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Original Article

A Study of Insulin Resistance Among Individuals with Acne Vulgaris

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ABSTRACT

Background: Acne vulgaris is a common chronic inflammatory disorder of the pilosebaceous unit affecting adolescents and young adults. Emerging evidence suggests that insulin resistance may contribute to acne pathogenesis through increased androgen production, sebaceous gland activity, and follicular hyperkeratinization.

Aim: To evaluate insulin resistance in patients with acne vulgaris and to assess its association with severity of acne.

Materials and Methods : This hospital-based cross-sectional observational study was conducted in the Department of Dermatology, Venereology and Leprosy at Saratchandra Chattopadhyay Government Medical College over a period of 12 months. The study included clinically diagnosed acne vulgaris patients attending the Dermatology Outpatient Department (OPD), with a total sample size of 82 patients.

Results: Among the acne patients (n=38), 11 patients (28.9%) had normal fasting serum insulin levels, while 27 patients (71.1%) had elevated fasting serum insulin levels. Among the controls (n=44), 29 participants (65.9%) had normal fasting serum insulin levels and 15 participants (34.1%) had elevated levels. The difference in fasting serum insulin levels between the two groups was statistically highly significant ($p < 0.0001$).

Conclusion: Insulin resistance is more prevalent among patients with acne vulgaris, particularly in moderate and severe cases. Assessment of insulin resistance may help in identifying metabolic abnormalities at an early stage and may contribute to better therapeutic management of acne patientse.

Keywords*: Acne vulgaris, Insulin resistance, HOMA-IR, Hyperinsulinemia, Sebaceous gland, Metabolic syndrome.*

INTRODUCTION

Acne vulgaris is one of the most common chronic inflammatory diseases involving the pilosebaceous unit and is characterized by comedones, papules, pustules, nodules, and cysts. It commonly affects adolescents and young adults, although it can persist into adulthood or appear later in life. The condition has a multifactorial etiology involving increased sebum production, follicular hyperkeratinization, microbial colonization, inflammation, hormonal imbalance, and genetic predisposition. Acne significantly affects physical appearance and may lead to psychological consequences such as anxiety, depression, and reduced self-esteem.[1]

In recent years, increasing attention has been focused on the role of metabolic and endocrine factors in the pathogenesis of acne vulgaris. Among these factors, insulin resistance has emerged as a possible contributing mechanism. Insulin resistance refers to a decreased sensitivity of peripheral tissues to insulin action, resulting in compensatory hyperinsulinemia. Elevated insulin levels are known to influence androgen metabolism and sebaceous gland activity, both of which play important roles in acne development.[2]

Hyperinsulinemia stimulates ovarian and adrenal androgen synthesis and decreases hepatic production of sex hormone-binding globulin (SHBG), thereby increasing circulating free androgen levels. Increased androgen activity enhances sebaceous gland enlargement and sebum production, creating an environment favorable for acne formation. Insulin and insulin-like growth factor-1 (IGF-1) also stimulate keratinocyte proliferation and follicular hyperkeratinization, which contribute to comedone formation and inflammatory acne lesions.[3]

Dietary habits have also been implicated in the development of insulin resistance and acne vulgaris. Consumption of high glycemic index foods leads to rapid increases in blood glucose and insulin levels. Elevated insulin promotes increased IGF-1 activity, which further stimulates sebaceous gland function and androgen production. This relationship provides a biological explanation for the association between Western dietary patterns and higher acne prevalence. Studies have shown that low glycemic load diets can improve acne severity by reducing insulin levels and inflammatory activity.[4]

Insulin resistance has been extensively studied in relation to several dermatological disorders, including acanthosis nigricans, psoriasis, hidradenitis suppurativa, and acne vulgaris. Various studies have reported increased fasting insulin levels and elevated Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) values among acne patients compared to healthy controls. Moderate to severe acne has particularly been associated with higher degrees of insulin resistance, suggesting that metabolic disturbances may influence disease severity.[5]

Acne vulgaris is also commonly associated with systemic disorders such as obesity, metabolic syndrome, and polycystic ovarian syndrome (PCOS), conditions in which insulin resistance is a major pathogenic factor. Even in individuals without overt endocrine abnormalities, subclinical insulin resistance may contribute to acne pathogenesis. Therefore, acne may serve as an external marker indicating underlying metabolic dysfunction. Early identification of insulin resistance in acne patients may help prevent future metabolic complications.[6]

