Received: 4 March 2023 | Revised: 23 May 2023 | Accepted: 24 May 2023

DOI: 10.1002/hsr2.1322

ORIGINAL RESEARCH

Health Science Reports Open Access WILEY

Alterations in lipid and hormonal titers in patients with acne and their relationship with severity: A case-control study

Ghada Farouk Mohammed[1] | Mohammed Saleh Al-Dhubaibi[2] | Saleh Salem Bahaj[3] | Ahmed Ibrahim AbdElneam[4,5]

[1]Department of Dermatology, Venereology, and Sexology, Faculty of Medicine, Suez Canal University, Ismailia, Egypt

[2]Department of Dermatology, Department of Medicine, College of Medicine, Shaqra University, Dawadmi, Saudi Arabia

[3]Department of Microbiology and Immunology, Faculty of Medicine and Health Sciences, Sana'a University, Sana'a, Yemen

[4]Department of Clinical Biochemistry, Shaqra University, Dawadmi, Saudi Arabia

[5]Department of Molecular Genetics and Enzymology, Human Genetics and Genome Research Institute, National Research Center, Cairo, Egypt

Correspondence

Saleh Salem Bahaj, Department of Microbiology and Immunology, Faculty of Medicine and Health Sciences, Sana'a University, Sana'a, Yemen.
Email: salehbahaj2025@gmail.com

Abstract

Background and Aims: Acne is a frequently diagnosed skin condition that causes pilosebaceous apparatus clogs and/or inflammatory responses in the majority of teenagers. It is a multifactorial disease that can develop due to various factors. We aimed to evaluate lipid profiles and hormonal levels in patients with acne and correlate them to acne severity. We also aim to explore the alteration of lipid profiles and hormonal levels and their effect on the occurrence of acne.

Methods: A case-control study was performed on 100 individuals with acne vulgaris and 100 healthy controls. The biochemical analysis included; lipid profiles such as triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), and hormonal levels such as estradiol (E), total testosterone (TT), and free testosterone (FT) were measured for both patients and controls.

Results: Comparison between patients with acne and controls disclosed that; TC, TG, LDL-C, and HDL-C levels were significantly higher in patients, especially when compared to controls ($p \leq 0.05$); also, the same results were found in hormonal levels results ($p \leq 0.05$).

Conclusion: These altered lipid profiles and androgen levels should be considered in the pathophysiology of acne and taken into consideration when treating patients with acne.

KEYWORDS

acne, case-control study, hormonal levels, lipid profiles

1 | INTRODUCTION

Acne is the much more frequently diagnosed clinical ailment in adolescents.[1] Acne influences roughly 85% of adolescents, and young adults between the ages of 12 and 25.[1] Acne has been reported to affect most teenagers between the ages of 15 and 17.[2]

Acne is a complex disorder of the pilosebaceous unit characterized by excessive sebum production, inflammation, follicular hyperkeratinization, and *Propionibacterium acnes* bacterial colonization. Hormonal stimulation is the primary regulator of the pilosebaceous apparatus. Androgens are essential in the pathophysiology of acne, and sebum excretion is

androgen-dependent.[3,4] Changes in sebum excretion are thought to contribute to acne.[5]

Human sebum comprises squalene, wax, glycerol esters, cholesterol, free cholesterol, and fatty acids.[6] According, to the previous works, acne patients' lipid profiles in serum differ significantly from those found in healthy controls. Acne patients, both genders, have abnormally low serum levels of high-density lipoprotein cholesterol (HDL-C). Affected individuals have higher levels of testosterone, progesterone, total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C).[7,8]

Other researches aim to find the relation between either lipid profile and acne or hormonal profile and acne.[3,4,7,8] However, this study seeks to find the correlation between both factors-lipids and hormones-and acne all together to the same patients. More research is considered necessary to establish the link between the serum lipid profiles of acne vulgaris and hormonal levels as serum estradiol (E), sex hormone binding globulin (SHBG), total testosterone (TT), and free testosterone (FT). There have been a few studies, with varying degrees of success, on the blood lipid profiles and hormone levels in individuals with acne vulgaris.

The goal of this study was to look at lipid profiles as well as hormonal imbalances in patients who reported acne and see how they were strongly associated with acne severity. Also, we aimed to study the pathogenic effects of hyper- or hypolipidemia and hormonal alteration in acne severity.

