Research Article

Study of lipid profile in patients of psoriasis with relation to lifestyle factors

Prakash K Kute[1], Manohar G Muddeshwar[2], Amol R Sonare[3]

[1]Department of Biochemistry, Jawaharlal Nehru Medical College, Wardha, Maharashtra, India, [2]Department of Biochemistry, Government Medical College, Nagpur, Maharashtra, India, [3]Department of Biochemistry, Vimtas Laboratories, Hyderabad, Telangana, India

Correspondence to: Prakash K Kute, E-mail: prakashkute@gmail.com

Received: July 15, 2019; **Accepted:** August 09, 2019

ABSTRACT

Background: Psoriasis is a chronic inflammatory disease. The chronic inflammation was also observed to be associated with increased risk of cardiovascular episodes as a result of dyslipidemia. Thus, psoriasis and dyslipidemia may share a common causative mechanism. Lifestyle factors such as smoking and alcoholism may further deteriorate the condition. **Objective:** The objective of this study was to study the lipid profile pattern in psoriasis with relation to lifestyle factors. **Materials and Methods:** This was a cross-sectional study with comparison groups. Fifty diagnosed cases of psoriasis were chosen, out of that 16 were smoker and 12 were alcoholic, and 50 age- and sex-matched healthy normal subjects as controls were also selected for the study. In this study, we measured total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and very LDL cholesterol (VLDL-C) with relation to lifestyle factors, i.e. smoking and alcoholism. **Results:** The mean levels of serum total cholesterol, triglycerides, LDL-C, and VLDL-C were 213.5 ± 39.56 mg%, 223.94 ± 34.74 mg%, 136.42 ± 29.1 mg%, and 44.8 ± 6.95 mg% in cases, respectively, and 157.96+ 24.71 mg%, 142.98 ± 21.87 mg%, 80.6 ± 16.79 mg%, and 28.6 ± 4.30 mg% in controls, respectively ($P < 0.0001$); HDL-C levels were 42.64 ± 6.92 mg% in case and 48.8 ± 8.46 mg% in controls ($P = 0.0001$). Moreover, levels of serum total cholesterol, triglycerides, LDL-C, and VLDL-C were significantly increased and HDL-C level was significantly decreased in cases with smoking and alcohol habits compared to the cases without smoking and alcoholism. This change indicates dyslipidemia in psoriasis. **Conclusions:** Lipid profile may be monitored in the psoriasis cases to minimize the severity of disease and modification in lifestyle factors may be suggested to improve clinical condition.

KEY WORDS: Psoriasis; Lipid Profile; Smoking; Alcoholism

INTRODUCTION

Psoriasis is a chronic inflammatory disease with multisystem involvement. It may be due to abnormalities in eicosanoid metabolism, essential fatty acid metabolism, lipid peroxidation, free radical generation, and lymphokine secretion. It has also been associated as risk factor for cardiovascular events.[1] Psoriasis is a skin disease with the prevalence approximately more than 2% of global population. The exact etiology of psoriasis is still not known; however, certain genetic, metabolic, and immunologic mechanisms have been proposed.[2] The loss of scale from the surface observed in the psoriasis, it may be related to lipid disorders in epidermis and in serum.[3]

During the recent past, link between psoriasis and cardiovascular disease has been reported by several studies; however, the exact pathogenesis of increased cardiovascular events in patients with psoriasis is still not well established.[4]

Access this article online	
Website: http://www.ijmsph.com	Quick Response code
DOI: 10.5455/ijmsph.2019.08223082019	

Certain studies have postulated psoriasis as an autoimmune disease similar to atherosclerosis. The inflammatory changes observed to be involved in both of the diseases, inflammation triggers several changes known as acute-phase response (APR). The characteristic aberrations of lipid metabolism found during APR are increased serum triglycerides concentration and decreased high-density lipoprotein cholesterol (HDL-C). During APR, changes in total cholesterol and low-density lipoprotein cholesterol (LDL-C) also occur that may promote atherogenesis. Cytokines, such as tumor necrosis factor (TNF) or interleukin (IL)-1, are recognized as prime mediators of these metabolic changes during infection and inflammation when this inflammatory response is not able to repair injury, it turns into a harmful reaction, and the lipid changes become chronic by overwhelming stimulus and it leads to enhance the process of atherosclerosis.[5]

