LIPID PROFILE IN PATIENTS OF PSORIASIS

Madhur Gupta+, Suresh Chari++, Milind Borkar+++, Manju Chandankhede++++

Abstract

Objectives : Psoriasis is known to be associated with metabolic syndrome and studies have found discordant results of lipid profile in psoriasis. Hence, this study aims to study lipid profile in patients with psoriasis.

Methods : A case control study was carried out at NKP Salve Institute of Medical Sciences, Nagpur. Lipid profile was estimated in 25 patients of psoriasis. They were age and sex matched with 25 normal healthy controls.

Results : An increase in the levels of cholesterol, triglycerides, LDL and VLDL ($p<0.001$) with a decrease in the HDL cholesterol ($p<0.001$) was found in patients of psoriasis when compared with control.

Conclusions : Altered lipid profile may be responsible for the higher prevalence of cardiovascular disease in psoriasis patients.

Key Words : Psoriasis, lipid profile, metabolic synprome.

INTRODUCTION

Psoriasis is an inflammatory dermatosis characterized by hyperproliferation of the keratinocytes and inflammatory infiltration in the epidermis and dermis. There is evidence that the disease is associated with a high impact on the health-related quality of life and considerable cost. Psoriasis produces significant adverse effects on the psychological and social aspects of life mainly because of its visibility (1,2). There is a growing number of population-based studies providing worldwide prevalence estimates of psoriasis. In India, the incidence of psoriasis among total skin patients ranges between 0.44 and 2.2% (3,4,5).

The underlying pathogenetic mechanisms remain unclear. Multiple factors, including systemic inflammation, oxidative stress, aberrant lipid profile, and concomitant established risk factors, are known to be responsible for the development of psoriasis. (6) Psoriasis is known to be associated with metabolic syndrome and various studies have found discordant results of lipid profile in patients of psoriasis (7,8 9,10).

Hence it was thought worthwhile to assess the lipid profile in patients of psoriasis.

+ Prof. & Head, ++ Director Research & MET,
++++ Lecturer, Dept. of Biochemistry, NKPSIMS, Nagpur.
+++ Prof. & Head, Dept. of Skin, NKPSIMS, Nagpur.
E-mail : drmadhur20@rediffmail.com

METHODS AND MATERIALS

A case control study, approved by the ethics committee was carried out at NKP Salve Institute of Medical Sciences, Nagpur. Twenty five patients of psoriasis with a mean age of 40.31 ±12.32 were included in the study. Twenty five age and sex matched normal healthy controls with a mean age of 42 ± 13.64 were selected as controls. The clinical severity was determined according to the Psoriasis Area and Severity Index (PASI) score. Disease duration of the patients ranged from 1 month to 25 years. All the patients being treated only with topical agents such as corticosteroids, vitamin D analogues, dithanol during the six month period have been included in the study. Patients with any chronic inflammatory disease, diabetes mellitus, renal disorders, IHD, hypothyroidism, nephritic syndrome, obstructive liver disease, severe psoriasis, high BMI (>30kg/m2), hypertension, smoking, alcohol consumption and personal or family history of hyperlipidemia and any other skin disorder were excluded from the study. All the patients of systemic drug therapy of beta blockers, thiazides, retinoids, cyclosporine

TABLE I

Lipid profile in patients with psoriasis

	Group I (n=50)(controls)	Group II (n=50)(patients)
Total cholesterol (mg%)	132.02 ± 18.9	207.09 ± 15.4*
Triglyceride (mg%)	131.4 ± 16.35	223.12 ± 16.55*
VLDL (mg%)	26.71 ± 3.23	44.51 ± 3.51*
LDL (mg%)	66.89 ± 11.98	134.27 ± 9.81*
HDL(mg%)	48.43 ± 4.32	39.32 ± 4.68*

*P<0.001 when patients of group II compared with group I

and lipid lowering agents in the recent 6 months were excluded from the study. 5 ml of fasting venous blood samples was collected in plain bulb from patients with psoriasis and normal healthy individuals. Estimation of lipid profile i.e. Serum total cholesterol, triglyceride and HDL-chlolesterol were measured by an enzymatic kit. LDL-C was calculated according to the following formulae: VLDL-C=Triglyceride/5 and LDL-C= Total cholesterol-(VLDL-C + HDL-C).

