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Original Research Paper

Dermatology

# "Evaluation on whether Psoriasis Severity is Affected by the Lipid Profile in Indian Patients: A Hospital Based Study"

**Col. (Dr) Mahendra Singh Deora**

Associate Professor, Department of Dermatology, Armed Forces Medical College, Pune-411040 (Maharashtra)

**ABSTRACT**

Psoriasis is a lifelong chronic inflammatory skin disorder characterized by increased T helper cell activity and associated with abnormal lipid metabolism and reduced differentiation of keratinocytes. Aim of study was to evaluate the lipid profile status in patients with psoriasis and compare with healthy controls and whether it affects the severity of psoriasis. In conclusion we found that cholesterol, LDL-c and triglyceride levels are as reported previously to be higher in psoriasis patients while HDL-c levels are as reported previously to be low which makes a difference in the severity of psoriasis.

**KEYWORDS** : Psoriasis, Lipid Profile & Dyslipidemia.

## Introduction:

Psoriasis is a lifelong chronic inflammatory skin disorder characterized by increased T helper cell activity and associated with abnormal lipid metabolism and reduced differentiation of keratinocytes affecting up to 1-2% of the world's population.[1]Increased risk of cardiovascular abnormalities, hypertension, dyslipidemia, atherosclerosis, type2 diabetes mellitus, obesity, cerebral stroke, osteoporosis, cancer, and depression were noticed in psoriatic patients.[2]Psoriasis vulgaris has not only been associated with several comorbidities like metabolic disorders and cardiovascular diseases, but also with neurologic disorder like multiple sclerosis. Our aim was to evaluate the lipid profile status in patients with psoriasis and compare with healthy controls and whether it affects the severity of psoriasis.

## Material and Methods:

The study was carried out on 35 psoriatic patients and 35 healthy controls of age group 20-65 yrs who attended OPD of department of Dermatology, Command Hospital (Southern Command), affiliated teaching hospital of Armed Forces Medical College, Pune during the period from February 2013 to January 2014. A consent was taken from each patient and control before the study. All patients were subjected to full history taking, clinical dermatological examination, Psoriatic Area and Severity Index (PASI scoring).[3]Patients with diseases that can cause secondary hyperlipidemia were excluded from study such as hypothyroidism, diabetes mellitus, nephrotic syndrome, chronic renal insufficiency, obstructive liver disease, and connective tissue disorders. Also Patients on medications such as beta blockers, thiazides, corticosteroids, retinoids and cyclosporine were excluded from the study.

## Biochemical Analysis:

An overnight fasting blood sample was collected under all aseptic precautions. 5-8 ml of blood was collected and was analysed by various methods[4] for following parameters:

1. Total Cholesterol (TC) by enzymatic end point CHOD-POD method.
2. Triglyceride (TG) by enzymatic glycerol phosphate oxidase/ peroxidase method.
3. HDL-Cholesterol by direct enzymatic end point method.
4. LDL-Cholesterol by Friedewald's formula.
5. VLDL-Cholesterol by Friedewald's equation method.

*LDL-c = Tc-HDL-c(TG/5)*

## Statistical Analysis:

All values were expressed as mean±sd. We used student t-test to find the statistical significance. A p-value of <0.05 was considered as statistically significant.

## Result and Discussion:

The present study was conducted on 35 patients with psoriasis and 35 healthy volunteers served as a control group. Table-1 shows that mean total cholesterol, LDL cholesterol and triglyceride levels were elevated and the mean HDL cholesterol level was reduced in patients as compared to control group and the differences were highly significant (P<0.01).