2 | SUBJECTS AND METHODS

2.1 | Subjects

Following departmental research committee approval and informed verbal patient consent, this case-control research was executed on 100 female and male individuals with acne vulgaris (group A) visiting a dermatological outpatient department. These patients were given a thorough medical history as well as a general and local clinical examination. The serum lipid profiles and hormonal levels were both examined. Their findings were compared to 100 age-matched healthy controls, who are also examined by a dermatologist (group B) (Figure 1).

Sample size was calculated from https://www.calculator.net/sample-size-calculator.html

2.2 | Inclusion criteria

1. Gender: both.
2. Age: 18–45.
3. Patients with recent or old diseases (with acne vulgaris) were clinically diagnosed by a dermatologist.
4. Not having received systemic treatment for a minimum of 2 months before the investigation.

2.3 | Exclusion criteria

1. Individuals who smoked or were receiving treatment that had an effect on lipid metabolism.
2. Individuals with other skin diseases.
3. Individuals with other systemic diseases mainly affect lipid metabolism (nephrotic syndrome, hyper- and hypothyroidism, pancreatitis, uremia, obstructive liver disease, and uncontrolled diabetes mellitus).
4. Pregnant and lactating females.
5. Females received oral contraceptive pills.
6. Individuals with acne who used systemic retinoids or received topical acne treatment such as antibiotics, benzoyl peroxide, or retinoids for at least 2 months before the research.

2.4 | Diagnosis of acne vulgaris

A global acne grading system (GAGS) was utilized to grade the acne[8] (Table 1).

Each category of lesion is assigned a value (grades 0–4) based on its severity:

Comedones are one, papules are two, pustules are three, and nodules are four.

The following formula is used to calculate the score for each area (local score): factor × grade = local score (0–4).

A global score is the sum of the local scores, and the global score was used to grade acne severity.[8]

A score of:

1–18: mild.
19–30: moderate.
31–38: severe.
>39: very severe

3 | METHODS

3.1 | Body mass index (BMI) assessment

BMI was projected by splitting body weight by height squared (kg/m^2).

3.2 | Estimation of lipid profiles

The participants had been on usual nutrition and in a stable environment for at least 2 weeks before the research. In less than 24 h of sample collection, they prevented strenuous exercise or becoming exhausted. Specimens were taken immediately in ethylenediaminetetraacetic acid tubes and centrifuged at 4°C for 15 min at 2000 rpm. Plasma was detached and stored at 4°C within 1 h of blood sample collection. Within 24 h, the blood samples were evaluated. Commercially available enzymatic colorimetric tests were

