH, and selected lipid parameters: triglycerides (TG) and high-density lipoprotein (HDL) in patients with *acne vulgaris* and compare them to hormone and lipid profiles in a healthy population.

Material and methods

Participant recruitment

This study was conducted in the outpatient Department of Dermatology and Venereology of the Poznan University of Medical Sciences in Poland.

The inclusion criteria for all participants were age range of 18–65 and both female and male patients. The study group inclusion criterion was an *acne vulgaris* diagnosis. The study group was composed of 41 patients with *acne vulgaris* – 38 (93%) females and 3 (7%) males, with mean ± SD age of 30.6 ±10.6. In the control group, 47 healthy people were enrolled, of whom 24 (51%) were females and 23 (49%) were males, with mean ± SD age of 25.9 ±3.9. Each participant gave informed consent to be enrolled in the study. The study was approved by the Ethics Committee of the Poznan University of Medical Sciences in Poland.

Exclusion criteria for the study group were the presence of any hormonal disorders, including:

- Polycystic ovary syndrome,
- Thyroid dysfunction,
- Cushing syndrome,
- Congenital adrenal hyperplasia, diabetes mellitus (or family history of DM),
- Any hormonal treatment, including hormonal contraceptive therapy,
- High intake of high glycaemic-load carbohydrates, milk, and dairy products.

Exclusion criteria for the control group included:

- The presence of any chronic diseases, including *acne vulgaris* or any chronic treatment,
- High intake of high-glycaemic-load carbohydrates, milk, and dairy products,

Each participant was asked to complete a questionnaire on general health information, physical activity, and medication. The Investigator's Global Assessment Scale was used to assess the severity of *acne vulgaris*.

Subsequently, a physical examination was performed which included measurements of height, weight, and blood pressure. In both groups, body mass index (BMI) was assessed using the formula: weight [kg]/height^2 [m^2]. The results were categorized as normal (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obese (> 30 kg/m^2).

The results of the physical examination are presented in Table 1.

There were no significant differences between the study and the control groups in terms of weight, systolic blood pressure (SBP), and DBP. There was a significant difference in height between both groups. Based on measurements of weight and height, BMI was calculated for each participant. The differences of BMI values between both groups were close to statistical significance (p = 0.056).

Laboratory examination

The blood samples from the peripheral venous blood were collected from each participant. Testosterone, prolactin, LH, and FSH serum levels were evaluated using the ELISA method. The HDL and TG serum levels were evaluated in the hospital's central laboratory. Normal reference ranges for the hormones and lipid parameters used in our study method are presented in Table 2.

Statistical analysis

The statistical analysis was performed using IBM SPSS Statistics 29.0.0.0.

Table 1. Physical examination – weight, height, SBP, DBP, BMI as mean ± SD

Parameter	Study group	Control group	*P*-value
Age [years] mean ± SD	30.6 ±10.6	25.9 ±3.9	0.465
Sex, *n* (%):			
Female	37 (90%)	24 (51%)	
Male	4 (10%)	23 (49%)	
Weight [kg]	71.05 ±11.55	69.74 ±12.01	0.907
Height [m]	1.70 ±0.06	1.74 ±0.09	0.032*
SBP [mm Hg]	121.07 ±10.82	120.23 ±10.04	0.707
DPB [mm Hg]	72.15 ±8.78	73.43 ±8.52	0.402
BMI [kg/m^2]	24.49 ±3.83	22.9 ±2.56	0.056

**Statistical significance.*

Quantitative variables: for the interval scale with normal distribution the Student's *t*-test (*t*-st) or its Cochran-Cox correction (C-C) was used when the group variances differed. For the interval scale, when the condition of normal distribution was not met, the Mann-Whitney (M-W) test was used, similarly to the ordinal scale. The normality of the data distribution was tested with the Shapiro-Wilk test and the equality of variances with the Fisher-Snedecor (F-S) test. If no scale was marked for the variables being analysed, it was assumed that the data came from an interval scale.

Qualitative variables: for the nominal scale, the chi-square test score (χ^2) was determined, and when the Cochran condition was not met, Fisher's exact test (Fisher exact) or, in the case of 2x2 tables with a sample size greater than 40, the Yates correction (χ^2-Yates). For the ordinal scale, the c^2 test for the trend was used. If no scale was marked for the variables analysed, it was assumed that the data came from the nominal scale.

Results

Forty-one *acne vulgaris* patients and 47 healthy controls were enrolled in our study. Both groups were matched by age ($p = 0.465$). A physical examination was performed for each participant.