Certain other researchers demonstrated the aberration in plasma lipid and lipoprotein composition including an increase in total cholesterol, triglycerides, LDL-C, and decrease in HDL-C and postulated that psoriasis might be associated with the disorders of lipid metabolism.[1,2]

Significant abnormalities with fatty acid composition in epidermal cells, adipose tissue, and red blood cells in psoriasis patients have also been shown, and it was suggested that such perturbed lipid metabolism may be a generalized phenomenon in psoriasis.[6] Rocha-Pereira *et al*.[7] placed on record that the patients of psoriasis may present with an dyslipidemia and disruption in oxidants/antioxidant status, leading to atherogenesis.[7] Cigarette smoking was identified as a crucial factor in the initiation of psoriasis while alcohol consumption was attributed to increase severity of pre-existing disease.[8]

The present study was undertaken to corroborate the correlation of serum lipids in psoriatic patients and its significance with lifestyle factors such as smoking and alcoholism.

MATERIALS AND METHODS

Selection of Study Subjects

This was a cross-sectional study with comparison groups. The study protocol was approved by the Ethical Committee of the Institute. Informed written consent was obtained from all the study subjects enrolled in the study. Fifty diagnosed psoriasis patients were chosen; of which 16 were smokers and 12 were alcoholic. Fifty age- and sex-matched healthy subjects were selected as controls. Sample size was calculated using the formula.[9]

Average age of cases was 42.9 ± 7.3 and average age of control was 42.4 ± 9.4. The diagnosis of patients was done by clinical features such as itching, erythema, thickening, and scaling of the skin and Auspitz sign. The cases treated with topical agents such as dithranol and Vitamin D analogs during the 6-month period were included in the study. All the study subjects were examined and investigated according to predesigned pro forma. Information regarding sociodemographic factors, alcohol consumption, smoking habits, and personal medical history was obtained from psoriatic patients and controls using questionnaire. Patients with ischemic heart disease, renal disorders, obstructive liver disease, hypothyroidism, diabetes mellitus, any chronic inflammatory disease and any other skin disorder and cases taking systemic drug therapy of retinoids, cyclosporine, beta-blockers, and thiazides in the recent 6 months were excluded from the study.

Sample Collection and Processing

For serum lipid profile estimation, overnight fasting venous blood samples (2 cc) were collected. Serum total cholesterol, triglyceride, and HDL-C were measured using enzymatic kit. Very LDL (VLDL-C) and LDL-C were calculated according to the Friedewald formula.[10]

Statistical Analysis

Observed serum total cholesterol, triglyceride, and HDL-C, LDL-C, and VLDL-C levels were compared between cases and control by performing unpaired *t*-test. Further, serum total cholesterol, triglyceride, and HDL-C, LDL-C, and VLDL-C values were compared between cases with smoking habits and case without smoking habit, also between cases with alcohol habit and cases without alcohol habit by performing unpaired *t*-test. $P < 0.05$ was considered as statistically significant, statistical software STATA version 10.0 was used for statistical analysis.

RESULTS

Table 1 depicts that serum total cholesterol, triglyceride, HDL-C, LDL-C, and VLDL-C levels were significantly altered in patients of psoriasis. Table 2 indicates that total

Table 1: Comparison of serum lipid profile status in cases and controls

Parameter (mg %)	Cases (*n*=50)	Controls (*n*=50)	*P*-value
Total cholesterol	213.5±39.56	157.96±24.71	<0.0001
Triglycerides	223.94±34.74	142.98±21.87	<0.0001
Very low density lipoprotein	44.8±6.95	28.6±4.30	<0.0001
High density lipoprotein	42.64±6.92	48.8±8.46	0.0001
Low-density lipoprotein	136.42±29.1	80.6±16.79	<0.0001

