ORIGINAL ARTICLE

Prevalence of Insulin Resistance in Patients Presenting with Acne at a Tertiary Care Hospital, Lahore

SHAKILA YOUSUF[1], ERUM SIDDIQUE[2], SYED MUHAMMAD SAADAT SHAH[3], TOOBA MALIK[4], SANA GUL[5], FARINA ZAMEER[6]

[1]MBBS, FCPS Consultant Dermatologist PNS Shifa Karachi
[2,3] MBBS, FCPS Consultant Dermatologist Abbasi Shaheed Hospital, Karachi
[4]MBBS, FCPS Consultant Dermatologist PNS Shifa Karachi
[5]MBBS, FCPS, Consultant Dermatologist, Fatima Jinnah Medical and Dental College, Karachi
[6]Senior Registrar Dermatology, Jinnah Medical and Dental College, Karachi
Corresponding author: Dr Shakila Yousuf, Email Address: shakilajunaid84@gmail.com, Cell Phone: +923312504657

ABSTRACT

Objective: To determine the prevalence of insulin resistance in patients presenting with acne at Tertiary Care Hospital, in Punjab.

Methods: Cross sectional descriptive study was used in department of Dermatology, Combined Military Hospital, Lahore from 26th March 2016 to 25th September 2016.

A total of 110 patients with acne on the basis of Global Acne Grading System for more than 2 year and on treatment were included in the study. Blood sample was collected for serum fasting blood sugar and fasting insulin level. HOMA-IR >2.5 was labelled as insulin sensitivity. SPSS 21 was used for data analysis. Descriptive statistics were calculated. Stratification was done by applying chi-square test. P≤0.05 was considered as significant.

Results: There were 29 male and 81 female patients. Mean age was 30.80±6.42 years. 30.0% patients were smokers. Insulin resistance was observed in 41(37.3%) patients. No significant association of insulin resistance was observed with gender, age, duration of acne, socioeconomic status, and smoking.

Conclusion: High prevalence (37.3%) of insulin resistance was observed in patients with ACNE, mostly in females and with longer duration of acne.

Keywords: Prevalence, Insulin Resistance, Acne

INTRODUCTION

Acne vulgaris is a widespread skin disease that affects up to 80% of adolescents and many adults at some stage. While acne does not endanger life or weaken physically, it can have a social, physiological or psychological effect. [1] The disease is multifactorial, including genetic causes. The following four factors produce acne as a result of interplay: follicle epidermal hyper-proliferation with subsequent follicle plugging, excess sebum formation, presence and activity of commensal bacterial propionibacterium acne and inflammation. [2] The pilosebaceous units are chronic inflammatory. There is strong evidence that this disorder has a genetic basis. There is also a clear connection between the disease and hormone influences, especially androgens. [3]

The control of sebaceous gland function involves many hormones including androgens, progesterones, insulin and growth hormones such as growth factor [1], corticotrophins that release hormones adrenocorticotropic hormones and melanocortins, and glucocorticoids.[4, 5]

In patients with acne vulgaris, blood glucose levels are shown to be higher, stimulating secretion of insulin and lowering the binding protein for IGF1, thus increasing the cell proliferations. High levels of insulin aggravate acne through the spread of basal keratinocytes.[6-8] Acne is associated with insulin resistance, hyperinsulinemia and hyperandrogenesis in 70% of women with a polycystic ovarian syndrome. Insulin and IGF1 induce androgen synthesis and inhale the liver production from sex hormones binding globulins, which lead to increased levels of androgen binding on the pilosebaceous unit with androgen receptors increasing sebum production. [9-11]

The interplay of growth hormone (gh) insulin and insulin-like growth factor-1 (IGF-1) signals during puberty is being supported by increasing evidence, which have a causal role in acne pathogenesis due to adrenal and gonadal androgen metabolism. Milk intake, hyperglycemic diets, and PI3K mediated IGF-1 induces induced sebaceous lipogenesis, sebocyte, and proliferation of keratinocytes that can worsen acne. Acne in different syndromes also shows that the association between IGF-1 and acne is favored. [12-14] Del Prete et al. observed metabolic syndrome in 36 % of the 22 subjects with acne.[7]

The aim of this study is to determine the prevalence of insulin resistance in acne patients to establish the local perspective as local data are scarce. The current research was therefore intended to determine the prevalence of suspicious patients and to classify it via a routine screening. And if we have discovered insulin resistance to acne, we will be treated with hormone treatment instead of other traditional acne therapies.