The testosterone, prolactin, FSH, and LH serum levels were obtained from each participant. Statistical comparison of serum levels of hormones revealed a significant difference in the testosterone serum level, prolactin serum level, and FSH serum level between both groups. The mean ± SD testosterone serum level was lower in the group of patients with *acne vulgaris* (0.82 ±1.18 ng/ml) than in the control group (2.97 ±2.49 ng/ml). The mean ± SD prolactin serum levels were statistically higher in the study group (16.73 ±8.02 ng/ml) in comparison with the control group (13.74 ±8.71 ng/ml). The mean ± SD FSH serum levels were higher in acne patients (12.17 ±16.93 mIU/ml) compared to the controls (6.2 ±7.28 mIU/ml).

Moreover, the mean ± SD HDL serum level was higher in the study group than in the controls with no statistical difference between the groups ($p = 0.219$). Statistical analysis revealed significantly higher levels of TG in patients with acne (175.29 ±82.15 mg/dl) compared to the control group (87.32 ±30.64 mg/dl). All the results are presented in Table 2.

Considering the difference in hormone levels between female and male patients, we decided to evaluate mean ± SD levels of hormones and lipid parameters for both sexes separately in the study group and the control group. The results for healthy controls and the subjects are presented in Table 3.

Table 2. Comparison of test results in the control group and study group

Variable	Group	*N*	Mean	SD	Median	Q1; Q3	Min.	Max.	*P*-value
Testosterone serum level	Study	41	0.82	1.18	0.44	0.23; 0.78	0.052	5.126	0.00001*
	Control	47	2.97	2.49	1.6	0.71; 5.08	0.391	8.130	
Prolactin serum level	Study	41	16.73	8.02	15.84	11.05; 21.06	2.547	33.135	0.019*
	Control	47	13.74	8.71	11.83	8.34; 16.44	4.365	43.258	
FSH serum level	Study	41	12.17	16.93	8.3	5.7; 12.4	1.08	23.419	0.00011*
	Control	47	6.2	7.28	3.54	1.2; 7.38	0.157	22.731	
LH serum level	Study	41	18.44	19.71	10.1	6.8; 20.4	1.3	74.9	0.266
	Control	47	11.26	8	9.15	6.03; 13.71	1.987	36.542	
HDL	Study	41	65.63	15.67	64	52; 76	43	95	0.219
	Control	47	61.53	15.89	62	49.5; 71.5	39	117	
TG	Study	41	175.29	82.15	154	118; 235	35	332	< 0.00001*
	Control	47	87.32	30.64	80	66; 101.5	45	199	

**Statistical significance.*

Table 3. Test results in the control group and study group – comparison between female and male patients

Variable	Group (Normal range)	*N*	Mean	SD	Median	Q1; Q3	Min.	Max.	*P*-value
Control group:									
Testosterone serum level	Female (0.26–1.22 ng/ml)	24	0.73	0.28	0.71	0.56; 0.79	0.35	1.6	< 0.00001*
	Male (2.0–6.9 ng/ml)	23	5.3	1.32	5.09	4.19; 6.18	2.92	8.13	
Prolactin serum level	Female (2.39–25.15 ng/ml)	24	16.22	8.34	12.77	10.88; 19.11	7.31	41.42	0.003*
	Male (0.94–20.94 ng/ml)	23	11.16	8.5	8.56	5.96; 13.61	4.37	43.26	
FSH serum level	Female RA (2.0–10.0 mIU/ml) PM (20.0–100.0 mIU/ml)	24	7.97	8.66	4.38	1.88; 12.59	0.23	29.24	0.205
	Male (0.89–11.72 mIU/ml)	23	4.36	5.04	2.76	1.02; 5.44	0.16	20.15	
LH serum level	Female RA (up to 200 mIU/ml) PM (20.0–100.0 mIU/ml)	24	13.99	10.06	11.65	6.2; 16.61	1.99	36.542	0.067
	Male (3.0–12.0 mIU/ml)	23	8.41	3.4	8.72	5.73; 9.94	2.96	16.49	
HDL	Female (> 45 mg/dl)	24	65.63	12.01	67.5	58.75; 72	43	87	0.013*
	Male (> 35 mg/dl)	23	57.26	18.42	52	44.5; 63	39	117	
TG	Female (65–150 mg/dl)	24	79.75	19.72	76	65; 89.75	45	127	0.302
	Male (65–150 mg/dl)	23	95.22	37.8	81	67.5; 114	54	199	
Study group:									
Testosterone serum level	Female (0.26–1.22 ng/ml)	37	0.45	0.27	0.32	0.23; 0.71	0.05	1.09	0.001*
	Male (2.0–6.9 ng/ml)	4	4.24	0.68	4.1	3.73; 4.61	3.65	5.13	
Prolactin serum level	Female (2.39–25.15 ng/ml)	37	17.39	7.92	15.98	12.22; 21.43	2.54	33.28	0.011*
	Male (0.94–20.94 ng/ml)	4	10.66	7.08	8.34	5.58; 13.42	5.41	20.54	
FSH serum level	Female RA (2.0–10.0 mIU/ml) PM (20.0–100.0 mIU/ml)	37	12.37	17.67	7.8	5.7; 12.4	1.9	20.7	0.826
	Male (0.89–11.72 mIU/ml)	4	10.35	8.35	9.5	6.65; 13.2	1.08	21.3	
LH serum level	Female RA (up to 200 mIU/ml) PM (20.0–100.0 mIU/ml)	37	19.94	20.16	12.8	7.1; 23.1	3.6	74.9	0.026*
	Male (3.0–12.0 mIU/ml)	4	4.53	3.68	4.15	1.45; 7.23	1.3	8.5	
HDL	Female (> 45 mg/dl)	37	66.78	15.97	65	54; 82	43	95	0.159
	Male (> 35 mg/dl)	4	55	6.68	53.5	50.5; 58	49	64	
TG	Female (65–150 mg/dl)	37	171.95	84.97	147	115; 235	35	332	0.435