Table 2: Comparison of serum lipid profile status in psoriasis cases with smoking habits (Group I) and psoriasis cases without smoking habits (Group II)

Parameter (unit) (mg %)	Group I (*n*=16)	Group II (*n*=34)	*P*-value
Total cholesterol	240.37±28.73	200.82±37.85	0.0006
Triglycerides	256.56±25.25	208.58±27.26	<0.0001
Very low-density lipoprotein	51.37±4.89	41.70±5.48	<0.0001
High-density lipoprotein	40.25±4.89	44.94±7.10	0.0212
Low-density lipoprotein	164.68±24.44	123.11±22.42	<0.0001

Table 3: Comparison of serum lipid profile status in psoriasis cases with alcohol habits (Group I) and psoriasis cases without alcohol habits (Group II)

Parameter (unit) (mg %)	Group I (*n*=12)	Group II (*n*=38)	*P*-value
Total cholesterol	253.6±30.30	200.81±33.36	<0.0001
Triglycerides	249.16±12.56	215.97±35.77	0.0029
Very low-density lipoprotein	49.91±2.39	43.18±7.15	0.0025
High-density lipoprotein	37.0±4.63	44.42±6.59	0.0007
Low-density lipoprotein	161.91±13.13	128.36±28.23	0.0002

cholesterol, triglyceride, LDL-C, and VLDL-C levels were significantly elevated in psoriasis cases with smoking habits compared to psoriasis cases without smoking habits. Table 3 shows the levels of total cholesterol, triglyceride, LDL-C, and VLDL-C were raised significantly in psoriasis cases with alcohol habits as compared to psoriasis cases without alcohol habits. Our results also undoubtedly depicted the significant lowering of HDL-C in cases with both the lifestyle factors studied in psoriasis.

DISCUSSION

It has been shown earlier that dyslipidemia may play a role in pathogenesis of psoriasis coinciding with diabetes mellitus and cardiovascular diseases such as heart failure, ischemic heart disease, and hypertension.[11,12] Certain genetic studies corroborate the pathogenesis of psoriasis and cardiovascular disease shares common features like chronic inflammatory changes. Multiple cardiovascular risk factors shown to be influencing in psoriasis too such as hypertension, oxidative stress, dyslipidemia, endothelial cell dysfunction, and blood platelet adhesion.[13,14]

In this study, we could notice that in psoriatic cases significantly higher serum total cholesterol, triglycerides, LDL-C, and VLDL-C levels than controls. However, HDL-C was found to be significantly lowered. We also found the elevation in the serum levels of total cholesterol, triglyceride, VLDL-C, and LDL-C in cases with smoking habits as compared to cases of psoriasis without smoking habit. However, serum HDL-C was found to be significantly decreased in psoriasis with smoking habits. During the present study, attempt was also made to find out whether alcohol consumption has any added effect on lipid profile aberration in patients of psoriasis. Serum levels of total cholesterol, triglyceride, VLDL, and LDL-C in psoriasis cases with alcohol habit found to be significantly elevated compared to the non-alcoholic psoriasis cases. HDL-C level in serum was significantly lowered in the cases with alcohol habits compared to the non-alcoholic cases.

Earlier certain workers[3,7] had reported in psoriasis a rise in total cholesterol, triglycerides, LDL-C, and a reduction in HDL-C. Mallbris *et al*.[15] depicted the higher total cholesterol, VLDL-C, and HDL-C levels compared to normal controls. While Piskin *et al*.[11] working with psoriasis showed that serum total cholesterol and LDL-C levels were significantly higher when compared with controls. Increased triglycerides level secondary to VLDL-C elevation is associated with both procoagulant and prothrombotic factors in the blood. VLDL-C mediated platelet adhesion and may play role in atherosclerosis. Furthermore, VLDL-C remnants are susceptible to deposition within the arterial intima, thereby promoting atherosclerotic plaques growth,[3] and abnormal lipoprotein metabolism may be related to the increased incidence of atherosclerosis in psoriasis.[16] The HDL particles play the most important role in reverse cholesterol transport and contribute a preventive role atherosclerosis.[3] It is reported that macrophages engulf LDL and release large quantities of TNF-α and IL-1β.[17] Cytokine-mediated inflammation and tissue damage are a common concept for chronic inflammatory diseases such as psoriasis and atherosclerosis.[18]