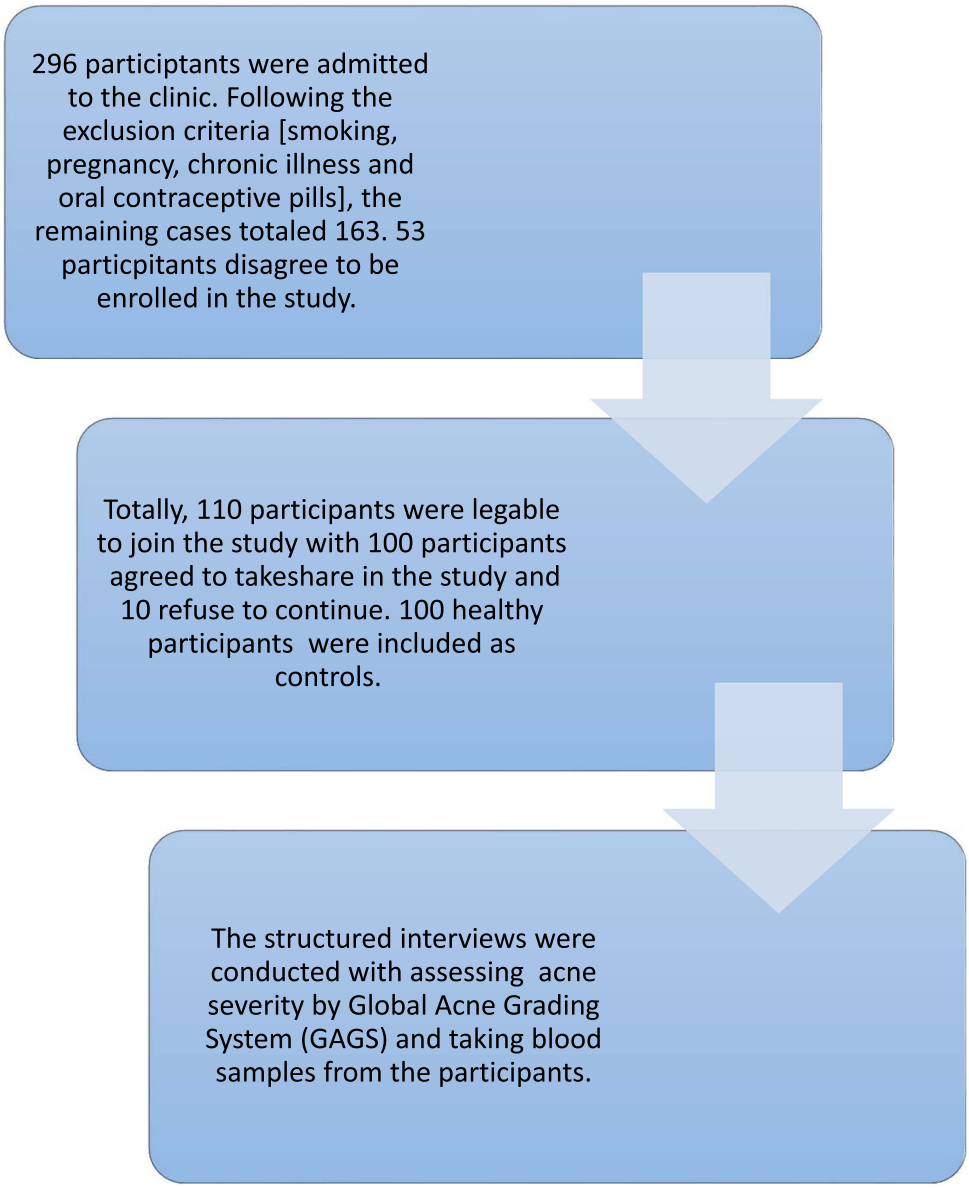

FIGURE 1 Shows the flowchart of the study.

TABLE 1 The global acne grading system.[9]

Location	Factor
Forehead	2
Right cheek	2
Left cheek	2
Nose	1
Chin	1
Chest and upper back	3

Note: Each type of lesion is given a value (grades 0–4) depending on severity: No lesions = 0, comedones = 1, papules = 2, pustules = 3, and nodules = 4. The score for each area (Local score) is calculated using the formula: local score = factor × grade (0–4). The global score is the sum of local scores, and acne severity was graded using the global score.[9] A score of 1–18 is considered mild, 19–30: moderate, 31–38: severe, >39: very severe.

used to evaluate serum TC, triglyceride (TG), HDL-C, and LDL-C levels. Roche Diagnostics used the Olympus AU 400 automated clinical chemistry analyzer to determine plasma lipid concentrations. The Friedwald et al.[10] formula for calculating LDL-C was used.

3.3 | Estimation of hormonal levels

Every participant donated 5 mL of blood to be evaluated for serum (E), (SHBG), TT, and FT. (VIDAS®/BIOMERIEUX SA/Marcy-l'Etoile France) assessed serum TT, FT, and E levels. The normal range for TT is 0.06–0.82 ng/mL, 0.04–4 pg/mL for FT, and 12.5–166 pg/mL for E (Follicular). The periovulatory phase (±3 days) ranges from 43.8 to 166 pg/mL. The luteal phase concentration ranges from 1.7 to 27 pg/mL. SHBG concentrations were determined using an immunoassay (DPC/Los Angeles) with a normal range of 18–114 nmol/L. The assays'

TABLE 2 General clinical and laboratory characteristics of acne patients and healthy controls.

Variables	Acne patients *N* = 100	Healthy controls *N* = 100	*p* value
Age (mean ± *SD*)	27.5 ± 4.2	28.1 ± 3.2	0.07
Gender male/female (*N*/%)	56 (56%)/44 (44%)	37 (51.4%)/35 (48.6%)	NA
Marital status			
- Yes	39	39	NA
- No	61	71	
Smoking			
- Yes	17	18	NA
- No	83	82	
Duration of disease (years)			NA
- <5	22	NA	
- ≥5	78		
Chronic illness			
- No	100	100	NA
- Others	NA	NA	
Acne vulgaris severity			
- Mild	30	NA	NA
- Moderate	50		
- Severe	20		
TG mg/dL (mean ± *SD*)	165.2 ± 23.5	58.3 ± 23.5	0.02[a]
TC mg/dL (mean ± *SD*)	226.4 ± 23.2	171.3 ± 20.1	0.03[a]
LDL-C mg/dL (mean ± *SD*)	127.2 ± 10.3	83.2 ± 10.5	0.02[a]
HDL-C mg/dL (mean ± *SD*)	58.2 ± 3.2	40.5 ± 1.1	0.046[a]
Estradiol pg/ml (mean ± *SD*)	85.1 ± 11.3	29.1 ± 13.4	0.03[a]
Free testosterone ng/dL (mean ± *SD*)	15.9 ± 12.1	2.4 ± 12.2	0.02[a]
Total testosterone ng/dL (mean ± *SD*)	389.1 ± 21.3	235.1 ± 12.3	0.03[a]

Note: N = number, % = percentage.