**Statistical significance, RA – reproductive age, PM – post-menopausal age.*

Table 4. Percentage and statistical analysis elevated parameters in the study group and control group

Parameter	Control group	Study group	*P*-value	Test
Elevated testosterone	5 (10.64%)	0 (0%)	0.0912	χ^2 Yates
Elevated FSH	5 (10.64%)	3 (7.32%)	–	–
Elevated LH	3 (6.38%)	0 (0%)	–	–
Elevated Prolactin	4 (8.51%)	6 (14.63%)	0.5712	χ^2 Yates
Elevated TG	2 (4.26%)	22 (53.66%)	< 0.00001*	χ^2 trend
Decreased HDL	1 (2.44%)	1 (2.13%)	0.5358	χ^2 Yates

**Statistical significance.*

There was a statistically significant difference in the percentage of elevated TG between both groups. The results of the study group and control group are presented in Table 4.

Discussion

Testosterone belongs to the group of steroid hormones which is produced in the gonads and adrenal cortex. The serum level of testosterone is ten times higher in male compared to female patients, however, females are more sensitive to testosterone serum level imbalance [17]. This hormone regulates the function of cells by binding to the nuclear androgen receptors, a high density of which occurs in sebaceous glands [18]. Receptors for androgens are located in the basal layer of sebaceous glands and keratinocytes of the hair follicle [19]. Testosterone interacting with nuclear receptors also influences dermal fibroblasts and stimulates the production of growth factor.

In the cells of the sebaceous glands, enzymes involved in the transformation of androgens are expressed, including 3β-hydroxysteroid dehydrogenase; 17β-hydroxysteroid dehydrogenase; 5α-reductase [19, 20]. Moreover, it has been hypothesized that an increased sebaceous gland level of 5α-reductase is related to the excessive proliferation of keratinocytes, which initiates the process of the formation of comedones. Dihydrotestosterone produced with the involvement of 5α-reductase has a greater affinity to the androgen receptors than testosterone, which makes this complex more stable and effective [21]. Androgens lead to hyperkeratosis and increased sebum production, which provides the appropriate environmental conditions for *Propionibacterium acnes* development. This bacterial factor is involved in *acne vulgaris* pathogenesis [1, 18, 22].

The testosterone serum level is also strongly correlated with other hormone levels, for example, LH, which is produced in the pituitary gland. The release of testosterone is controlled by the serum level of LH. There is also a correlation between an increased level of androgens and a negative feedback regulation effect on the hypothalamus-pituitary axis [19]. Elevated serum levels of androgens may influence the development of acne lesions, but *acne vulgaris* may also occur in patients with normal levels of testosterone due to increased sensitivity of sebaceous glands [19, 20].