Kavli *et al*.[19] found that male smokers had an increased risk of psoriasis. Naldi *et al*.[20] also showed that the risk for plaque psoriasis was higher with current smokers compare to exsmokers. Setty *et al*.[21] while examining incident of psoriasis found a significantly positive corelationship between the numbers of cigarettes smoked per day and development of psoriasis, especially in women. It has also been documented in the past that smoking worsens psoriasis and patients with psoriasis who smoke tend to be less responsive to treatment.[22] Similarly, Raychaudhuri and Gross[23] found that median percentage of skin involvement was higher in smokers than in non-smokers. Smoking increases catecholamine release, causing a surge in circulating free fatty acids, which may be responsible for dyslipidemia. Smoking also reduces lecithin-cholesterol acyl-transferase levels, the enzyme responsible for

esterifying free cholesterol and increasing HDL size, and may reduce levels of cholesterol ester transfer protein.[24]

During the recent past, several large epidemiological studies have revealed the possible association between alcohol consumption and severity of psoriasis. The present observations also point a correlation between alcohol consumption and psoriasis. It may be contended that high consumption of alcohol may initiate, exacerbate, and influence the severity and the course of the disease.[8] Poikolainen *et al*.[25] and Zimmerman[26] documented that symptoms of psoriasis are aggravated in alcoholics patients. Smith and Fenske[27] could notice that alcohol stimulates the release of histamine causing increase severity of skin lesions. Moreover, alcohol consumption and excessive intake of high-fat foods have also been attributed to lipid profile disturbances.

There are certain limitations of this study that it did not look into the gender differences in lipid profile parameters also did not compare the lipid profile parameters in subgroup of psoriasis on the basis of severity.

CONCLUSIONS

The results obtained in present study and several other studies conducted in Psoriasis patients showed associated dyslipidemia that suggests correlation of cardiovascular complications and psoriasis. This finding clearly anticipates that serum lipid levels may be a considerable marker in evaluating the disease and indicative of the coronary artery disease as a risk factor in psoriasis. The gross dyslipidemia recorded both with alcohol and smoking habits in psoriasis patients, indicates that during psoriasis treatment, smoking and alcohol habits may be taken into account to improve the clinical condition of patients.