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; SD, standard deviation; TC, total cholesterol; TG, triglycerides.

[a]Mild significant differences $p \leq 0.05$.

sensitivity was demonstrated by the minimum amount of hormones differentiated from the zero samples with 95% certainty and the intra-assay and inter-assay coefficients of variation for TT, FT, E, and SHBG. All participants had blood tests done simultaneously in the same laboratory using the same technique. This was done between menstrual days 8 and 15. All women had their blood drawn between 8:00 a.m. and 12:00 p.m. and stored at −20°C for 1–30 days before being analyzed.

3.4 | Statistical analysis

The data collected was analyzed using the Statistical Package for Social Sciences, version 20.0, developed by SPSS Inc. An analysis of variance test was conducted for more than two samples, and an independent sample *t* test was utilized for comparing means of the two samples. The significance level was set at $p \leq 0.005$. The quantitative data was presented as mean ± standard deviation (*SD*), while qualitative data was presented as a percentage and frequency.

4 | RESULTS

In the present work, we analyzed the lipid profiles and hormonal levels in acne vulgaris patients. Factors that might affect the levels of hormones and lipid profiles, such as education status, socioeconomic status, and marital status, show no significant difference between

groups; stressors were also excluded during general and clinical evaluation.

The research's population included 100 acne patients, whose mean age was 27.5 ± 4.2 years, and 100 healthy controls with age and sex matching as a control group. A comparison of patients with acne and controls demonstrated that; levels of TC, LDL, HDL, and TG in patients were elevated significantly more than controls ($p \leq 0.05$), and the same results were found in hormonal levels ($p \leq 0.05$) (Table 2). Ther was no significant difference of anthropometric characteristics of acne patients and healthy controls (Table 3). The mild grade of acne found in 30%, 50% were moderate grade, while sever form of acne was in 20% of participants (Figure 2).

When comparing the lipid profiles according to gender, it was found that all lipid profile parameters were higher in males with acne compared to healthy male controls ($p \leq 0.05$). Significant variations were observed in lipid levels between female patients and female controls ($p \leq 0.05$) for all lipid profile parameters (Table 4).

Our results indicated that only TG has a mild significant variation ($p = 0.04$) when we compare male patients with acne against female patients with acne (Table 5). Table 6 shows that all male and female patients with acne vulgaris had a mildly substantial rise in hormonal levels as compared to controls. There was a high statistical difference between male and female acne vulgaris patients for both FT and TT ($p \leq 0.001$) (Table 7).

At baseline, the TG was 58.4 ± 5.6 mg/dL for individuals with severe acne and 45.1 ± 4.2 mg/dL for patients with moderate type, while in patients with mild type were 34.1 ± 4.3 mg/dL. There were minor differences in TG levels in between all clusters of acne ($p = 0.04$). Also, TC was greater in severe patients with acne vulgaris than in those with mild and moderate types ($p = 0.037$). LDL and HDL were higher in severe types, especially in comparison to other types ($p = 0.041$ and 0.029), respectively. Hormonal E, FT, and TT were greater in severe types compared to control groups, with ($p = 0.032$, 0.045, and 0.031), respectively (Table 8).

TABLE 3 Anthropometric characteristics of acne patients and healthy controls.

Variables	Acne patients $N = 100$	Healthy controls $N = 100$	p value
BMI			
- Underweight <18.5	1	–	
- Normal 18.5–24.9	89	83	NA
- Obese 30–34.9	10	17	
- Extremely obese <35	–	–	

Abbreviation: BMI, body mass index.

5 | DISCUSSION

Acne is a multifaceted inflammatory process of the pilosebaceous apparatus that is chronic.[11] Acne can occur in a variety of regional, demographic, and cutaneous lesion types.[12] Notwithstanding the numerous risk factors, the etiopathogenesis and pathophysiology of acne are unknown.[13]

In the present study, we noted that there were significant statistical discrepancies ($p \leq 0.05$) in TG, TC, LDL-C, and HDL-C levels between those with acne and controls ($p = 0.02$, 0.03, 0.02, and 0.05, respectively). This difference was found in patients of both genders compared to controls.

