The Relationship between Blood Lipid Profile and Acne in Non-obese, Non-PCOS Patients

Sheikh Manzoor[1], Suhail Rather[2], Syed Shahab-ud-din[3], Farah Sameen[4], Samia Aleem[5], Shazia Jeelani[6]

ABSTRACT

Introduction: Acne vulgaris is a common skin disorder affecting adolescents and young adults. It`s pathophysiology includes increased sebum production and sebum analysis in some studies have shown increased triglycerides and wax/cholesterol esters in acne patients.But is this local alteration of surface lipids and seborrhoea associated with any alteration of lipid profile in acne patients.

Material and methods: 150 acne patients and 80 age and sex matched controls were included in study. Fasting lipid profile of acne patients was compared with controls. GAGS was used for grading acne.

Results: The predominant acne grade was grade I (54.67%). 45.34% of Acne patients had deranged lipid profile compared to 30% in controls. Total cholesterol was raised in 17.33% of Acne patients compared to 5% in controls, while the HDL was low in 10.67% of Acne patients compared to 10% of controls. LDL was raised in 8% of Acne patients and 2.5% of the controls. Triglycerides were raised in 33.34% of Acne patients and 20% of controls. Total cholesterol /HDL ratio was raised in 34.67% of Acne patients compared to 15% of controls. There is a statistically significant difference in the level of Total cholesterol, VLDL, triglyceride and total cholesterol: HDL ratio between acne patients and controls. There is a statistically significant rise in levels of LDL, VLDL, Triglyceride and TC/HDL and decrease in levels of HDL with the severity of acne.

Conclusion: Acne patients are more likely to have some abnormality in lipid profile, particularly patients with severe acne. The abnormal lipid profile should be considered in disease pathogenesis and maybe even in treatment.

Keywords: Acne, lipid profile, severity, non-PCOS, non-obese

INTRODUCTION

Acne is chronic inflammatory disease of pilosebaceous unit and is clinically characterized by seborrhoea, open and closed comedones, papules and pustules and in severe cases nodules, deep pustules and pseudocysts. The condition starts after puberty and is a common skin disorder in adolescents and young adults.[1] Though excess sebum production is considered prerequisite for development of acne other factors involved in the pathogenesis include hypercornification of the pilosebaceous duct, colonization of pilosebaceous duct with Propionibacterium acnes and local release of inflammatory mediators.[2] Acne subjects not only excrete more sebum the secreation rates have been seen to correlate well with the severity of acne.[3] Triglycerides (40-60%), wax esters (19-26%) and squalene (11-15%) are major components of sebum.[4] Squalene which is a non-polar hydrocarbon and most unsaturated molecule is present in unusually high levels in sebum.[5] Decreased concentration of linoleic acid has been observed in skin surface lipids of acne patients.[6] It seems that β-oxidation of linoleic acid is specific of sebocytes and that it is correlated with their differentiation.[7] Sebum analysis in some studies have shown increased triglycerides and wax/cholesterol esters in acne patients.[8] But is this local alteration of surface lipids and seborrhoea associated with any alteration of lipid profile in acne patients. Alteration in lipid profile of acne patients is not well known, but few studies in both male and female acne patients have shown some alterations. The aim of this study is to evaluate the association of acne with lipid profile.

MATERIAL AND METHODS

The study was conducted in Sher-i-Kashmir Institute of Medical sciences Hospital. 150 cases of acne in age group of 12-35 years and 80 age and sex matched healthy controls attending Dermatology Clinic between 1st August 2015 to 1st October 2015 were included in the study.

Exclusion criteria

1. Obesity
2. History of cardiovascular disease
3. History of dyslipidemia or drugs that affect lipid metabolism
4. Female subjects with PCOS, history of oral contraceptives or hormonal therapy

Informed consent was taken from the subjects. Ethical clearance was obtained from the institute's ethical clearance committee. Acne grading for each patient was performed by only one dermatologist based on Global Acne Grading System (GAGS).[9]

This system considers six locations on the face, chest and upper back, with a factor for each location based roughly on the affected surface area, distribution and density of pilosebaceous units. Each grade was calculated as the sum of the local scores for the face, chest and upper back [Table 1]. The subjects chosen in this study were interviewed, and each completed consent and a questionnaire form that contained information about their age, sex, weight, height, personal history of acne, personal or family history of dyslipidemia,