The molecular basis linking insulin resistance and acne involves activation of the mammalian target of rapamycin complex 1 (mTORC1) signaling pathway. Increased insulin and IGF-1 levels stimulate mTORC1 activity, leading to enhanced sebaceous lipogenesis, cellular proliferation, and inflammatory responses. Overactivation of this pathway has been implicated in acne development and progression. Understanding these molecular mechanisms has expanded the concept of acne from a localized skin disorder to a condition influenced by systemic metabolic factors.[7]

Assessment of insulin resistance is commonly performed using fasting plasma glucose, fasting serum insulin levels, and calculation of the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). HOMA-IR is considered a simple and reliable method for evaluating insulin sensitivity in clinical practice and research settings. Increased HOMA-IR values have been observed in many patients with acne vulgaris, supporting the hypothesis that insulin resistance contributes to acne pathogenesis.[8]

Recognition of the association between insulin resistance and acne vulgaris has important therapeutic implications. Lifestyle modification, dietary regulation, weight reduction, and physical activity may improve insulin sensitivity and help control acne severity. In selected cases, insulin-sensitizing agents such as metformin have shown beneficial effects in reducing acne lesions, especially in patients with associated metabolic abnormalities. These findings suggest that metabolic evaluation may be useful in the comprehensive management of acne vulgaris.[9]

Despite growing evidence, the exact relationship between insulin resistance and acne vulgaris remains incompletely understood, and available studies show variations across different populations and age groups. Further research is needed to establish the extent of insulin resistance in acne patients and its correlation with disease severity. Evaluating insulin resistance in patients with acne vulgaris may contribute to early diagnosis of metabolic abnormalities and development of more effective treatment strategies. [10]

The aim of this study is to evaluate the presence of insulin resistance in patients with acne vulgaris and to assess its possible association with the severity of acne. The study also focuses on understanding the metabolic profile of acne patients and the role of insulin resistance as an underlying contributing factor.

The main objectives are to determine the prevalence of insulin resistance among patients with acne vulgaris and to compare it with the severity of acne lesions. Additionally, the study aims to analyze the relationship between insulin resistance and

clinical severity of acne, and to highlight the importance of metabolic evaluation in patients with persistent or severe acne vulgaris.

MATERIALS AND METHODS

Study design: Hospital-based cross-sectional observational study.

Study place: Department of Dermatology, Venereology and Leprosy in a Saratchandra Chattopadhyay government. Medical College, Uluberia , Howrah, West Bengal.

Study duration: The study was conducted over a period of 12 months.

Study population: Patients diagnosed clinically with acne vulgaris attending the Dermatology Outpatient Department (OPD).

Sample size: A total of 82 patients.

Study variables:

- Age Group (Years)
- Gender
- Fasting Serum Insulin
- HOMA-IR Status
- Acne Severity

Inclusion criteria:

- Patients aged 15–35 years diagnosed with acne vulgaris.
- Patients willing to participate and provide informed consent.
- Both male and female patients.

Exclusion criteria:

- Patients with diabetes mellitus or other endocrine disorders.
- Patients with polycystic ovarian syndrome, Cushing syndrome, or thyroid disorders.
- Patients receiving systemic steroids, oral contraceptive pills, isotretinoin, or hormonal therapy within the last 3 months.
- Pregnant and lactating women.
- Patients with chronic systemic illness or other dermatological diseases affecting assessment.
- Patients unwilling to participate in the study.

Statistical Analysis:

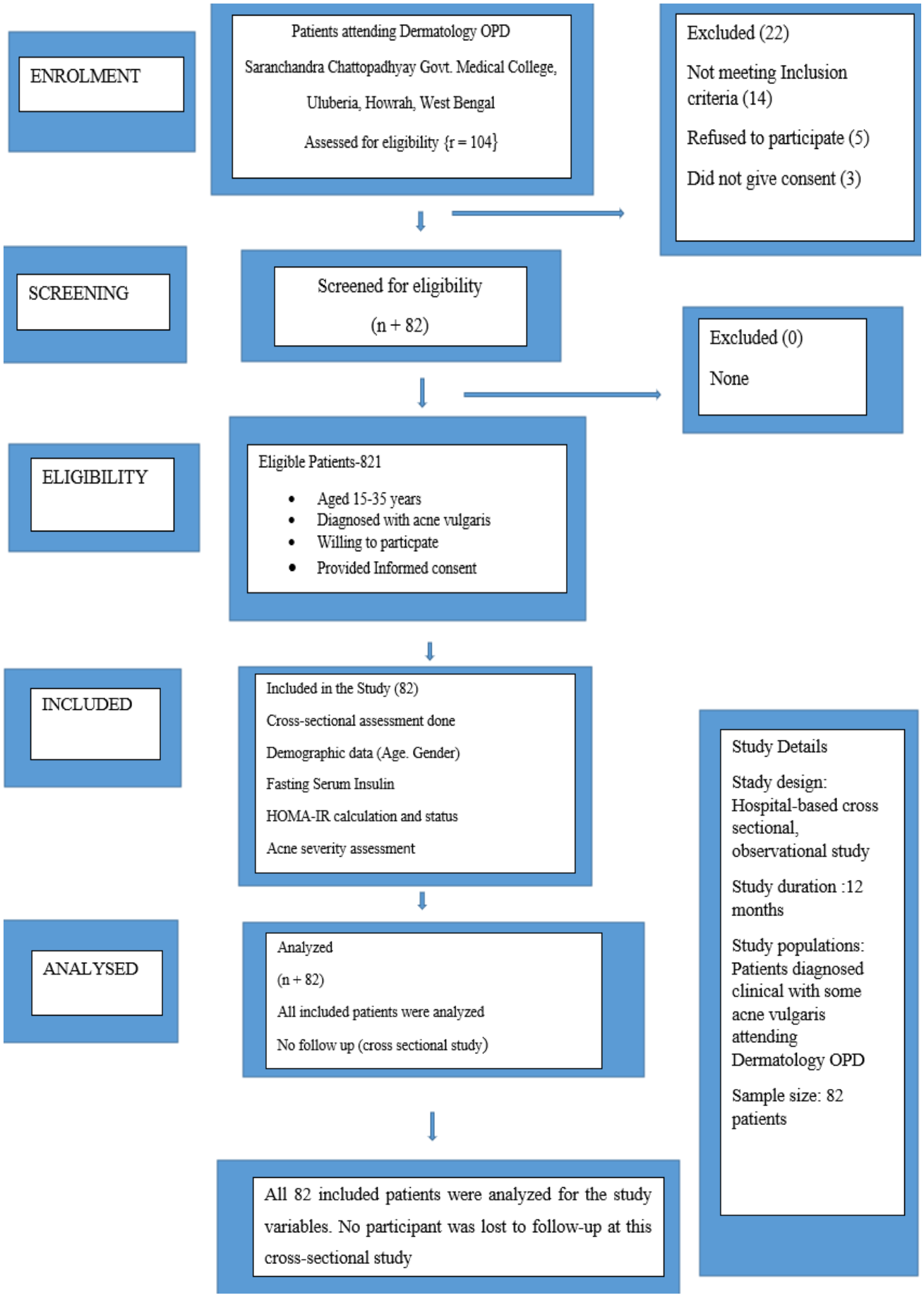
For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. Two-sample t-tests for a difference in mean involved independent samples or unpaired samples. Paired t-tests were a form of blocking and had greater power than unpaired tests. A chi-squared test ($\chi 2$ test) was any statistical hypothesis test wherein the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true. Without other qualification, 'chi-squared test' often is used as short for Pearson's chi-squared test. Unpaired proportions were compared by Chi-square test or Fischer's exact test, as appropriate.

Explicit expressions that can be used to carry out various t-tests are given below. In each case, the formula for a test statistic that either exactly follows or closely approximates a t-distribution under the null hypothesis is given. Also, the appropriate degrees of freedom are given in each case. Each of these statistics can be used to carry out either a one-tailed test or a two-tailed test.

Once a t value is determined, a p-value can be found using a table of values from Student's t-distribution .If the calculated p-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favour of the alternative hypothesis.

CONSORT FLOWCHART

Insulin Resistance in Patients with Acne Vulgaris (Hospital-based Cross-sectional Observational Study)


ENROLMENT
Patients attending Dermatology OPD
Saranchandra Chattopadhyay Govt. Medical College,
Uluberia, Howrah, West Bengal
Assessed for eligibility {r = 104}
Excluded (22)
Not meeting Inclusion criteria (14)
Refused to participate (5)
Did not give consent (3)
SCREENING
Screened for eligibility
(n + 82)
Excluded (0)
None
ELIGIBILITY
Eligible Patients-821
Aged 15-35 years
Diagnosed with acne vulgaris
Willing to particpate
Provided Informed consent
INCLUDED
Included in the Study (82)
Cross-sectional assessment done
Demographic data (Age. Gender)
Fasting Serum Insulin
HOMA-IR calculation and status
Acne severity assessment
ANALYSED
Analyzed
(n + 82)
All included patients were analyzed
No follow up (cross sectional study)
All 82 included patients were analyzed for the study variables. No participant was lost to follow-up at this cross-sectional study
Study Details
Stady design: Hospital-based cross sectional, observational study
Study duration :12 months
Study populations: Patients diagnosed clinical with some acne vulgaris attending Dermatology OPD
Sample size: 82 patients