In contrast to our findings, Jiang[9] and El Akawi[7] implied that individuals of both genders had lower plasma HDL-C levels when compared to healthy controls. Vergani[14] and Finzi found the same thing in male patients with acne with severe acne grades, moreover,

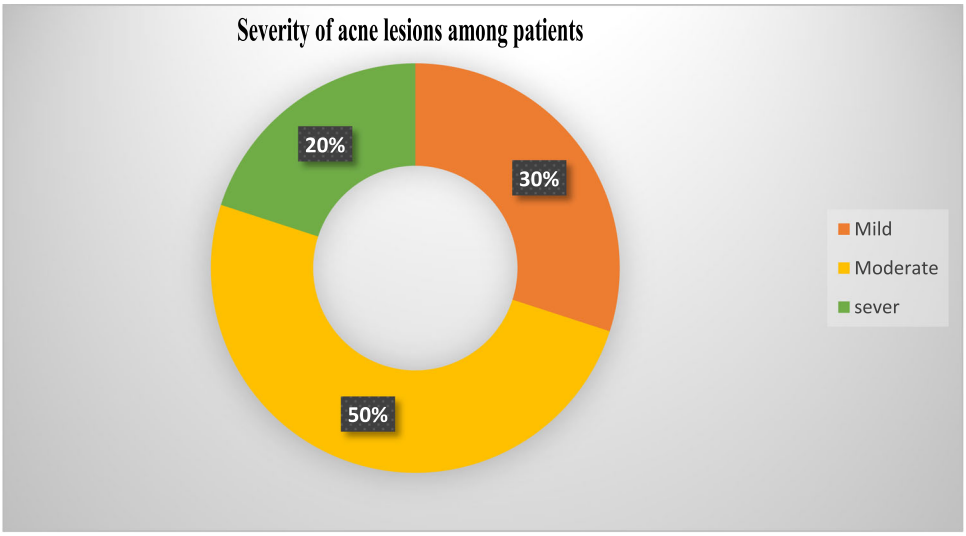

FIGURE 2 Severity of acne among patients by Global Acne Grading System (GAGS) shows that the mild grade of acne found in 30%, 50% were moderate grade, while sever form of acne was in 20% of participants.

TABLE 4 Comparison of lipid profile of male and female acne patients and control groups.

	Male			Female		
Parameters mg/dL	Acne patients *N* = 56 (mean ± *SD*)	Controls *N* = 56 (mean ± *SD*)	*p* value (*t* test)	Acne patients *N* = 44 (mean ± *SD*)	Controls *N* = 44 (mean ± *SD*)	*p* value (*t* test)
TG mg/dL	190.1 ± 33.5	71.2 ± 23.5	0.02[a]	145.2 ± 12.3	65.4 ± 55.4	0.03[a]
TC mg/dL	230.8 ± 33.1	162.7 ± 25.1	0.03[a]	222.8 ± 20.1	189.1 ± 25.1	0.04[a]
LDL-C mg/dL	134.2 ± 21.4	81.2 ± 13.2	0.02[a]	121.2 ± 42.5	75.2 ± 12.2	0.04[a]
HDL-C mg/dL	60.1 ± 5.6	41.2 ± 8.2	0.05[a]	55.5 ± 8.2	39.1 ± 6.6	0.04[a]
LDL/HDL-C	2.232 ± 3.82	1.970 ± 1.48	0.04[a]	2.1837 ± 5.18	1.9232 ± 1.84	0.05[a]

Note: N = number, % = percentage.

Abbreviations: HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; SD, standard deviation; TC, total cholesterol; TG, triglycerides.

[a]Mild significant differences $p \leq 0.05$.

TABLE 5 Comparison of lipid profile of male and female acne patients.

Parameters mg/dL	Acne patients male *N* = 56 (mean ± *SD*)	Acne patients female *N* = 44 (mean ± *SD*)	*p* value (*t* test)
TG mg/dL	190.1 ± 33.5	145.2 ± 12.3	0.04[a]
TC mg/dL	230.8 ± 33.1	222.8 ± 20.1	0.562
LDL-C mg/dL	134.2 ± 21.4	121.2 ± 42.5	0.725
HDL-C mg/dL	60.1 ± 5.6	55.5 ± 8.2	0.998

Note: N = number, % = percentage.

Abbreviations: HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; SD, standard deviation; TC, total cholesterol; TG, triglycerides.