REFERENCES

1. Vanizor Kural B, Orem A, Cimşit G, Yandi YE, Calapoglu M. Evaluation of the atherogenic tendency of lipids and lipoprotein content and their relationships with oxidant-antioxidant system in patients with psoriasis. Clin Chim Acta 2003;328:71-82.
2. Tekin NS, Tekin IO, Barut F, Sipahi EY. Accumulation of oxidized low-density lipoprotein in psoriatic skin and changes of plasma lipid levels in psoriatic patients. Mediators Inflamm 2007;2007:78454.
3. Javidi Z, Meibodi NT, Nahidi Y. Serum lipids abnormalities and psoriasis. Indian J Dermatol 2007;52:89-92.
4. Kremers HM, McEvoy MT, Dann FJ, Gabriel SE. Heart disease in psoriasis. J Am Acad Dermatol 2007;57:347-54.
5. Esteve E, Ricart W, Fernández-Real JM. Dyslipidemia and inflammation: An evolutionary conserved mechanism. Clin Nutr 2005;24:16-31.
6. Ferretti G, Simonetti O, Offidani AM, Messini L, Cinti B, Marshiseppe I, *et al.* Changes of plasma lipids and erythrocyte membrane fluidity in psoriatic children. Pediatr Res 1993;33:506-9.
7. Rocha-Pereira P, Santos-Silva A, Rebelo I, Figueiredo A, Quintanilha A, Teixeira F, *et al.* Dislipidemia and oxidative stress in mild and in severe psoriasis as a risk for cardiovascular disease. Clin Chim Acta 2001;303:33-9.
8. Luty-Frąckiewicz A, Markiewicz-Górka I, Januszewska L. Influence of smoking and alcohol consumption on total antioxidant status in patients with psoriasis. Adv Clin Exp Med 2006;15:463-9.
9. Rifai N, Warnick GR. Lipids, lipoproteins, apolipoproteins, and other cardiovascular risk factors. In: Burtis CA, Ashwood ER, Bruns DE, editors. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 4th ed. New Delhi: Saunders Elsevier; 2006. p. 903-81.
10. Charan J, Biswas T. How to calculate sample size for different study designs in medical research? Indian J Psychol Med 2013;35:121-6.
11. Piskin S, Gurkok F, Ekuklu G, Senol M. Serum lipid levels in psoriasis. Yonsei Med J 2003;44:24-6.
12. Henseler T, Christophers E. Disease concomitance in psoriasis. J Am Acad Dermatol 1995;32:982-6.
13. Wakkee M, Thio HB, Prens EP, Sijbrands EJ, Neumann HA. Unfavorable cardiovascular risk profiles in untreated and treated psoriasis patients. Atherosclerosis 2007;190:1-9.
14. Torkhovskaia TI, Fortinskaia ES, Ivanova LI, Nikitina NA, Zakharova TS, Kochetova MM, *et al.* Characteristics of the lipid transport system in psoriasis. Vopr Med Khim 2002;48:297-303.
15. Mallbris L, Akre O, Granath F, Yin L, Lindelöf B, Ekbom A, *et al.* Increased risk for cardiovascular mortality in psoriasis inpatients but not in outpatients. Eur J Epidemiol 2004;19:225-30.
16. Seishima M, Seishima M, Mori S, Noma A. Serum lipid and apolipoprotein levels in patients with psoriasis. Br J Dermatol 1994;130:738-42.
17. Takeda H, Okubo Y, Koga M, Aizawa K. Lipid analysis of peripheral blood monocytes in psoriatic patients using fourier-transform infrared microspectroscopy. J Dermatol 2001;28:303-11.
18. Andreakos E, Foxwell B, Feldmann M. Is targeting toll-like receptors and their signaling pathway a useful therapeutic approach to modulating cytokine-driven inflammation? Immunol Rev 2004;202:250-65.
19. Kavli G, Førde OH, Arnesen E, Stenvold SE. Psoriasis: Familial predisposition and environmental factors. Br Med J (Clin Res Ed) 1985;291:999-1000.
20. Naldi L, Chatenoud L, Linder D, Belloni Fortina A, Peserico A, Virgili AR, *et al.* Cigarette smoking, body mass index, and stressful life events as risk factors for psoriasis: Results from an Italian case-control study. J Invest Dermatol 2005;125:61-7.
21. Setty AR, Curhan G, Choi HK. Smoking and the risk of psoriasis in women: Nurses' health study II. Am J Med 2007;120:953-9.
22. Armstrong AW, Armstrong EJ, Fuller EN, Sockolov ME, Voyles SV. Smoking and pathogenesis of psoriasis: A review of oxidative, inflammatory and genetic mechanisms. Br J Dermatol 2011;165:1162-8.
23. Raychaudhuri SP, Gross J. Psoriasis risk factors: Role of lifestyle practices. Cutis 2000;66:348-52.
24. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH, *et al.* Effects of smoking and smoking cessation on lipids and lipoproteins: Outcomes from a randomized clinical trial. Am Heart J 2011;161:145-51.

25. Poikolainen K, Reunala T, Karvonen J. Smoking, alcohol and life events related to psoriasis among women. Br J Dermatol 1994;130:473-7.
26. Zimmerman GM. Alcohol and psoriasis. Arch Dermatol 1999;135:1541-2.
27. Smith KE, Fenske NA. Cutaneous manifestations of alcohol abuse. J Am Acad Dermatol 2000;43:1-6.

How to cite this article: Kute PK, Muddeshwar MG, Sonare AR. Study of lipid profile in patients of psoriasis with relation to lifestyle factors. Int J Med Sci Public Health 2019;8(10):872-876.

Source of Support: Nil, **Conflict of Interest:** None declared.