SUBJECTS AND METHODS

The cross sectional descriptive studywas conducted at the Department of Dermatology, Combined Military Hospital, Lahore from 26th March 2016 to 25th September 2016. The sample size needed was 110 patients. Both sexes between the age of 20-50 with acne based on the Global Acne Grading System have been included for more than 2 years on care. This study was carried out following approval by the Pakistan University of Physicians and Surgeons. Consent cases, requirements for inclusion were enrolled from the Lahore Ambulatory Dermatology Department, Combined Military Hospital. The institutional

ethical review committee was permitted before the analysis was conducted. Both patients received informed consent to allocate them to the study and use their data during testing. A brief acne history has been taken. After a quick overnight sample for serum fasting blood sugar and fasting insulin level was obtained in a sterile manner and resistance to HOMA-IR >2.5 patients with HOMA-IR >2.5 were determined to be insulin resistant. The researchers have proformaed the findings of the variables as described above.

Data was analyzed in version 21 of SPSS. Mean and standard deviation for age and length of acne were determined. Sex, socio-economic status, smoking and insulin resistance (yes/no) were measured in the frequency and percentages. In order to see its impact on the outcome variable, effect modifiers were regulated using age, gender, period of acne, socio-economic status and smoking status stratification. Post stratification chi square test was applied taking p-value of ≤ 0.05 as statistically significant.

RESULTS

The results showed that there were 29 male and 81 female patients. The mean age was 30.80±6.42 years. The age was stratified in two groups. The age of 49 patients was ≤30 years and age of 61 patients was > 30 years. The results about socioeconomic status showed that monthly income of 42 patients was ≤10,000, 39 patient had monthly income 11,000 – 40,000, and monthly income of rests of the 29 patients was >40,000. The smoking status showed that 30.0% patients were smokers.

The final outcome i.e. insulin resistance was observed in 41(37.3%) patients.

Table 1.Summary of respondent characteristics

Variable		Frequency	Percentage
Gender	Male	29	26.4%
	Female	81	73.6%
Age	≤ 30 years	49	44.5%
	>30 years	61	55.5%
Socio economic status	≤ 10,000per month	42	38.2%
	11,000-40,000 per month	39	35.5%
	>40,000 per month	29	26.4%
Smoking	Yes	33	30.0%
	No	77	70.0%
Insulin Resistance	Yes	41	37.3%
	No	69	62.7%

Table 2.Descriptive statistics

	Age (years)	Duration of acne (years)	Age (years) According to Insulin Resistance Yes (41)No(69)		Duration of Acne (years) According to Insulin Resistance Yes (41) No (69)	
Mean ±SD	30.80±6.42	5.94±1.80	29.83±6.09	31.38±6.58	5.90±1.67	5.96±1.89
95%CI (LB – UB)	29.59 – 32.01	5.60 – 6.28	27.90 – 31.75	29.80 – 32.96	5.38 – 6.43	5.50 – 6.41
Median (IQR)	32.00 (10)	6.00 (3)	30.00 (10)	32.00 (12)	6.00 (2)	6.00 (4)
Range	24	6	23	24	6	6
Minimum	20	3	20	20	3	3
Maximum	44	9	43	44	9	9

Table 3: Frequency and association of insulin resistance according to gender, age groups and duration of acne

		INSULIN RESISTANCE		Total	P-value
		Yes (n=41)	No (n=69)		
Gender	Male (n=29)	7	22	29	0.088**
	Female (n=81)	34	47	81	
Age groups	≤ 30 years (n=49)	21	28	49	0.278**
	> 30 years (n=61)	20	41	61	
Duration of acne	≤ 4 years (n=25)	8	17	25	0.535**
	> 4 years (n=85)	33	52	85	
Total		41	69	110	

Chi square test was applied, P-value ≤0.05 was considered as significant
** Not Significant at 0.05 levels

The descriptive statistics about age and duration of acne were calculated according to insulin resistance. It was observed that mean age was 29.83±6.09 years and mean duration of acne was 5.90±1.67 years, among patients who were found with insulin resistance. Stratification with respect to gender, age, duration of acne, socioeconomic status, and smoking was done to observe effect of these modifiers. The detailed descriptive statistics are presented in Table-2

Post stratification Chi square test was applied and p-value ≤0.05 was considered as significant. The results showed that no significant association of insulin resistance was observed with gender (p=0.088), age (p=0.278), duration of acne (p=0.535), socioeconomic status (p=0.936), and smoking (p=0.322). The detailed results about frequency and association are presented in Table3.

DISCUSSION

Multifactorial acne vulgaris also confers severe psychosocial morbidity and, occasionally, death. [15] Acne is caused by several causes, including increased androgen secretion, increased sebum development, follicular hypercheratination, Propionibacterium acnes microbial colonization, and inflammation. [2] It is suspected that other causes, such as diet, sun exposure, poor grooming, stress and genetics cause or exacerbate acne symptoms. [16]

In a study 56.67% of patients claimed that diet was an important factor for acne, with fat/oil-rich food the most involved food. A proportion of 46.67% thought bad grooming was caused by acne, 40% thought it to be the cause of biology, 33.33% believed it was stressful, and 26.67% felt that sun exposure worsens acne. [17]

