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Serum lipids abnormalities and psoriasis

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Abstract

Context: Psoriasis is a chronic proliferative inflammatory skin disease with unknown etiology. The loss of scale from the surface observed in the course of psoriasis may be related to lipid disorders in epidermis and in serum. Moreover a predisposition to occlusive vascular disease and increased cardiovascular morbidity has been reported. **Aims:** In this study, the serum lipid levels of psoriatic patients were investigated to detect any relation in this respect. **Materials and Methods:** 60 psoriatic and 120 nonpsoriatic individuals were included in this case control study and were analogized for sex, age and body mass indices. Total cholesterol, triglyceride, high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) levels were measured. **Statistical Analysis Used:** Collected data were analyzed by statistical tests of Chi square, Mann Whitney and Kruskal Wallis applying SPSS software. **Results:** Total cholesterol, triglyceride and LDL-C were found to be significantly higher than in normal control group (P <0.05). No significant statistical difference was observed between HDL level of the two groups. **Conclusions:** We concluded that psoriatic patients should be evaluated for hyperlipidemia and obstructive vascular diseases. Administrating lipid-lowering medicines for patients particularly cases with severe disease may be beneficial prognosis.

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Full Text

Psoriasis is a chronic proliferative skin disorder with reported incidence of 1.5%-4.8% in different countries. [1] Its etiology is still unknown, while genetic, metabolic and immunological mechanisms have been recommended as its causes. Literature suggests that lipid metabolism maybe playing a role in pathogenesis of psoriasis. [2]

Lipid metabolism disorders may play a role in psoriasis pathogenesis. [3] Increasing coincidence of cardiovascular diseases including heart failure, myocardial infarction, cardiovascular hypertension and diabetes proved in several studies. [4] To justify high mortality and morbidity particularly in patients with severe and prolonged disease. [5]

Though it seems that structural changes in plasma lipids may cause increased risk of atherosclerosis in psoriasis, current data are contradictory in this respect. [6]

Uyanik in his study on 72 patients found, significantly higher serum lipoprotein A and Triglyceride level; and not significant low density lipoprotein (LDL) and high density lipoprotein (HDL) lipoprotein level corresponding to normal control group. [7]

In 2005, Mallbris et al., in their study on psoriasis patients proved significant serum lipid abnormalities in the early course of the disease. [8]

Since there has been no study on serum lipids profile of psoriasis patients prior to systemic treatment and in the initial period of the disease and during the course of disease and previous systemic treatments on serum lipids level as a damaging factor have not been considered in existing studies, we undertook to study serum lipids levels of psoriasis disease patients during the first two beginning years of and prior to systemic treatment sand then comparing the results with a sex, age, body mass indices (BMI) and physical activities matched control group.

Materials and Methods

In this case control study, 60 psoriatic patients admitted in the Dermatology ward of Imam Reza Hospital during September 2003 to September 2005 who were afflicted with psoriasis in the previous two years and had not yet received

any systemic treatment were selected as a case group. Inclusion criteria were untreated patients of plaques and guttate psoriasis with clinical and histopathological confirmations and those within two years of the disease. Exclusion criteria were: diabetes, obesity, family history of hyperlipidemia, renal and liver failure, hypothyroidism, taking systemic drugs especially lipids lowering agents, smoking and drinking spirits (alcoholic beverages) in order to eliminate damaging factors on serum lipids level of the patients. The disease was categorized in to three groups of mild (less than 30%), moderate (30%-50%) and severe (over 50%) of body surface being afflicted with the disease.

One hundred and twenty individuals among those referred to the Dermatology ward of Imam Reza Hospital for cosmetic treatment without dermatologic diseases or family history of psoriasis or other exclusion criteria as in cases, formed normal control group and were analogized for age, sex, BMI, blood pressure, physical activities (low, medium, high) with case study group. Demographic data of patients and control group were collected in specified forms for this purpose.

Letters of consent was received from all patients and the patients were sent to the hospital laboratory for lipids levels. Fasting serum cholesterol, triglyceride, HDL in venous blood 14h after having eaten were measured by colorimetric enzymatic method.

FBS was recommended to rebut any diabetes possibilities for all cases. Collected data were analyzed applying Chi-Square, Mann-Whitney and Kruskal Wallis tests statistical and SPSS statistical software.

Results

In this case control study on 60 psoriasis patients and 120 normal control group, age range was 15 to 78 years. The age range in 34 males (56.7%) was 40.5 ± 15.2 and in 26 females (43.3%) was 38.9 ± 16.5. T-student statistical test did not prove significant difference in psoriasis-afflicted patients according to age in both sexes (P =0.05). Age range was 39.8 ± 16 in case and 39.8±15.6 in the control group. T-student test showed no significant difference in age of both the groups (P =0.98). Both groups were analogized for sex as well as age, therefore 34 patients (56.7%) in case group and 68 individuals (56.7%) in control group were males (P =1). There was no significant difference in BMI for both groups (P =0.81).