[a]Mild significant differences $p \leq 0.05$.

TABLE 6 Comparison of hormonal profile of male and female acne patients and control groups.

	Male			Female		
Parameters	Acne patients *N* = 56 (mean ± *SD*)	Controls *N* = 56 (mean ± *SD*)	*p* value (*t* test)	Acne patients *N* = 44 (mean ± *SD*)	Controls *N* = 44 (mean ± *SD*)	*p* value (*t* test)
Estradiol pg/mL	91.2 ± 12.4	16.4 ± 21.4	0.04[a]	81.1 ± 42.1	41.3 ± 14.2	0.02[a]
Free testosterone ng/dL	33.1 ± 11.2	5.9. ± 21.1	0.03[a]	3.8 ± 22.2	1.5 ± 24.2	0.04[a]
Total testosterone ng/dL	865.4 ± 34.4	426.2 ± 23.2	0.04[a]	68.2 ± 1.21	16.1 ± 0.12	0.02[a]

Note: N = number.

Abbreviation: SD, standard deviation.

[a]Mild significant differences $p \leq 0.05$.

TABLE 7 Comparison of hormonal profile of male and female acne patients.

Parameters	Acne patients (male) *N* = 56 (mean ± *SD*)	Acne patients (female) *N* = 44 (mean ± *SD*)	*p* value (*t* test)
Estradiol pg/mL	91.2 ± 12.4	81.1 ± 42.1	0.324
Free testosterone ng/dL	33.1 ± 11.2	3.8 ± 22.2	0.001[a]
Total testosterone ng/dL	865.4 ± 34.4	68.2 ± 1.21	0.001[a]

Note: N = number.

Abbreviation: SD, standard deviation.

[a]High significant differences $p \leq 0.001$.

TABLE 8 Correlation coefficients among acne severity, hormonal profile, and lipid profile.

Parameter	Acne severity Mild *N* = 30 (mean ± *SD*)	Moderate *N* = 50 (mean ± *SD*)	Sever *N* = 20 (mean ± *SD*)	*p* value (*t* test)
TG mg/dL	34.1 ± 4.3	45.1 ± 4.2	58.4 ± 5.6	0.043[a]
TC mg/dL	89.1 ± 23.2	109.1 ± 11.1	134.2 ± 21.4	0.037[a]
LDL-C mg/dL	162.1 ± 12.1	195.2 ± 23.4	225.1 ± 21.2	0.041[a]
HDL-C mg/dL	98.3 ± 12.3	109.4 ± 55.4	110.3 ± 15.3	0.029[a]
LDL/HDL-C	1.6490 ± 0.98	1.7870 ± 0.42	2.0407 ± 1.38	0.04[a]
Estradiol pg/mL	45.2 ± 24.1	81.2 ± 12.2	92.2 ± 12.2	0.032[a]
Free testosterone ng/dL	2.6 ± 21.3	12.5 ± 35.1	25.2 ± 15.1	0.045[a]
Total testosterone ng/dL	243.2 ± 12.2	314.3 ± 21.7	391.2 ± 11.2	0.031[a]

Note: N = number.

Abbreviation: HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; SD, standard deviation; TC, total cholesterol; TG, triglycerides.

[a]Mild significant differences $p \leq 0.05$.

El Akawi[7] and colleagues 2007 discovered that the mean plasma TC, TG, and LDL-C levels in both genders did not differ substantially from controls, with the exception of LDL-C levels in those with severe type, and that there was a tendency for serum HDL-C levels in acne patients to lessen as the severity of the acne ailment risen. In this study, patients with severe form had a significantly greater LDL/HDL cholesterol ratio than the controls ($p \leq 0.05$). The LDL/HDL cholesterol ratio strongly predicts the threat of major cardiovascular events.[15]

In the current study, there was a statistically significant increase in serum levels of E, FT, and TT in patients, especially in comparison to controls ($p \leq 0.05$). These findings were consistent with Marynick's findings that affected women with cystic acne had higher serum dehydroepiandrosterone (DHEAS) and testosterone levels than controls ($p \leq 0.05$).[16]

In 2010, Arora and coworkers[17] indicated that serum concentrations of TT in females with acne were normal. They were, however, near the upper limit and significantly greater than the controls. The growth was statistically valuable. The patients had varying degrees of acne severity.[17]

This study exhibited a significant correlation among serum E, FT and TT levels, and acne severity ($p = 0.032$, 0.045, and 0.31, respectively). Furthermore, levels of serum in patients were all within normal limits.