RESULTS

Table 1. Age Distribution among Study Patients

Age Group (Years)	Acne Patients (n=38)	Controls (n=44)	p-value
15–20	14 (36.8%)	12 (27.3%)	0.412
21–25	13 (34.2%)	15 (34.1%)	0.998
26–30	8 (21.1%)	11 (25.0%)	0.684
31–35	3 (7.9%)	6 (13.6%)	0.438
Total	**38 (100.0%)**	**44 (100.0%)**	

Table 2. Gender Distribution among Study Patients

Gender	Acne Patients (n=38)	Controls (n=44)	p-value
Male	16 (42.1%)	20 (45.5%)	0.761
Female	22 (57.9%)	24 (54.5%)	
Total	**38 (100.0%)**	**44 (100.0%)**	

Table 3. Comparison of Fasting Serum Insulin Levels

Fasting Serum Insulin	Acne Patients (n=38)	Controls (n=44)	p-value
Normal	11 (28.9%)	29 (65.9%)	<0.0001
Elevated	27 (71.1%)	15 (34.1%)	

Table 4. Comparison of HOMA-IR Values between Groups

HOMA-IR Status	Acne Patients (n=38)	Controls (n=44)	p-value
Insulin Resistance Present	25 (65.8%)	12 (27.3%)	<0.0001
Insulin Resistance Absent	13 (34.2%)	32 (72.7%)	
Total	**38 (100.0%)**	**44 (100.0%)**	

Table 5. Association between Acne Severity and Insulin Resistance

Acne Severity	Insulin Resistance Present (n=25)	Insulin Resistance Absent (n=13)	p-value
Mild	5 (20.0%)	7 (53.8%)	0.018
Moderate	12 (48.0%)	5 (38.5%)	0.041
Severe	8 (32.0%)	1 (7.7%)	0.026
Total	**25 (100.0%)**	**13 (100.0%)**	