The range of serum cholesterol level was 228.8 ± 50.9 in case and 202.8 ± 37.5 in the control group, which was statistically significant (P =0.001). The range of serum LDL-C were 145.4 ± 39.7 in case and 127.7 ± 31.6 in the control group which proves significant difference in both (P =0.003).

Non-parametric tests such as Mann-Whitney were applied since triglyceride and HDL did not have normal distribution in this study. The range of triglyceride was 183.0 ± 87.5 in case and 144.3 ± 89.9 in the control group which shows significant difference (P =0.001).

The range of HDL-C serum value was 43.8 ± 7.9 in case and 43.9 ± 6.3 in the control group, showing no significant difference (P =0.52).

One way variance analyze test (one way ANOVA) showed average cholesterol in groups with varying disease intensity were different (P =0.03), while increasing disease intensity had parallel increased cholesterol value, LDL-C also increased accordingly, but there was not significant difference which can be due to low number of samples and should be investigated with large numbers in groups.

No significant relation between disease intensity and triglyceride was proved by Kruskal Wallis test (P =0.16). Furthermore similar result was achieved for HDL (P =0.09).

Besides HDL/LDL decreased as disease intensity increased [Table 1].

Discussion

Almost half a century ago Lea et al., reported of increased density of serum lipids in psoriatic patients. [9]

Some factors which may affect blood lipids value additional to laboratory lipids measuring methods are, age, sex, weight, diets, season, race, genetic aptness, hormones levels, alcohol and narcotics. Age, sex, BMI, physical activity of patients and control groups were analogized and there was no significant difference noted. Negative family history of hyperlipidemia was sufficient to rebut genetic predisposition.. Most patients were from lower socioeconomic strata, hence they were considered to follow similar diet. None used alcohol and cigarette smokers were excluded. Consulting season was not regarded in this study; hepatic, renal, hypothyroid and diabetic patients were not included. Age range of psoriatics in different studies is reported to be between 16-86 years. [2] This scope in our study was 15-74 and an average of 40.5 ± 15.2 for males, 17-78 and on average of 38.9 ± 16.5 for females and no significant difference between the two sexes was proved in our study (P =0.98).

Rocha-preira reported increased cholesterol value, triglyceride, VLDL, LDL, apolipoprotein B, lipoprotein A and HDL decrease. [10] Yet Seishima proved normal results measuring total cholesterol and HDL values of 38 psoriatic patients, but serum apoB, apoC2 and C3 were significantly increased corresponding to control group. [6] Pierzak showed significant triglyceride increase in psoriatic females regardless of BMI. [11]

Uyanik proved significant increase of serum lipoprotein A [Lp(a)] and triglyceride of 72 psoriatic patients corresponding to normal group in his study. Total cholesterol, HDL, LDL and Apo- A1 in patients and control group were similar. [7]

In a study by Torkhovskaia on 192 psoriatic patients high percentage of them had hypo and hypercholesterolemia, which their distribution and dispersing were related with disease intensity. Contrary to normal people psoriatic patients had a wide range not only in HDL-2 cholesterol level but also in HDL-3 cholesterol; furthermore cholesterol ester transmitting protein (CETP) activity had decreased in patients. [12]

Mallbris et al., in a study on 200 psoriatic cases proved that there was higher total cholesterol, VLDL and HDL levels corresponding to normal control group but the difference was significant for only HDL. [8]

Piskin in his study on 100 psoriasis cases showed serum total and LDL cholesterol levels to be significantly higher than that of control group. [3]

Takeda noted serum cholesterol ester and monocytes cholesterol ester levels of psoriatic patients were significantly higher than normal group. [13]

Collectively in various studies serum cholesterol level of psoriatic patient is high, [10],[14] low [15] and has even been reported normal, [16] but this has been significantly higher than control group in our study (P =0.001). Serum triglyceride level has reported to be high, [10] low [15] and normal [14] in different sources. Yet serum triglyceride level of patient was significantly higher than normal group in our study (P =0.003). This contradiction was also observed for HDL and VLDL. HDL level had no significant difference corresponding normal group (P =0.52). LDL-C was proved to be normal [10] or high [3] in different assays and we received higher result (P =0.003).

Considering intensity of the disease in the case group, there was observed higher serum cholesterol level parallel with disease intensity (P =0.03). LDL-C was increased respectively but the difference was not significant which can be due to low number of patients and its validity should be assayed with adding up cases. There was observed no relation between serum TG and HDL level with disease intensity (P =0.16, P =0.09). Serum lipid changes had parallel accompaniment with psoriasis aggravation. [10],[12]

McDonald and Calabresi proposed a predisposition to occlusive vascular diseases in psoriatic patients, particularly in men in 1978. [3] Furthermore, abnormal lipoprotein metabolism may be related to the high incidence of atherosclerosis in psoriasis. [6]