Our findings agreed with those of Lucky[18] and colleagues, who conducted a 5-year longitudinal cohort study of 439 black and 432 white girls and discovered that females with severe forms of comedonae-type acne had significantly greater testosterone levels than those with mild or moderate forms.

We can explain the relationship between the disturbance of lipid profiles and hormonal levels in patients with acne by the effective role of cholesterol. But since androgens in the adrenals are manufactured from cholesterol deduced from the blood, cholesterol levels may influence acne development. Cholesterol, like adrenal steroids, is an immediate precursor for gonadal steroids. Sex hormones play a significant role in the progression of acne.[17]

For many years, there has been a link between sebaceous gland activity and teenage years; when puberty starts, sex hormone-mediated activation of the sebaceous gland leads to high secretion in both genders.[19] Whereas sex hormones play a part in developing acne, the levels of these hormones are regularly within the normal range, according to several studies. Hypertestosteronemia is most likely caused by a defect in receptor affinity.[20]

The association between both sex hormones and serum lipid profiles could shed light on their contribution in the pathophysiology of acne. Past researchs have linked sex hormones to poor lipid profiles. Human visceral and subcutaneous adipocytes contain estrogen and androgen receptors, implying that sex hormones impact lipid biotransformation in adipose tissues. Sex hormone metabolism-altering drugs also influence TC, LDL-C levels, as well as TG.[21]

The relation between lipid profiles and hormonal levels changes more clearly in females with polycystic ovary syndrome (PCOS), who have elevated lipid profiles and a higher threat of developing cardiovascular disease (CVD) than healthy controls.[22] Acne is a prevalent PCOS signe and adverse reaction. Females with PCOS have a greater LDL/HDL cholesterol ratio and a higher threat of CVD than normal controls.[22] Another CVD risk factor in women with acne and PCOS is a higher LDL/HDL cholesterol ratio. A further prototype for a significant connection between lipid profiles and hormonal levels shows that patients with metabolic syndrome have higher lipid levels and a higher risk of CVD than healthy controls.[23]

Acne is a frequent sign and symptom of the metabolic syndrome. Patients with metabolic syndrome have a higher LDL/HDL cholesterol ratio and a higher risk of CVD than healthy controls.[23] One other risk factor for CVD in acne and metabolic syndrome patients is a higher LDL/HDL cholesterol ratio.

All of this may lead to the conclusion that blood lipids and androgens play a role in determining acne severity in women. This could help clinicians develop new therapeutic strategies for acne vulgaris.

The research discovered that the serum lipid profiles of acne patients not only rise, but that several patients with acne have higher lipid profiles than normal. Inadequate evidence suggests that patients with acne who have abnormal lipid profiles are more likely to develop certain ailments in the not-too-distant future. It does, however, provide a unique foundation for future research into the pathophysiology of acne and the innovation of new treatments.

6 | CONCLUSION

Acne patients of varying severity have abnormal lipid and hormonal levels. These irregularities contribute to the advancement of the ailment and must be taken into account when treating acne. Even if all androgen levels are in the normal range, hormone treatment may be beneficial.

AUTHOR CONTRIBUTIONS

Ghada Farouk Mohammed: conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; software; supervision; validation; visualization; writing—original draft; writing—review & editing. **Mohammed Saleh Al-Dhubaibi**: conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; software; supervision; validation; visualization; writing—original draft; writing—review & editing. **Saleh Salem Bahaj**: data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; software; supervision; validation; visualization; writing—original draft; writing—review & editing. **Ahmed Ibrahim AbdElneam**: conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; software; supervision; validation; visualization; writing—original draft; writing—review & editing.

ACKNOWLEDGMENTS

There are no sponsors or fund for the research, it was supported by the author.

CONFLICTS OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions. "All authors have read and approved the final version of the manuscript [Saleh Salem Bahaj] had full access to all of the data in this study and takes complete responsibility for the integrity of the data and the accuracy of the data analysis."

ETHICS STATEMENT

Institutional review board and Research Ethical Committee in accordance with the Helsinki Declaration guidelines.