[1]Associate Professor and Head, [2]Senior Resident, [3]Senior Resident, [4]Assistant Professor, [5]Senior Resident, [6]Senior Resident, Department of Dermatology, Sher-i-Kashmir Institute of Medical Sciences- Medical College and Hospital, Srinagar, J and K, India

Corresponding author: Suhail Rather, Jawahar-nagar, Government Quarter No. J11, Srinagar, J and K, India

How to cite this article: Sheikh Manzoor, Suhail Rather, Syed Shahab-ud-din, Farah Sameen, Samia Aleem, Shazia Jeelani. The relationship between blood lipid profile and acne in non-obese, non-PCOS patients. International Journal of Contemporary Medical Research 2016;3(4):1096-1099.

Location	Factor
Forehead	2
Left cheek	2
Chin	1
Right cheek	2
Nose	1
Chest and upper back	3
Calculation: Each type of lesion is given a value 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. A score of 1-18 is considered mild, 19-30, moderate; 31-38, severe; and>39, very severe	

Table-1: The global acne grading system

Parameters	Controls	Acne patients	P-value
TC	151.25±32.40	167.87±38.04	0.0011*
HDL	50.10±12.72	50.4±9.66	0.8414
LDL	78.45±16.56	80.49±31.16	0.5859
VLDL	20.65±5.99	25.04±12.91	0.0044*
TG	112.70±36.62	127.45±59.91	0.0456*
TC/HDL	3.18±0.81	3.43±0.96	0.0486*
Data are presented as mean±SD, *represents P<0.05.			

Table-2: Comparison of lipid profile between controls and acne patients.

before blood samples were collected. Subjects were fasting 12–14 hr at the time of blood withdrawal. Venous blood specimens were collected in ethylenediaminetetraacetic acid (EDTA) tubes, then immediately centrifuged using low speed refrigerated centrifuge 1500. In suspected cases of PCOS hormonal profile and ovarian ultrasonography was done. The American Association of Clinical Endocrinologists (AACE), American College of Endocrinology (ACE), and Androgen Excess and PCOS Society (AES) released guidelines were used in the diagnosis of PCOS.[10] Patients with Body Mass Index more than 30 kg/m^2 were excluded from the study.

Dyslipidemia: National Cholesterol Education Programme (NCEP) guidelines[11] were used for definition of dyslipidemia as follows:

Hypercholesterolemia – serum cholesterol levels ≥200 mg/dl (≥5.2 mmol/l).

Hypertriglyceridemia – serum triglyceride levels ≥150 mg/dl (≥1.7 mmol/l).

Low HDL cholesterol – HDL cholesterol levels <40 mg/dl (<1.04 mmol/l).

High LDL cholesterol – LDL cholesterol levels ≥130 mg/dl (≥3.4 mmol/l) calculated using the Friedewald equation.

High VLDL–>50mg/dl

High total cholesterol to HDL-C ratio: This is defined as total cholesterol to HDL ratio of ≥4.5.

TC, HDL, and TG were determined using an enzymatic colorimetric test, which measured oxidase and peroxidase activities. LDL and VLDL values were calculated by the Friedewald formula

STATISTICAL ANALYSIS

Results were collected, tabulated and statistically analyzed by SPSS version 11. Data were described in terms of mean ± SD, frequencies and relative frequencies. For comparison Student`s t-test and ANOVA (F-test) was used. P< 0.05 was considered to be statistically significant.

RESULTS

230 subjects, aged from 12 to 35 years, were enrolled in this study. One hundred and fifty were newly diagnosed untreated acne patients from both sexes, attending Dermatology Clinic in Sher-i-Kashmir Institute of Medical sciences Hospital, with 80 age and sex matched healthy controls.

Age of the subjects varied between 12 to 35 years. The predominant acne grade was grade I (54.67%), followed by grade II (40%) and grade III (5.33%). 45.34% of Acne patients had deranged lipid profile compared to 30% in controls. Total cholesterol was raised in 17.33% of Acne patients compared to 5% in controls, while the HDL was low in 10.67% of Acne patients compared to 10% of controls. LDL was raised in 8% of Acne patients and 2.5% of the controls. VLDL was raised in 8% of Acne patients but none of controls. Triglycerides were raised in 33.34% of Acne patients and 20% of controls. Total cholesterol /HDL ratio was raised in 34.67% of Acne patients compared to 15% of controls. There is a statistically significant difference in the level of Total cholesterol, VLDL, triglyceride and total cholesterol: HDL ratio between acne patients and controls, as shown in Table 2.