According to the study by Iftikhar and Choudhry, patients with *acne vulgaris* presented elevated serum levels of testosterone and other androgens compared to controls. The correlation between the serum level of testosterone and the severity of acne was also proved [18]. This finding is similar to a study conducted by Gayen *et al.,* which also indicated elevated serum levels of testosterone in 49.4% of patients [23]. In the study conducted by Anaje *et al.,* no correlation was found between an elevated testosterone serum level and the occurrence of acne [22]. Zhang *et al.* investigated the correlation between sex hormone levels and acne grades. In their study, testosterone levels were within the normal range independent of acne severity, but an increase in the ratio of androgen to oestrogen correlated with acne grades [24]. The results of our study, which indicate a decreased testosterone serum levels in the acne group, may have been induced by groups consisting mainly of female patients. However, the ambiguity of research on the correlation between testosterone and acne forces the necessity for more investigations on a larger group of participants.

Prolactin is a polypeptide hormone secreted by the lactotroph cells of the pituitary gland. Its action is controlled by the hypothalamic-pituitary-adrenal axis, and prolactin secretion is also inhibited by dopamine [25]. Prolactin controls the reproductive and lactative processes, although prolactin receptors (PRLR) are located all over the body, leading to the symptoms of hyperprolactinaemia in different tissues. Hyperprolactinaemia is described as the serum level of prolactin being over 20 ng/ml [26]. Physiologically, the prolactin serum level is elevated in situations including pregnancy, lactation, sleep, stress, exercise or sexual intercourse. Pathological causes of hyperprolactinaemia include systemic diseases like renal insufficiency, primary hypothyroidism, hypothalamic diseases like prolactinomas, thyrotropinomas, acromegaly, stalk and neurogenic disorders, and many more. Drugs, including e.g. antipsychotics, antidepressants, antihypertensive drugs, prokinetic drugs, or H2-receptor blockers, often lead to hyperprolactinaemia as well [27].

The prolactin serum level increase may be indicative of different disorders, e.g. levels up to 100 ng/ml are characteristic of primary hypothyroidism, drug intake, macroprolactinaemia or non-functioning pituitary adenomas, whereas values up to 250 ng/ml may suggest microprolactinomas and up to 1000 ng/ml are indicative of macroprolactinomas [27]. Despite the fact that the majority of patients with hyperprolactinaemia present

no symptoms, in 26% of cases they may include menstrual disorders like amenorrhea, oligomenorrhea, and infertility, due to the inhibition of the hypothalamic-pituitary-gonadal axis, resulting in a decrease in GnRH, LH and, FSH and finally, causing a reduction in oestrogen and testosterone secretion. 13% of patients suffer from galactorrhea in the course of hyperprolactinaemia [28].

Prolactin receptors are also located in the human skin, in cells including keratinocytes, fibroblasts, and sebocytes, where PRL acts as a growth hormone and immune system modulator. Physiologically, the hormone plays a role in proper hair growth, osmoregulation, and thermoregulation, while abnormal levels of prolactin may be involved in the development of many skin disorders, including psoriasis, lupus erythematosus, systemic sclerosis, hyperseborrhea, and *acne vulgaris* [26]. A good example of hyperseborrhea in the course of hyperprolactinaemia is seborrheic dermatitis in patients suffering from Parkinson's disease [29]. Moreover, in terms of *acne vulgaris*, prolactin increases the androgen serum level, which affects the sebaceous glands and leads to acne formation.

According to the study by Borzyszkowska *et al.,* patients suffering from *acne vulgaris* presented a statistically higher serum level of prolactin (20.66 ng/ml) compared to the control healthy no-acne group (17.91 ng/ml) ($p = 0.0010$). Furthermore, this study also proved a statistically significant difference ($p = 0.001$) in testosterone serum levels between groups [30]. The importance of prolactin measurement in *acne vulgaris* cases was demonstrated previously by Arora *et al.* in 2010. Their results also indicate statistically higher serum levels of prolactin in the group of *acne vulgaris* patients [31]. In the study by Akdogan *et al.,* the prolactin serum level was higher in patients with acne than in the control group, but this result was not statistically significant [13]. The study by Meena *et al.* revealed an elevated prolactin serum level in 2 out of 60 acne patients (3.3%). Moreover, 6.7% of patients presented a raised total testosterone level [32]. In the study by Dhaher *et al.,* in up to 32% of patients with acne, the measured prolactin serum level was above the normal range [33]. Previous results are consistent with our study – statistical analysis also revealed a significant difference in prolactin serum levels between acne patients and healthy controls. The mean ± SD was higher in patients suffering from *acne vulgaris* than in the controls. However, in the study conducted by Khunger *et al.*, none of the 280 acne patients presented an elevated level of prolactin serum [34].