Increased LP (a) level may be a factor for thrombotic cardiovascular diseases in psoriatic patients. Cases with more severe cutaneous affliction have rather higher LP (a). [17] Hypertriglyceridemia secondary to VLDL elevation is associated with both procoagulant and prothrombotic factors in the blood. VLDL- mediated platelet adhesion may play an important role in atherosclerosis. Furthermore, VLDL remnants are susceptible to retention within the arterial intima, thereby promoting atherosclerotic plaques growth. In a study by Mallbris apolipoprotein and lipid contents of the HDL fraction were different between the case and control groups. APO A-1 and HDL-C concentrations were significantly higher in the patients. The most important role of HDL particle is reverse cholesterol transport. Modified HDL particles in atherosclerotic plaques stimulats cholesterol efflux from foam cells, endothelium-dependent vasoreactivity and antioxidative activity and also generates a proatherogenic species that inhibits nitric oxide synthesis in endothelial cells. [8],[18]

It is reported that macrophages activated by engulfing low density lipoprotein (LDL) immune complexes release large quantities of tumor necrosis factor (TNF) -alpha and IL-1β.[13] Cytokine-driven inflammation and tissue destruction is a common theme of chronic inflammatory diseases such as psoriasis and atherosclerosis. [19]

It is understood that predisposition to vascular obstructive diseases is due to the intensity of psoriasis disease. The cause for changes in lipid metabolism in psoriatic patients is not clearly explained in the article, though it seems to be partially due to abnormality in gastrointestinal tract. Functional and structural abnormalities in almost all parts of gastrointestinal system are observed in psoriatic patients. [3]

Consequently serum lipid level particularly cholesterol, LDL and TG should be carefully assayed in psoriatic patients at presentation and during follow-up for evaluating risk to atherosclerosis and vascular obstructive disorders.

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References

1 Mallbris L, Larsson P, Bergquist S, Vingard E, Granath F, Stahle M. Psoriasis phenotype at disease onset: Clinical characterization of 400 adult cases. J Invest Dermatol 2005;124:499-504.
2 Pietrzak A, Lecewicz-Tourn B. Activity of serum Lipase [EC 3.1.1.3] and the diversity of serum lipid profile in Psoriasis. Med Sci Monit 2002;8:CR9-13.
3 Piskin S, Gurkok F, Ekuklu G, Senol M. Serum lipid levels in Psoriasis. Yonsei Med J 2003;44:24-6.
4 Henseler T, Christophors E. Disease concomitance in psoriasis. J Am Acad Dermatol 1995;32:982-6.
5 Mallbris L, Akre O, Granath F, Yin L, Lindelof B, Ekbom A, *et al* . Increased risk for cardiovascular mortality in psoriasis inpatients but not in outpatients. Eur J Epidemiol 2004;19:225-30.
6 Seishima M, Seishima M, Mori S, Noma A. Serum lipid and apolipoprotein levels in patients with psoriasis. Br J Dermatol 1994;130:738-42.
7 Uyanik BS, Ari Z, Onur E, Gunduz K, Tanulku S, Durkan K. Serum lipids and apolipoproteins in patients with psoriasis. Clin Chem Lab Med 2002;40:65-8.
8 Mallbris L, Granath F, Hamsten A, Stahl M. Psoriasis is associated with lipid abnormalities at the onset of skin disease. J Am Acad Dermatol 2006;54:614-21.
9 Lea WA Jr, Cornish HH, Black WD. Studies on serum lipids protein and lipoproteins in psoriasis. J Invest Dermatol

1958;30:181-5.
10 Rocha-Preira P, Santos-Silva A, Rebelo I, Figueiredo A, Quintanilha A, Teixeira F. Dyslipdemia and oxidative stress in mild and in severe psoriasis as a risk for cardiovascular disease. Clin Chim Acta 2001;303:33-90.
11 Pietrzak A, Lecewicz-Torun B, Jakimiuk A. Lipid and hormone profile in psoriatic females. Ann Univ Mariae Curie Sklodowska 2002;57:478-83.
12 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.
13 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.
14 Gurkok F, Piskin S, Ekuklu G. Serum lipid and lipoprotein levels in psoriasis. Bull Leprosy 1999;30:105-11.
15 Fortinskaia ES, Torkhovskaia TI, Sharapova GI, Loginova TK, Kliuchnikova ZI, Khalilov EM. Features of distribution of free and esterified cholesterol in the epidermis, biological membranes and plasma lipoproteins in psoriasis. Klin Lab Diagn 1996;4:38-43.
16 Utas S, Pasaoglu H, Muhtaroglu S, Unver U, Utas C, Kelestimur F. Serum lipid profile in patients with psoriasis. T Klin J Dermatol 1995;5:18-29.
17 Seekin D, Tokgozoglu L, Akkaya S. Are Lipoprotein profile and lipoprotein (a) levels altered in men with psoriasis? J Am Acad Dermatol 1994;31:445-9.
18 Zheng L, Nukuna B, Brenn ML, Sun M, Goormastic M, Settle M, *et al* . Apolipoprotein A-I is a selective target for myeloperoxidase-catalysed oxidation and functional impairment in subjects with cardiovascular disease. J Clin Invest 2004;114:529-41.
19 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.