**Table 1: Comparison of various parameters between psoriatic patients and control group:**

| Parameters | Controls (n=35) (mean±sd) | Cases (n=35) (mean±sd) | P-value |
|---|---|---|---|
| Total Cholesterol(mg/ dl) | 159±3.02 | 229±1.09 | <0.01 |
| Triglycerides (mg/ dl) | 98±2.04 | 128±4.3 | <0.01 |
| HDL-c(mg/ dl) | 38±1.02 | 32±3.4 | <0.01 |
| LDL-c(mg/ dl) | 101±7.2 | 142±3.0 | <0.01 |

**Statistically Significant (P<0.05)*

**Table 2: Comparison of lipid levels and severity of disease:**

| Parameters | TC (mean±sd) | TG (mean±sd) | HDL-c (mean±sd) | LDL-c (mean±sd) |
|---|---|---|---|---|
| Mild (n=17) | 182 ± 3.07 | *141.4 ± 21 | 32.6 ± 5.02 | 121 ± 6.03 |
| Moderate (n=16) | 205 ± 21.06 | *154.2 ± 3.2 | 29.03 ± 4.0 | 140 ± 26..03 |
| Severe (n=2) | 248 ± 12.4 | *167 ± 13.05 | 25.08 ± 6.1 | 170.9 ± 34.2 |
| P-value | 0.01 | 0.21 | 0.01 | 0.01 |

**Statistically Significant (P<0.05)*

Table-2 shows that there is a relation between total cholesterol level in case group and degree of disease severity (P=0.01). Furthermore, a similar result was observed for HDL-c (P=0.01) and LDL-c (P=0.01) while no significant relation between disease severity and triglyceride level was proved (P=0.21). Psoriasis is a chronic inflammatory skin disease characterized by increased T helper-1 and T helper-17 cells activity.[5] Complex network of cytokines and chemokines mediate the pathological reaction, whereas the abnormal function of psoriatic regulatory T cells is responsible for the chronic nature of psoriasis.[6]A number of conflicting findings have been reported about the various parameters of lipid profile studied among psoriatic patients, with some studies reporting high levels, and some reporting normal levels across a number of the same measures.

We found significantly higher levels of total cholesterol, triglycerides and LDL-cholesterol while lower HDL-cholesterol levels were observed in patients of psoriasis as compared to controls and there was a highly significant difference in the levels between the two groups(p<0.01). Considering the severity of disease in the case group, it was observed that higher total serum cholesterol levels parallel with disease severity. LDL-c (p=0.01) and HDL-c (p=0.01) levels were highly significant for disease severity. There was no relation between serum TG level and disease severity (P=0.21).

The results of this study matches with previous studies, where total cholesterol, LDL-c and triglyceride levels were reported to be higher in psoriasis patients while HDL-c levels were reported to be low.[7] There may be several mechanisms for the increased lipid levels in psoriasis. Psoriasis is a chronic inflammatory state characterized by an increase in the immunological activity of T helper cells and chronic inflammation has been suggested as a part of the metabolic diseases. Both psoriasis and the metabolic diseases are characterized by increased immunological activity of T helper cells.[8] Chronic systemic inflammation induces endothelial dysfunction, altered glucose metabolism, and insulin resistance that plays a significant role in the development of obesity, diabetes mellitus, dyslipidemia, and cardiovascular diseases such as atherosclerosis and myocardial infarction or stroke.[9]Cytokines such as TNF-α and IL-6 seem to play a central role. TNF-α plays a critical role in the activation of innate and acquired immune responses leading to chronic inflammation, tissue damage and keratinocyte proliferation. TNF-α levels are markedly increased in skin lesions, synovium and serum of patients with psoriasis and these correlate with the severity of the disease. Decreased levels are associated with clinical resolution.[10]

Furthermore, interleukin-6, IL-8, IL-1, and IL-17 are also implicated in generation of pro-atheromatous abnormalities like dyslipidaemia, insulin resistance, endothelial dysfunction, clotting system activation, and pro-oxidative stress. TNF-α may affect endothelial dysfunction by decreasing the levels of nitric oxide synthetase and cyclooxygenase-1.[11]

Antipsoriatic drugs such as oral retinoids and cyclosporine can also be responsible for lipid profile disturbances in psoriatic patients because of their action on the circulating lipids, including hypercholesterolemia, hypertriglyceridaemia and low HDL-cholesterol level.[12]Recently it has been shown that infliximab which is used to treat patients with psoriatic arthritis, can also increase triglyceride levels in psoriatic patients.[13]Both psoriasis and dyslipidemia are risk factors for cardiovascular diseases and it is important to predict the risk of cardiovascular disease in patients with psoriasis. Psoriasis has also been shown to be an independent risk factor for cardiovascular mortality.[14]In addition, there appears to be a significant association between psoriasis and traditional risk factors for atherosclerosis and heart disease in the general population, such as type-2 diabetes mellitus ,coronary artery disease, peripheral vascular disease and hypertensive heart disease.[14,15]The present study has some potential limitations due to small sample size because of our strict exclusion criteria. Future studies with larger sample size having both sexes along with quantification of body fat content are needed to understand the role of lipids in pathogenesis of psoriasis.