Acne is commonly found at locations rich in pilosebaceous units.[15] In a study conducted by Katsambas et al., the effects of face were observed in all patients with acne vulgaris (100 %), face and back at 76.67 %, face and chest at 21 %, and face and neck lesions were observed in 9 percent. The exam reveals both inflammatory lesions (paples, pustules, nodules) and noninflammatory lesions (comedones, open and closed). These conclusions are based on data from previous studies. [18]

Peter et al. found that in patients with acne vulgaris blood glucose levels were substantially higher than in stable controls. This is mainly because the rise in blood glucose levels promotes insulin secretion, which reduces the binding protein of IGF-I, which facilitates the effect of IGF-I on cell proliferation. This may contribute to increased basal keratinocytes in an irregular follicular corneocyte desquamation in the pilosebaceous canal. Insulin can also increase androgen development and activity as the levels of sex hormone binding globulin decrease and thus facilitate acne formation. [19]

Insulin's role in the development of acne is also confirmed by its high prevalence in women with PCOS, a condition associated with insulin resistance, hyperinsulinemia and hyperandogenicity. Resistance to insulin is supposed to be the basis of PCOS disorder, since it generally precedes and causes the cluster of endocrine abnormalities which characterize PCOS (high concentrations of andrhogen and IGF-I and low sex hormonal globulins. [17]

A research by Balta et al has shown that post-adolescent acne is not linked to resistance to insulin. Furthermore, there were no major discrepancies between eating or fasting blood glucose levels or HOMA-IR and acne intensity. [14] Kaymak et al. have identified no major differences in serum glucose, insulin and HOMA-IR in younger acne vulgaris and control patients. Furthermore, they found no statistically significant connection between acne intensity and insulin resistance. [20]

The occurrence of acne has been linked rather than to changes in androgen levels to insulin and IGF-1 circulation. In both puberty and adolescence, insulin sensitivity is reduced, associated with a rise in IGF-1 and serum insulin levels and a decrease in SHBG and serum IGFBP-1. Insulin and IGF-1 in particular peak during late puberty and decrease steadily until the third decade. Acne starts at the same time as pre-adolescent growth in plasma insulin, IGF-1 and BMI and, despite circulating androgens, resolves usually at the end of puberty. [21]

Smith et al. suggests that increases in dietary glycemic load can increase both IGF-1 and sex hormones' biological activity, which suggests that carbohydrate-rich dietary regimen may trigger potentially acne-development factors.[21]

Two classes of male subjects with resistant acne and pair controls were considered irrespective of the type of diet. The BMI, SBP, DBP, HOMA-IR, and Serum Insulin levels have increased significantly in all subjects with acne and the HDL cholesterol values have decreased significantly. In most cases, young males with acne displayed a poor metabolic profile and reduced insulin sensitivity resulting in 36 percent metabolic syndrome, whereas in this population 32 percent had already had a family history of type 2 diabetes mellitus showing an imbalance in glucose/insulin metabolism For women with PCOS with hyperinsulinemia and hyperandrogenism [22], the androgenic profile has been shown to be common in all men with acne, meaning that acne may be affected by hyperinsulinemia, but not by androgen activity in these patients. In females with acne, a relationship between low HDL cholesterol and insulin resistance was identified. [17]

Methods such as quick-insulin amount, fast-glucose-insulin ratio (FGIR), insulin resistant homeostasis model (HOMA-IR) and insulin-sensitivity quantitative index (QUICKI) should be used in population studies.[23]

HOMA-IR is a widely used clinical research parameter. [23] Although this validated procedure is used for the assessment of insulin resistance in many different countries, cut-off points vary from country to country. [24] In a Brazilian analysis, the cut-off value of HOMA-IR was recently calculated at 2,7,24 which is the same value agreed in accordance with the Turkish Metabolic Syndrome Guides. [25]

In a study by Emiroğlu et al, a positive association between resistance to insulin and acne vulgaris was found because the disparity in HOMA values was highly important between patients and control groups (p<0.001, 2.87±2,56 vs. 1.63±0.65).[26]

This research included two hundred and forty-three acne vulgaris and 156 healthy people. According to their global acne ratings, both patients were in the acne

category. Although there was no difference between groups in the fasting blood glucose levels (p >0.05, 82.01±9.76 versus 80.26±8.33), in the patient group the rapid insulin level was considerably higher than in the control group (p <0.001, 14.01±11.94 versus 9.12±3.53). In addition, the difference in HOMA values between patients and control groups was very large (p < 0.001, 2.87 ±2.56, vs 1.63 ±0.65). [26]

The role of androgens in the activity of the sebaceous gland is well known. Studies have shown that women with acne have plasma Androgen (although still within normal range) substantially higher levels than females without acne[27]. Insulin increases the proliferation of human sebocytes substantially.[28]

CONCLUSION

The results showed a high prevalence of insulin resistance (37.3%) in patients with ACNE, especially in women with longer acne length. This correlation between resistance to insulin and acne suggests that insulin resistance therapy could be worth studying for acne vulgaris therapy.

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