A comparison of our results connected with prolactin serum levels with those of selected authors is presented in Table 5.

Gonadotropins, which include FSH and LH produced in the pituitary gland, are peptide hormones that regulate ovarian and testicle function [35]. Their production is regulated by the gonadotropin releasing hormone (GnRH). FSH in women is responsible for the maturation of the follicles, and when at its peak – for ovulation, whereas in men FSH regulates spermatogenesis. LH promotes progesterone production in women, while in men leads to testosterone production by the testes.

In the diagnostic approach, the LH/FSH ratio is often considered, and in healthy women values between 1 and 2 are expected. Values over 2 may be indicative of the diagnosis of polycystic ovary syndrome [36]. In the study by Borzyszkowska *et al.,* LH and FSH serum levels did not statistically vary between the acne group versus control group. Moreover, there was no correlation between the hormone level and acne severity [30]. In the study by Meena *et al.,* elevated LH and FSH serum levels were found in 3.3% and 18.3% of acne patients, respectively [32]. The study by Khunger *et al.* revealed an elevated LH serum level in 2 out of 280 patients and a decreased FSH serum level in 1 patient. The LH/FSH ratio was more than 2 in 2 patients [34]. The study by Dhaher *et al.* showed an elevated LH/FSH ratio in up to 25% of patients, although all patients had previously been diagnosed with PCOS [33]. The result of the study presented by Zhang *et al.* confirmed a correlation between elevated levels of FSH and the severity of acne [24]. In our study, statistical analysis also revealed higher mean ± SD FSH serum levels in acne patients compared to the healthy controls.

Acne vulgaris lesions are strongly related to the excessive production of sebum, which consists of wax, cholesterol, fatty acids, esters of glycerol, and squalene [37]. Due to the relationship between acne lesions and the increased production of lipid substances in the sebaceous glands, attempts have been made to assess the correlation between lipid profile changes and the occurrence and severity of acne.

In research conducted by Abulnaja *et al.,* patients suffering from *acne vulgaris* presented elevated levels of TG and LDL-C and significantly lower HDL-C. Moreover, abnormalities in hormonal profiles in patients with acne were also proved [1]. These results are consistent with the results of a study by Younis *et al.* [38]. Chandak *et al.* [39] showed a statistically significant difference in the percentage of patients with hypertriglyceridemia between acne patients and controls, but the means with SD were not statistically different.

Furthermore, abnormalities in lipid profiles are closely related to elevated prolactin serum levels [40]. An increased amount of prolactin receptors during adipocyte differentiation suggests the direct influence of prolactin on lipid metabolism. In our study, patients suffering from *acne vulgaris* also presented elevated serum levels of prolactin. Arora *et al.* investigated the relationship between lipid serum levels and the menstrual cycle in *acne vulgaris* patients. They showed increased levels of total cholesterol, LDL-C, testosterone, and prolactin and decreased HDL-C and oestrogens in the acne group [31].

Jiang *et al.* showed increased TC, LDL-C, and LP(a) levels in the study groups compared to the controls. Like

Table 5. The comparison of our results connected with prolactin with those of other authors – results presented as mean ± SD, median (Q1:Q3), and percentage of patients with an elevated prolactin serum level