The lipid profile, particularly Triglycerides showed progressive derangement with the severity of Acne. There is a statistically significant rise in levels of LDL, VLDL, Triglyceride and TC/HDL and decrease in levels of HDL with the severity of acne, as shown in Table 3, Fig. 1.

DISCUSSION

Acne vulgaris is a chronic inflammatory disease of pilosebaceous unit. It has a multifactorial pathogenesis, but excess sebum production is considered prerequisite for the development of acne.[4]

Current study showed increase in levels of total cholesterol, VLDL, triglycerides and TC/HDL ratio in acne patients compared to controls. HDL and LDL were comparable in both groups. Jaing et al in their study found increased ratio of total cholesterol and triglycerides in acne patients.[12] Similarly Abulnagaand Arora et al in their study conducted on female patients found increased levels of cholesterol in acne patients.[13,14] But unlike these studies LDL levels were comparable in both groups in our study, the finding consistent with study conducted by Vergani et al.[15]

Almost all cholesterol enters sebocytes by the LDL receptor mediated endocytosis and it`s synthesis within sebocytes is interrupted at the level of squalene.[16] Increased serum Total cholesterol levels may affect the development of acne vulgaris by increasing androgens, as both adrenal and gonadal androgens are synthesized from plasma cholesterol.[14] Exogenous fatty acids from lipoproteins is released within cells by lipoprotein lipase, which has been shown to be expressed in sebaceous glands at mRNA level, de novo synthesis oc-

Parameters	Grade I	Grade II	Grade III	P-value
Total cholesterol	161.05±36.11	176.37±39.78	174.00±34.04	0.053
HDL	52.90±9.42	47.47±9.67	46.75±2.96	0.002*
LDL	77.80±23.4	92.80±38.09	88.25±22.64	0.014*
VLDL	21.76±11.26	29.63±14.02	25.75±8.95	0.001*
triglyceride	110.78±45.57	143.93±71.04	174.75±41.05	0.000*
TC/HDL	3.14±0.91	3.80±0.91	3.74±0.82	0.000*
Data are presented as mean ± SD, * represents P<0.05.				

Table-3: Comparison of mean lipid levels in different grades of acne.

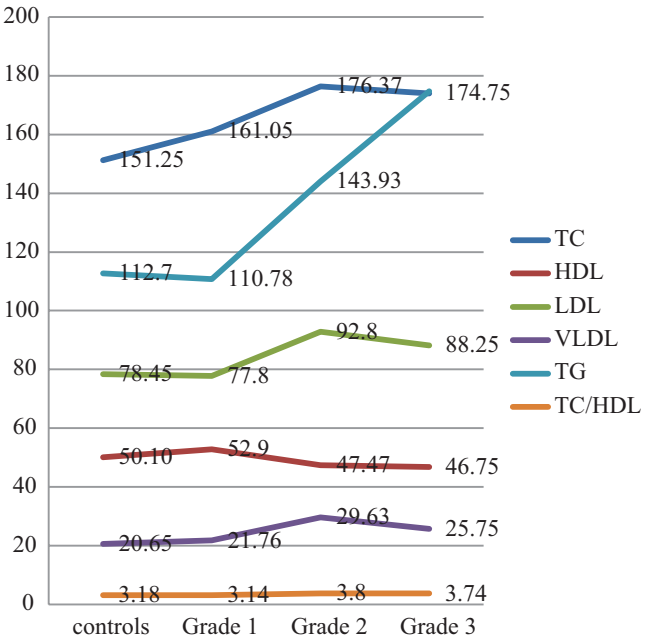

Figure-1: Change in mean levels of lipids with severity of acne

curs to lesser extent. Free fatty acids are mostly translocated to the cytoplasm through active mechanism involving a six member Fatty Acids Transport Protein (FATP) family, in particular FATP4. FATP4 also acts as very long-chain acyl-CoA synthetase, finding which implies that sebaceous glands have capacity to sequester dietary cholesterol and fatty acids from serum.[16] There are also studies claiming that sebum production is increased by the consumption of dietary fat.[17]

In our study both triglyceride and VLDL were raised in acne patients compared to controls. VLDL cholesterol is produced in liver and released in bloodstream to supply body tissues with triglyceride. In a study conducted by Apostolas et al it was seen that triglycerides are increased in the sebum of acne patients.[8]