RESULTS AND DISCUSSION

Psoriasis is a common chronic inflammatory dermatologic disorder, but its etiology is poorly understood. Our study reveals an increase in the cholesterol, triglyceride, LDL and VLDL levels while HDL-cholesterol is decreased (Table I) which is in accordance with the study of Akhyani et al (7). However, conflicting reports are available regarding the serum lipid values in psoriasis (11,12,13,14).

The reasons for the various changes in the lipid metabolism in patients of psoriasis are not well established. Pietrzak A et al (12) suggested that structural and functional abnormalities occur in nearly all the segments of the gastrointestinal tract resulting in deranged decomposition, modification and synthesis of many organic compounds including lipids. It has also been suggested that lipid abnormalities affect the immune system and inflammation in the patients of psoriasis is due to T cell cytokines, which is characteristic for the T cell helper I cell response (15).

Whether the changes in the lipid compostion are primary or secondary in nature is still controversial. Psoriasis is associated with lipid abnormalities at the onset of skin disease and the patients appear to have increased cardiovascular morbidity due to the associated abnormal lipid metabolism (8). Autoantibodies recognizing oxidized LDL are found and are reported to correlate with the disease severity (10). Also medications such as oral retinoids and cyclosporine may induce dyslipidemia in the patients. Hence lipid changes in psoriatic patients need to be assessed for the prevention of development of any cardiovascular accident. Moreover, lipid profile assessment may be useful for screening and treatment in psoriasis to prevent atherosclerosis.

REFERENCES

1. de Arruda LH, De Moraes AP. The impact of psoriasis on quality of life. Br J Dermatol 2001;144:33-6.
2. Gelfand JM, Feldman SR, Stern RS, Thomas J, Rolstad T, Margolis DJ. Determinants of quality of life in patients with psoriasis: a study from the US population. J Am Acad Dermatol 2004;51:704-8.
3. Okhandiar RP, Banerjee BN. Psoriasis in the tropics: An epidemiological survey. J Indian Med Assoc 1963;41:550-6.
4. Bedi TR. Psoriasis in north India. Geographical variations. Dermatologica 1977;155:310-4
5. Bedi TR. Clinical profile of psoriasis in North India. Indian J Dermatol Venereol Leprol 1995;61:202-5.
6. Dreiher J, Weitzman D, Davidovici B, Shapiro J, Cohen AD. Psoriasis and dyslipidemia: A population based study. Acta Derm Venerol 2008; 88: 561-565
7. Akhyani M, Ehsani AH, Robati RM, Robati AM. The lipid profile in psoriasis: a controlled study JEADV 2007; 2: 1330-1332.)
8. Rocha-Pereira P, Santos-Silva A, Rebelo I, Figueiredo A, Quintanilha A, Teixeira F. Dyslipidemia and oxidative stress in mild and in severe psoriasis as a risk for cardiovascular disease. Clin Chim Acta. 2001 Jan; 303(1-2):33-9.
9. Sommer DM Jenisch S, Suchan M, Christopher E, Weichental M. increased prevalence of the metabolic syndrome in patients with moderate to severe psoriasis. Arch Dermatol Res 2006; 298: 321-328.
10. Mallbris L, Granath F, Hamsten A, Stable M. Psoriasis is associated with lipid abnormalities at the onset of skin diseases. J Am acad Dermatol 2006 54: 614-621
11. Seishima M, Seishima M, Mori S, Noma A. Serum lipid and apolipoprotein levels in patients with psoriasis. Br J Dermatol 1994; 130: 738-42.
12. Pietrzak A, Lecewicz-Torun B. Activity of serum lipase and the diversity of serum lipid profile in psoriasis. Med Sci Monit 2002;8: CR9-CR13.
13. Niemann AL, Shin DB, Wang X, Margolis DJ, Trosxel AB, Gelfand JM. Prevalance of cardiovascular risk factors in patients with psoriasis J Am Acad Dermatol 2006:55:829-835.
14. Piskin S, Gurkok F, Ekulu G, Senol M. Serum lipid levels in psoriasis. Yonsei Med J 2003;44: 24-26.
15. Frostegard J, Ulfgren AK, Nyberg P etal. Cytokine expression in advanced human atherosclerotic plagues: dominance of pro-inflammatory (Th1) and macrophages-stimulating cytokines Atherosclerosis 1999; 145: 33-43.