Author, year	Group	Number of patients	Sex (*N*)	Prolactin serum levels	*P*-value	Results presentation
Our study	Study group	41	37 F 4 M	16.73 ±8.02	0.019	Mean ± SD
	Controls	47	24 F 23 M	13.74 ±8.71		
	Study group	41	37 F 4 M	15.84 (11.05; 21.06)	0.019	Median (Q1–Q3)
	Controls	47	24 F 23 M	11.83 (8.34; 16.44)		
Zhang *et al*., 2022	Grade I	78	71 F	283.94 (156.20–427.85)	For female 0.232	Median (Q1–Q3)
			7 M	300.16 (186.04–371.23)		
	Grade II	259	233 F	256.93 (142.10–415.72)		
			26 M	188.00 (142.23–340.63)		
	Grade III	238	187 F	239.00 (116.50–357.39)	For male 0.505	
			51 M	201.31 (127.70–276.08)		
	Grade IV	118	74 F	258.86 (144.94–389.80)		
			44 M	214.00 (157.88–308.66)		
Borzyszkowska *et al*., 2022	Study group	99	F	20.66 ±7.99	0.001	Mean ± SD
	Controls	69	F	17.91 ± 8.33		
Arora *et al*., 2010	Study group	30	F	13.43 ±0.74	< 0.001	Mean ± SD
	Controls	30	F	9.74 ±0.56		
Aktar *et al*., 2020	Study group	30	F	16.3 ±5.3	0.752	Mean ± SD
	Controls	30	F	15.8 ±7.9		
Akdogan *et al*., 2018	Study group	47	F	13.1 ±11.1	0.073	Mean ± SD
	Controls	47	F	11.67 ±9.28		
Elevated prolactin serum level in %						
Our study	Study group	41	37 F 4 M	14.63%	0.571	Percentage
	Controls	47	24 F 23 M	8.51%		
Meena *et al*., 2022	Study group	60	F	3.3%	–	Percentage
Dhaher *et al*., 2022	Adolescent acne	160	F	21.25%	–	Percentage
	Early adult-onset acne	80	F	30%		
	Post-adolescent acne	100	F	32%		

our results, the levels of TG were significantly higher in patients with acne compared to the healthy controls [37]. A study conducted by Sobhan *et al.* confirmed increased cholesterol and TG levels in acne patients with a strong correlation with the male sex. HDL serum levels in both males and females in this study were higher in the group

Table 6. The comparison of our results connected with TG with those of other authors – results presented as mean ± SD and percentage of patients with elevated TG

Author, year	Group	Number of patients	Sex (*N*)	TG	*P*-value
Our study	Study group	41	37 F 4 M	175.29 ±82.15	< 0.00001
	Controls	47	24 F 23 M	87.32 ±30.64	
Arora *et al*., 2010	Study group	30	F	133.4 ±4.07	> 0.05
	Controls	30	F	131.7 ±1.96	
Jiang *et al*., 2015	Study group	101	F	95.4 ±47.21	0.447
	Controls	68	F	90.35 ±26.57	
	Study group	80	M	121.35 ±39.86	0.11
	Controls	62	M	111.6 ±23.915	
Chandak *et al*., 2022	Study group	65	25 M 40 F	121.60 ±41.52	0.157
	Controls	65	25 M 40 F	112.29 ±32.37	
Younis *et al*., 2021	Study group	530	–	231.04 ±30.02	< 0.001
	Controls	550	–	196.49 ±29.72	
Abulnaja *et al*., 2009	Obese AV	15	F	197 ±13.7	< 0.05
	Obese controls	15	F	171 ±11.5	
	Non-obese AV	15	F	180 ±10.4	< 0.05
	Non-obese controls	15	F	163 ±8.9	
Sobhan *et al*., 2020	Study group	45	28 F 17 M	94.6 ±51.4	0.725
	Controls	45	31 F 14 M	86.7 ±36.6	
Elevated TG serum level in %					
Our study	Study group	41	37 F 4 M	53.7%	< 0.00001
	Controls	47	24 F 23 M	24.6%	
Chandak *et al*., 2022	Study group	65	25 M 40 F	55.4%	0.024
	Controls	65	25 M 40 F	24.6%	
Jiang *et al*., 2015	Study group	101	F	8.9%	> 0.05
	Controls	68	F	2.9%	
Jiang *et al*., 2015	Study group	80	M	37.5%	< 0.05
	Controls	60	M	6.45%	

of subjects, but the difference was not statistically significant [41]. The results for HDL levels in the course of acne in the Sobhan *et al.* study are consistent with our results. Gayen *et al.* assessed the correlation between acne and hormonal and lipid changes. This investigation showed a correlation between not only hormonal imbalance in acne, but also lipid abnormalities, especially in patients with acne and hirsutism [23].

A comparison of our results connected with TG serum levels with those of selected authors is presented in Table 6.

Conclusions

The results of our study indicate the presence of abnormalities in the levels of selected hormones, especially

prolactin, in the group of patients with *acne vulgaris*. Moreover, the imbalance in the lipid profile could be correlated with hormonal abnormalities in acne, which is shown in statistically higher TG serum levels in patients suffering from *acne vulgaris*. Our results correspond with those of some previous studies, which indicates the importance of examining both hormone and lipid profiles in patients with acne. Diagnosing either hormonal or lipid abnormalities could be crucial in treatment decisions and could influence the occurrence of complications and the course of acne.

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Conflict of interest

The authors declare no conflict of interest.

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