Original Research Article

A comparative study of insulin resistance in acne vulgaris

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ABSTRACT

Background: Acne is common skin problem among adolescents and young adults. Recently the role of insulin resistance in acne is being widely researched. The objectives of the study were to evaluate insulin resistance in acne, to compare the insulin resistance among cases and controls using homeostasis model assessment of insulin resistance
Methods: 45 cases and 45 controls were recruited. Acne severity was graded using the global acne grading system (GAGS). Fasting glucose, fasting insulin levels were done and insulin resistance was assessed using homeostasis model assessment of insulin resistance (HOMA-IR).
Results: We did not find any statistically significant difference in fasting insulin levels between cases and controls. However, a weak positive correlation between acne severity and fasting insulin levels (r =0.3, p=0.04) were observed. Fasting glucose levels and HOMA-IR values observed between cases and controls were not statistically significant (p=0.05, p=0.59 respectively).
Conclusions: Our study did not suggest a major role of insulin resistance in acne.

Keywords: Insulin, HOMA-IR, Acne

INTRODUCTION

Acne is common skin problem among adolescents and young adults. The lesions can range from comedones to nodules and cysts leaving behind scars. It has a significant psychological impact on the patients. Acne is influenced by factors like diet, lifestyle and hormones. Recently the role of insulin resistance in acne is being widely researched.

Acne vulgaris is a multifactorial disease of the skin.[1] Even though acne is considered an androgen‑dependent disease, occurrence of acne doesn't correlate with plasma androgen levels. Increased serum levels of insulin like growth factor (IGF‑1) have been observed in adult women and men with acne, giving rise to the possibility of the role of growth hormone (GH), hyperinsulinemia, and IGF1 in acne. A positive correlation between the mean facial sebum excretion rate and serum IGF‑1 levels has been demonstrated in post-adolescent acne patients.[2]

Some studies have discussed about the correlation between insulin resistance and severity of acne. This was attributed to increased blood glucose which in turn stimulates increased insulin secretion. Increased insulin decreases availability of binding protein for IGF-1 which facilitates the effects of IGF-1 on basal keratinocyte proliferation. Also insulin stimulates synthesis of androgens which can cause acne.[3]

In India, prevalence data from a dermatology clinic in a teaching hospital in Varanasi reported acne in 50.6% of boys and 38.13% of girls in the age group of 12-17 years. There are believed to be no gender differences in acne prevalence, although such difference are often reported and, very likely represent social biases. In clinics in the

urban areas, there is a clear preponderance of girls seeking treatment.[4]

However there is a paucity of studies about insulin resistance in acne among Indian population.

This study has been taken up in view of the changing food habits, lifestyle and the paucity of study of insulin resistance in patients with acne among Indian population.

Objectives

1. To evaluate insulin resistance in acne.
2. To compare the insulin resistance among cases and controls using homeostasis model assessment of insulin resistance (HOMA-IR).

METHODS

This cross sectional study was conducted from September 2015 to August 2016 in department of Dermatology, Vydehi institute of medical sciences and research centre. We recruited 45 cases and 45 controls in the Department of Dermatology, Vydehi Institute of Medical Sciences & Research Centre for our study. Clinically diagnosed acne patients attending dermatology clinic in Vydehi hospital consenting to be part of the study were taken as cases. An equal number of consenting age and sex matched subjects without acne were taken as controls. Informed consent was taken from cases and control subjects.

Inclusion criteria were patients with acne aged more than 12 years attending the dermatology clinic in Vydehi Hospital.

Exclusion criteria were patients with age less than 12 years, other acne (mechanical acne, tropical acne, drug induced acne, cosmetic acne), patients with diabetes mellitus, pregnant and lactating women.

Inclusion criteria for controls were age and sex matched patients without acne who gave consent for the study. Exclusion criteria for controls were not applicable.

A detailed history was taken regarding symptoms, duration, severity, aggravating factors, use of cosmetics, use of medications, occupation. A general physical examination was done which included vitals and BMI. A detailed examination of acne prone areas was carried out.

Examination of other areas of skin, mucosa, hair and nails were carried out. Ten ml of venous blood sample was drawn after overnight fasting and fasting glucose, fasting insulin levels and other relevant investigations when necessary were done.