Figure 1: Comparison of HOMA-IR Values between Groups

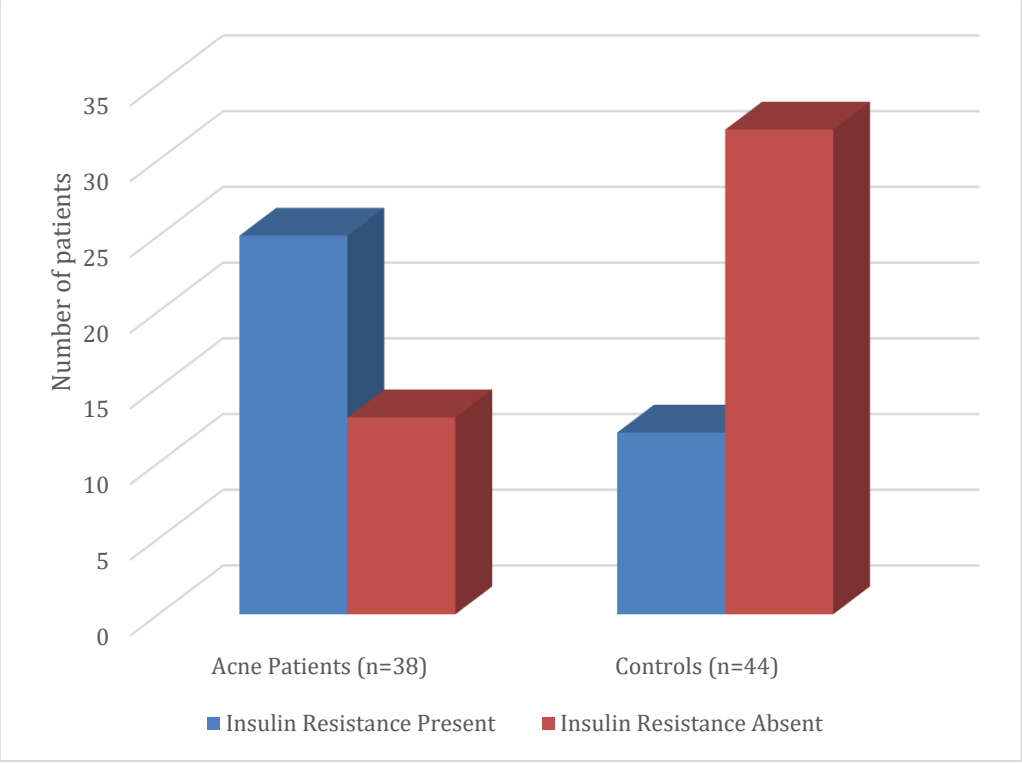
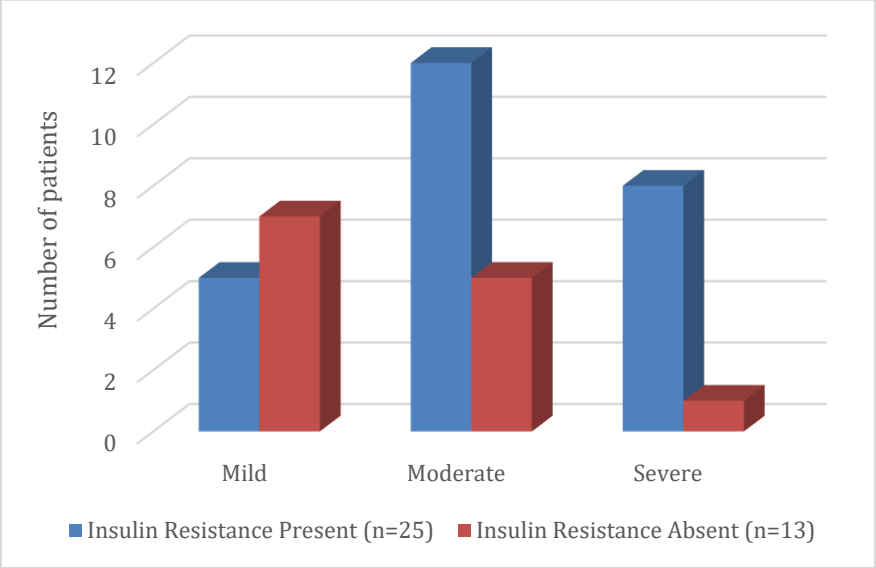


Figure 2: Association between Acne Severity and Insulin Resistance



Among the acne patients (n=38), 14 patients (36.8%) belonged to the 15–20 years age group, whereas among controls (n=44), 12 participants (27.3%) were in the same age group. The difference between the two groups was statistically not significant (p=0.412).In the 21–25 years age group, 13 acne patients (34.2%) and 15 controls (34.1%) were observed. The distribution between the groups was statistically not significant (p=0.998).In the 26–30 years age group, 8 acne patients (21.1%) and 11 controls (25.0%) were included. The difference was statistically not significant (p=0.684).In the 31–35 years age group, 3 acne patients (7.9%) and 6 controls (13.6%) were observed. The difference between the groups was statistically not significant (p=0.438).

Among the acne patients (n=38), 16 patients (42.1%) were males and 22 patients (57.9%) were females. Among the controls (n=44), 20 participants (45.5%) were males and 24 participants (54.5%) were females. The gender distribution between the two groups was statistically not significant (p=0.761).

Among the acne patients (n=38), 11 patients (28.9%) had normal fasting serum insulin levels, while 27 patients (71.1%) had elevated fasting serum insulin levels. Among the controls (n=44), 29 participants (65.9%) had normal fasting serum insulin levels and 15 participants (34.1%) had elevated levels. The difference in fasting serum insulin levels between the two groups was statistically highly significant ($p < 0.0001$).

Among the acne patients (n=38), insulin resistance was present in 25 patients (65.8%) and absent in 13 patients (34.2%). Among the controls (n=44), insulin resistance was present in 12 participants (27.3%) and absent in 32 participants (72.7%). The difference in HOMA-IR status between the two groups was statistically highly significant ($p < 0.0001$).

Among patients with insulin resistance present (n=25), 5 patients (20.0%) had mild acne, 12 patients (48.0%) had moderate acne, and 8 patients (32.0%) had severe acne. Among patients without insulin resistance (n=13), 7 patients (53.8%) had mild acne, 5 patients (38.5%) had moderate acne, and 1 patient (7.7%) had severe acne.The association between mild acne severity and insulin resistance was statistically significant (p=0.018). The association between moderate acne severity and insulin resistance was also statistically significant (p=0.041). Similarly, severe acne showed a statistically significant association with insulin resistance (p=0.026).