TRANSPARENCY STATEMENT

The lead author Saleh Salem Bahaj affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

ORCID

Ghada Farouk Mohammed http://orcid.org/0000-0003-3074-1347

Mohammed Saleh Al-Dhubaibi http://orcid.org/0000-0002-6418-6647

Saleh Salem Bahaj http://orcid.org/0000-0001-6582-907X

REFERENCES

1. Chen H, Zhang TC, Yin XL, Man JY, Yang XR, Lu M. Magnitude and temporal trend of acne vulgaris burden in 204 countries and territories from 1990 to 2019: an analysis from the Global Burden of Disease Study 2019. *Br J Dermatol*. 2022;186:673-683.
2. Ferdowsian HR, Levin S. Does diet really affect acne? *Skin Therapy Lett*. 2010;15(1–2):1-2.
3. Gollnick HPM. From new findings in acne pathogenesis to new approaches in treatment. *J Eur Acad Dermatol Venereol*. 2015;29:1-7.
4. Dréno B. What is new in the pathophysiology of acne, an overview. *J Eur Acad Dermatol Venereol*. 2017;31(suppl 5):8-12.
5. Li X, He C, Chen Z, Zhou C, Gan Y, Jia Y. A review of the role of sebum in the mechanism of acne pathogenesis. *J Cosmet Dermatol*. 2017;16:168-173.
6. Ottaviani M, Camera E, Picardo M. Lipid mediators in acne. *Mediators Inflamm*. 2010;2010:1-6.
7. El-Akawi Z, Abdel-Latif N, Abdul-Razzak K, Al-Aboosi M. The relationship between blood lipids profile and acne. *J Health Sci*. 2007;53:596-599.
8. Doshi A, Zaheer A, Stiller M. A comparison of current acne grading systems and proposal of a novel system. *Int J Dermatol*. 1997;36:416-418.
9. Jiang H, Li CY, Zhou L, et al. Acne patients frequently associated with abnormal plasma lipid profile. *J Dermatol*. 2015;42:296-299.
10. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem*. 1972;18:499-502.
11. Robeva R, Assyov Y, Tomova A, Kumanov P. Acne vulgaris is associated with intensive pubertal development and altitude of residence—a cross-sectional population-based study on 6,200 boys. *Eur J Pediatr*. 2013;172:465-471.
12. Schnopp C, Mempel M. Acne vulgaris in children and adolescents. *Minerva Pediatr*. 2011;63:293-304.
13. Zhang J, Shen Y, Wang T, et al. Prevalence of acne vulgaris in Chinese adolescents and adults: a community-based study of 17,345 subjects in six cities. *Acta Dermato Venereologica*. 2012;92:40-44.
14. Vergani C, Finzi AF, Pigatto PD, et al. Low levels of HDL in severe cystic acne. *N Engl J Med*. 1982;307:1151-1152.
15. Barter P, Gotto AM, LaRosa JC, et al. HDL cholesterol, very low levels of LDL cholesterol, and cardiovascular events. *N Engl J Med*. 2007;357:1301-1310.

16. Marynick SP, Chakmakjian ZH, McCaffree DL, Herndon JH. Androgen excess in cystic acne. *N Engl J Med*. 1983;308:981-986.
17. Kataria Arora M, Seth S, Dayal S. The relationship of lipid profile and menstrual cycle with acne vulgaris. *Clin Biochem*. 2010;43:1415-1420.
18. Lucky AW, Biro FM, Simbartl LA, Morrison JA, Sorg NW. Predictors of severity of acne vulgaris in young adolescent girls: results of a five-year longitudinal study. *J Pediatr*. 1997;130:30-39.
19. George R, Clarke S, Thiboutot D. Hormonal therapy for acne. *Semin Cutan Med Surg*. 2008;27:188-196.
20. Bershad SV. Diet and acne—slim evidence, again. *J Am Acad Dermatol*. 2005;53:1102.
21. Wranicz JK, Cygankiewicz I, Rosiak M, Kula P, Kula K, Zareba W. The relationship between sex hormones and lipid profile in men with coronary artery disease. *Int J Cardiol*. 2005;101:105-110.
22. Kazemi M, Pierson RA, Lujan ME, et al. Comprehensive evaluation of type 2 diabetes and cardiovascular disease risk profiles in reproductive-age women with polycystic ovary syndrome: a large Canadian cohort. *Journal of Obstetrics and Gynaecology Canada*. 2019;41:1453-1460.
23. Smith RN, Braue A, Varigos GA, Mann NJ. The effect of a low glycemic load diet on acne vulgaris and the fatty acid composition of skin surface triglycerides. *J Dermatol Sci*. 2008;50:41-52.

How to cite this article: Mohammed GF, Al-Dhubaibi MS, Bahaj SS, AbdElneam AI. Alterations in lipid and hormonal titers in patients with acne and their relationship with severity: a case-control study. *Health Sci Rep*. 2023;6:e1322. doi:10.1002/hsr2.1322