Insulin resistance was assessed using homeostasis model assessment of insulin resistance.[6]

$$HOMA\text{-}IR = \frac{Glucose \times Insulin}{405}$$

Fasting glucose in mg/dl, insulin in μIU/ml. Values of 2.5 and above were taken as indicator of insulin resistance.[7]

The clinical assessment of acne vulgaris was done using The global acne grading system[5] and graded as follows:

Table 1: The global acne grading system (GAGS).

Location		Factor×Grade (0-4)* =Local score
I	Forehead (F)	2
II	Right cheek (R)	2
III	Left cheek (L)	2
IV	Nose (N)	1
V	Chin (C)	1
VI	Chest and upper back (B)	3

*0: No lesions; 1: ≥one comedone; 2: ≥one papule; 3: ≥one pustule; 4: ≥one nodule

Global score= ∑ (Factor×Grade) F+R+L+N+C+B

Table 2: Global score.

0	None
1-18	Mild
19-30	Moderate
31-38	Severe
>39	Very severe

Statistical analysis

The results were analysed and tabulated using percentages, proportions, student t test, Mann-Whitney U test. Correlation between severity of acne and fasting glucose, fasting insulin, HOMA-IR was calculated using Spearman's rank correlation using SPSS software version 21.

RESULTS

Our study included 45 cases, out of which 26 were females and 19 were males with equal number of age and sex matched controls. The Global Acne Grading System (GAGS) was used for assessing the severity of acne. Among females 7 had mild, 16 had moderate and 3 had severe acne. Among males 4 had mild, 13 had moderate and 2 had severe acne. None of the cases had very severe acne.

Table 3: Severity of acne in males and females.

	Mild	Moderate	Severe
Female	7	16	3
Male	4	13	2

Table 4: Comparison of fasting insulin levels between cases and controls.

Variable	Case		Control		P value
	Mean	SD	Mean	SD	
Fasting insulin	8.0	3.2	6.8	3.3	0.07

Independent samples t test.

Table 5: Comparison of HOMA-IR values between cases and controls.

Variable	Case		Control		P value
	Mean	SD	Mean	SD	
HOMA-IR	1.7	0.7	1.4	0.7	0.05

Independent Samples t test.

**Table 6: Comparison of fasting glucose levels between cases and controls.**

Variable	Case		Control		P value
	Median	IQR	Median	IQR	
Fasting glucose	86	13.5	86	13.5	0.59

Mann-Whitney U test.

Table 7: Correlation between severity of acne and fasting glucose, fasting insulin, HOMA-IR.

| Variable	Correlation coefficient	P value
Fasting glucose	-0.03	0.84
Fasting insulin	0.3	0.04
HOMA-IR	0.28	0.05

Spearmans rank correlation.
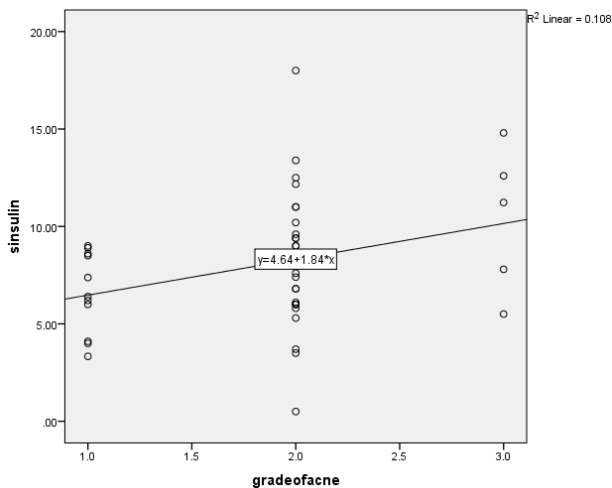
Figure 1: Correlation between severity of acne and fasting insulin.

The mean fasting insulin levels among cases was 8.0 and 6.8 among controls, which showed no statistically

significant difference (p=0.07). Mean HOMA-IR value for cases was 1.7 and 1.4 for controls. The difference among the mean values was not statistically significant (p=0.05). The median for fasting glucose levels for both cases and controls was 86 (p=0.59).