## Conclusion:

In conclusion we found that total cholesterol, LDL-cholesterol and triglyceride levels are as previously reported to be higher in psoriasis patients while HDL levels are as previously reported to be low which makes a difference in the severity of psoriasis. Patients of psoriasis must be considered as a group at high risk for cardiovascular diseases. Lipid derangement correlates with the severity of disease and also acts as a prognostic sign. We suggest early screening with serum lipid profile assay in psoriatic patients at the time of presentation and follow-up for evaluating risk and treatment of hyperlipidemia to modify and prevent the risk of cardiovascular diseases.

## REFERENCES


1. Icen M. Crowson CS, McEvoy MT, Gabeiel SE, Kremers HM.et al. Trends in incidence of adult-onset psoriasis over three decades: a population based study. J of American Academy of Dermatology. 2009, 60 (3); 394-401.
2. Nijsten T, Wakkee M (2009) Complexity of the association between psoriasis and comorbidities. J Invest Dermatol 129: 1601-1603.
3. Fredriksson T and pettersson U (1978): Severe psoriasis-oral therapy with a new retinoid .dermatologica; 157(4):238-44.
4. M K Yadav, T K Mohapatra, R K Mohapatra et. al. Study on Glycated Hemoglobin & lipid profile in Type-2 Diabetes Mellitus. International Journal of Science & Research (IJSR) 2015;4(6): 1917-1919.
5. Pietrzak A, Michalak-Stoma A, Chodorowska G, Szepietowski JC (2010) Lipid disturbances in psoriasis: an update. Mediators Inflamm 2010.
6. Gyulai R, Kemény L (2006) [The immunology of psoriasis: from basic research to the bedside]. Orv Hetil 147: 2213-2220.
7. Tekin NS, Tekin IO, Barut F, Sipahi EY (2007) Accumulation of oxidized low-density lipoprotein in psoriatic skin and changes of plasma lipid levels in psoriatic patients. Mediators Inflamm.
8. Langley RG, Krueger GG, Griffiths CE (2005) Psoriasis: epidemiology, clinical features, and quality of life. Ann Rheum Dis 64 Suppl 2: ii18-23.
9. Boehncke WH, Boehncke S, Tobin AM, Kirby B (2011) The 'psoriatic march':a concept of how severe psoriasis may drive cardiovascular comorbidity. Exp Dermatol 20: 303-307.
10. Joshi R (2004) Immunopathogenesis of psoriasis. Indian J Dermatol Venereol Leprol 70: 10-12.
11. Karadag AS, Yavuz B, Ertugrul DT, Akin KO, Yalcin AA, et al. (2010) Is psoriasis a pre-atherosclerotic disease? Increased insulin resistance and impaired endothelial function in patients with psoriasis. Int J Dermatol 49: 642-646.
12. Wakkee M (2010) Psoriasis: comorbidity and treatment. Rotterdam, The Netherlands: Erasmus University Rotterdam.
13. Castro KR, Aikawa NE, Saad CG, Moraes JC, Medeiros AC, et al. (2011) Infliximab induces increase in triglyceride levels in psoriatic arthritis patients. Clin Dev Immunol 2011: 352686.
14. Gelfand JM, Mehta NN, Langan SM (2011) Psoriasis and cardiovascular risk: strength in numbers, part II. J Invest Dermatol 131: 1007-1010.
15. Gkalpakiotis S, Arenberger P, Gkalpakioti P, Meluzinova E, Chandran D, et al. (2014) Management of psoriasis vulgaris and multiple sclerosis with fumaric acid. J Am Acad Dermatol 70: e60-61.