In current study except total cholesterol all parameters showed statistically significant rise with severity of acne, particularly triglycerides and TC/HDL ratio. HDL levels showed statistically significant decrease with severity of acne. Vergani and Finzi et al had similar observation in their study where patients with severe acne had significantly reduced levels of HDL compared with healthy individuals.[15] Similar observations were made by Cunha et al in their study on female patients.[18] Pigatto et al in their study found that HDL levels even returned to normal at the end of treatment with Isotretinoin.[19]

CONCLUSION

In conclusion, acne patients are more likely to have some abnormality in lipid profile, particularly patients with severe acne. The abnormal lipid profile should be considered in disease pathogenesis and maybe even in treatment.

REFERENCES

1. Thiboutot D, Gollnick H, Bettoli V, Dréno B, Kang S, Leyden JJ, et al. New insights into the management of acne: An update from the Global Alliance to Improve Outcomes in Acne group. J Am AcadDermatol 2009;60:S1-50.
2. Kurokawa I, Danby FW, Ju Q, Wang X, Xiang LF, Xia L, et al. New developments in our understanding of acne pathogenesis and treatment. Exp Dermatol. 2009;18:821-832.
3. Cunliffe WJ. In: Acne.Dunitz M, editor. London: Martin Dunitz; 1989.
4. Zouboulis CC, Akamatsu H, Stephanek K, Orfanos CE. Androgens affect the activity of human sebocytes in culture in a manner dependent on the localization of the sebaceous glands and their effect is antagonized by spironolactone. Skin Pharmacol 1994;7:33-40.
5. Stewart ME. Sebaceous glands lipids. Seminars in Dermatology 1992;11:100-5.
6. Stewart ME, Grahek MO, Cambier LS, Wertz PW, Downing DT. Dilutional effect of increased sebaceous gland activity on the proportion of linoleic acid in sebaceous wax esters and in epidermal acylceramides. J Invest Dermatol. 1986;87:733–736.
7. Downing DT, Stewart ME, Wertz PW, Strauss J. Essential fatty acids in acne. J Am AcadDermatol. 1986;14:221–225.
8. Pappas A, Johnsen S, Liu JC, Eisinger M. Sebum analysis of individuals with and without acne. Dermatoendocrinol. 2009;1:157-61.
9. Doshi, A., Zaheer, A. and Stiller, M. J. A comparison of current acne grading systems and proposal of a novel system.Int. J. Dermatol. 1997;6,416–418.
10. Goodman NF, Cobin RH, Futterweit W, Glueck JS, Legro RS, Carmina E. American Association of Clinical Endocrinologists, American College of Endocrinology, and Androgen Excess and PCOS Society disease state clinical review: guide to the best practices in the evaluation and treatment of polycystic ovary syndrome - part 1. EndocrPract. 2015;11:1291-300.
11. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) (2001) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 285:2486–2497.
12. Jiang H, Li CY, Zhou L,Lu B, Lin Y, Huang X,et al.Ac-

ne patients frequently associated with abnormal plasma lipid profile.J Dermatol. 2015.
13. Abulnaja KO. Changes in the hormone and lipid profile of obese adolescent Saudi females with acne vulgaris. Braz J Med Biol Res 2009;42:501-5.
14. Arora MK, Seth S, Dayal S. The relationship of lipid profile and menstrual cycle with acne vulgaris. ClinBiochem 2010;43:1415-20.
15. Vergani C, Finzi AF, Pigatto PD, Vigotti G, Negri M, Altomare G. Low levels of HDL in severe cystic acne. N Engl J Med 1982;307:1151-2.
16. Zouboulis CC, Katsambas A, Kligman AM. Pathogenesis and Treatment of Acne and Rosacea. New York, Dordrecht, London: Springer; 2004. 308p.
17. Llewellyn A. Variations in the composition of skin surface lipid associated with dietary carbohydrates. Proc Nutr Soc. 1967;26:11.
18. da Cunha MG, Batista ALF, Macedo MS, Machado Filho CAS, Fonseca FLA.Study of lipid profile in adult women with acne. ClinCosmetInvestigDermatol. 2015; 8: 449–454.
19. Pigatto PD, Finzi AF, Altomare GF, Polenghi MM, Vergani C, Vigotti G. Isotretinoin versus minocycline in cystic acne: a study of lipid metabolism.Dermatologica. 1986;172:154-9.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 19-02-2016; **Published online**: 20-03-2016