We did not find any significant correlation between acne severity and fasting glucose (r =-0.03) and HOMA-IR values (r =0.28). However, a weak positive correlation between acne severity and fasting insulin levels (r =0.3, p=0.04) were observed.
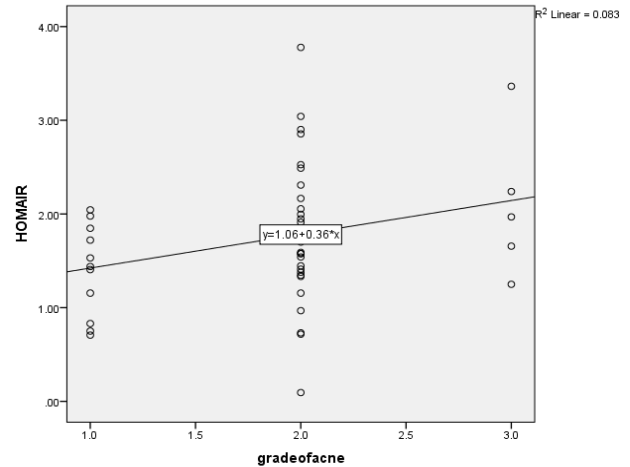
Figure 2: Correlation between severity of acne and HOMA-IR.

DISCUSSION

Acne is a multifactorial disease involving the pilosebaceous units. Altered follicular keratinization, colonization of pilosebaceous units by propionibacterium acnes, increased sebum production, hormonal influences and inflammation have been proposed in pathogenesis of acne vulgaris. IGF-1 has been proposed to be one of the mediators of acne vulgaris.

Acne as a part of syndromes like polycystic ovary (PCOS) and hyper-androgenism insulin resistance-acanthosis nigricans (HAIR-AN) indicate insulin resistance in acne.[8] There is increasing evidence in support of the interplay of growth hormone (GH), insulin and insulin‑like growth factor‑1 (IGF‑1) signalling during puberty, which have a causal role in pathogenesis of acne by influencing adrenal and gonadal androgen metabolism.[2] Hyperglycaemic carbohydrates and insulinotrophic milk/dairy products have been identified to drive acne pathogenesis by promoting increased insulin/ insulin like growth factor-1 signalling.[9] A study by Tasli et al has suggested that insulin like growth factor-1 cytosine–adenine repeat (IGF-1 CA 19) polymorphism may contribute to a predisposition to acne in Turkish patients.[1] A study by Balta et al suggested that insulin resistance may not play a major role in the pathogenesis of post-adolescent acne.[3]

Our study did not find any statistically significant difference in fasting insulin levels between cases and controls. We found a weak positive correlation between acne severity and fasting insulin levels (r =0.3, p=0.04). Balta et al in their study on 35 cases and controls of post-adolescent acne did not find any significant difference in insulin levels between cases and controls. Also they did not find any significant correlation between acne severity and insulin levels.[3]

Some studies have discussed about the positive correlation between blood glucose and severity of acne. This was attributed to increased blood glucose which in turn stimulates increased insulin secretion. Increased insulin decreases availability of binding protein for IGF-1 which facilitates the effects of IGF-1 on basal keratinocyte proliferation. Also insulin stimulates synthesis of androgens which can cause acne.[6] A study by Nagpal et al showed increased blood glucose levels in cases compared to controls.[10] A study by Balta et al has not shown any significant difference in fasting glucose levels between patients with acne and controls.[6] In our study we did not find significant difference in blood glucose between cases and controls. Also there was no correlation between blood glucose levels and severity of acne.

HOMA-IR is widely used as an index of insulin resistance. We have taken 2.5 as cut off value.[7] There was no statistically significant difference between mean HOMA-IR of cases and controls in our study. This is similar to the results of studies by Balta et al.[3] However a study by Nagpal et al showed significantly high values of HOMA-IR in cases with acne compared to controls.[10]

Since acne is a problem in adolescents, the early recognition of insulin resistance might help in better management of acne patients.

CONCLUSION

In our study we did not find any statistically significant association between insulin resistance and acne. We found a weak positive correlation between acne severity and fasting insulin levels. However, this needs to be further studied with a larger sample size.

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