DISCUSSION

In the present study, the majority of patients belonged to the 15–25 years age group, with female predominance observed among acne patients. Similar findings were reported by Xiao Z et al who observed that acne vulgaris predominantly affects adolescents and young adults, with increasing prevalence among females in post-adolescent age groups.[11] This similarity may be due to hormonal influences and lifestyle-related factors commonly observed in this age group.

The present study demonstrated that 71.1% of acne patients had elevated fasting serum insulin levels compared to 34.1% of controls, and the difference was statistically highly significant ($p<0.001$). Similar observations were made by Gruszczyńska M et al., who reported significantly higher fasting insulin levels among patients with acne vulgaris compared to healthy individuals. [12] Hyperinsulinemia increases androgen synthesis and sebaceous gland activity, thereby contributing to acne development.

In our study, insulin resistance assessed by HOMA-IR was present in 65.8% of acne patients compared to 27.3% of controls, with a statistically highly significant association ($p<0.0001$). These findings were comparable to the study conducted by Emiroğlu et al., who found significantly elevated HOMA-IR values in patients with severe acne vulgaris. [13] Similar results were also reported by Keerthana KS et al., who demonstrated increased insulin resistance among post-adolescent acne patients. [14]

The present study also found a significant association between acne severity and insulin resistance. Moderate and severe acne were more commonly observed among patients with insulin resistance. This finding is in agreement with the study conducted by Sadowska-Przytocka A et al. who reported that higher HOMA-IR levels were associated with increased acne severity.[15] Increased insulin and IGF-1 activity may promote sebaceous gland proliferation, increased sebum production, and inflammatory responses, thereby worsening acne lesions.

Dietary and metabolic factors have been increasingly implicated in acne pathogenesis. Çerman AA et al. demonstrated that low glycemic load diets improved acne severity and insulin sensitivity among acne patients.[16] Similar findings were reported by Melnik BC et al., who observed clinical improvement in acne lesions following dietary modification aimed at reducing insulin response.[17] These studies support the concept that insulin resistance plays an important role in acne pathophysiology.

The molecular mechanisms linking insulin resistance and acne vulgaris involve increased activity of insulin-like growth factor-1 (IGF-1) and mammalian target of rapamycin complex 1 (mTORC1). Bharti S et al reported that increased insulin and IGF-1 signaling stimulates sebaceous lipogenesis and follicular hyperkeratinization, contributing to acne development.[18] This mechanism may explain the higher prevalence of insulin resistance observed in acne patients in the present study.

Acne vulgaris may also act as a cutaneous marker of underlying metabolic disturbances. Several studies have suggested an association between acne, obesity, metabolic syndrome, and polycystic ovarian syndrome. A study by Aslam MB et al. highlighted the relationship between Western dietary patterns, hyperinsulinemia, and acne prevalence. [19] The current study findings further support the role of metabolic abnormalities in acne vulgaris.

The findings of the present study emphasize the importance of metabolic evaluation in patients with acne vulgaris, particularly those with moderate to severe disease. Early identification of insulin resistance may help in initiating lifestyle modifications and preventive measures to reduce future metabolic complications. In selected patients, insulin-sensitizing therapy may also provide additional therapeutic benefit along with conventional acne treatment.

However, the present study had certain limitations. The sample size was relatively small, and the study was conducted in a single tertiary care center. Longitudinal studies with larger sample sizes are required to establish a definite causal relationship between insulin resistance and acne vulgaris.

Overall, the present study demonstrated a significant association between acne vulgaris and insulin resistance, with higher HOMA-IR values observed among patients with moderate and severe acne. Hasrat NH et al these findings are consistent with previous studies and support the role of metabolic factors in acne pathogenesis . [20]

CONCLUSION

The present study demonstrated a significant association between insulin resistance and acne vulgaris. Patients with acne vulgaris showed higher fasting serum insulin levels and increased HOMA-IR values compared to healthy controls. Insulin resistance was found to be more prevalent among patients with moderate and severe acne, indicating a positive relationship between metabolic dysfunction and acne severity.

The findings of this study suggest that insulin resistance may play an important role in the pathogenesis and progression of acne vulgaris through hormonal and metabolic mechanisms. Early identification of insulin resistance in acne patients may help in recognizing underlying metabolic abnormalities and initiating appropriate lifestyle modifications and therapeutic interventions.

Therefore, assessment of insulin resistance may be considered in patients with moderate to severe acne vulgaris for better disease management and prevention of future metabolic complications.

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