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Study Of Lipid Profile In Psoriasis.

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ABSTRACT

Psoriasis is a common and recurrent proliferative inflammatory skin disease with an unknown etiology. It can occur due to abnormalities in essential fatty acid metabolism, lymphokine secretion, free radical generation, lipid peroxidation and eicosanoid metabolism. It has been associated with increased frequency of cardiovascular events. Several reports have demonstrated an increased risk for traditional cardiovascular risk factors, such as smoking, low physical activity, hypertension, obesity, diabetes mellitus and the metabolic syndrome (a combination of central obesity, diabetes mellitus type 2 or insulin-resistance, hypertension and combined dyslipidemia) in patients with psoriasis .Various disorders of plasma lipid and lipoprotein pattern occur in psoriasis which can predispose to occlusive vascular disease. In this study, Serum lipid levels were investigated in psoriatic patients to explore the knowledge of this association. The aim of this study was to estimate serum lipid profile in psoriasis patients and to compare the levels with healthy individuals. The study population comprised of 50 Psoriatic patients were selected as cases and 50 healthy individuals were selected as controls. Fasting lipid profile were estimated for the study group. Results were expressed as mean±SD . Student's t test was employed for the statistical analysis of data. P value less than 0.001 was taken as the significant value. Serum Cholesterol, Triglycerides, LDL - cholesterol & VLDL - cholesterol were found to be significantly higher in Psoriatic patients than in control groups. No significant difference was observed between HDL levels of the two groups. This study showed that psoriatic patients have significant alterations in lipid profile which serve as a risk factor for cardiovascular diseases. We conclude that these patients should be evaluated and followed up for the risk of hyperlipidemia and cardiovascular diseases.

Keywords: Psoriasis, Lipid profile, Hyperlipidemia.

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INTRODUCTION

Psoriasis is a common chronic and recurrent inflammatory skin disorder [1]. It is an immune-mediated skin disease characterized by hyperproliferation and reduced differentiation of keratinocytes which is initiated and maintained by inflammatory mediators [2]. Psoriasis is highly prevalent in the general population, mainly as a result of its chronic nature and the absence of a cure. Psoriasis is a chronic inflammatory dermatosis affecting more than 2% of the population [3]. The etiology is still unknown, while genetic, metabolic and immunological mechanism has been implicated.

It is characterized by an increased prevalence of obesity, hypertension, hyper lipoproteinaemia and oxidative stress, leading to occlusive vascular diseases, cardiovascular accidents, arthritis, Diabetes and liver diseases [4-6]. Psoriasis has been associated with an increased morbidity and mortality from high frequency of cardiovascular events. However, the pathogenesis of atherothrombotic events in psoriasis patients remains to be recognized.

Multiple factors including abnormal lipids and lipoprotein profiles and risk factors such as hypertension, obesity, diabetes mellitus have been associated with psoriasiss [7]. Several reports suggests that psoriatic patients have proatherogenic lipid profile including increased levels of serum triglycerides, LDL-cholesterol, VLDL-cholesterol and low HDL-cholesterol levels.

Recently the concept of "psoriatic march" has been proposed, in which chronic cutaneous inflammation in psoriasis leads to systemic inflammation, which, in conjunction with increased oxidative stress triggers a cascade of events including oxidative stress, dyslipidemia, endothelial dysfunction and insulin resistance which increases the risk of cardiovascular complications in these patients 8-10].

Alteration in lipid profile is mainly due to the cytokines. Low-density lipoprotein, on oxidation, induces monocyte infiltration and smooth muscle proliferation and that favors formation of atherosclerotic plaque [11]. High-density lipoprotein is involved in reverse cholesterol transport and inhibition of monocytic infiltration and thereby it suppresses atherogenicity [12]. Thus, atherogenic dyslipidemia has been linked to the inflammatory process in psoriasis.

In the present study, we investigated the lipid profile in a group of psoriasis patients and compared with the normal control group to look for increased risk of cardiovascular diseases.

Aims and Objectives:

The aim of this study was to estimate serum lipid profile in psoriasis patients and to compare the levels with age matched healthy individuals.

MATERIALS AND METHODS

The study group consisted of 50 Psoriatic patients as cases and 50 age and gender- matched healthy controls were selected as controls.

Individuals with history of coexisting inflammatory skin disease, smoking, alcoholics, diabetes mellitus, obesity, history of hyperlipidemia, renal and liver failure, hypothyroidism and systemic therapy, lipid lowering drugs for last 3months prior to blood collection in order to eliminate factors influencing the serum lipids levels were excluded from the study.

After obtaining a written informed consent, the case details and clinical examination was performed and recorded.

Serum was collected from study group following 12 hrs of fasting for determination of lipid profile. Serum triglycerides (TG) was measured enzymatically by modified glycerol-3-phosphate oxidase /peroxidase method (GPO-PAP) and Serum total cholesterol (TC) was measured enzymatically by modified cholesterol oxidase/peroxidase (CHOD-PAP) in autoanalyzer . HDL-Cholesterol was measured using the method referred for total cholesterol after precipitation of lipoproteins (LDL/VLDL/Chylomicrons) with sodium phosphotungstic acid & magnesium chloride mixture. LDL-Cholesterol was computed by Friedwalds formula:(LDL=TC-[HDL+TG÷5]). VLDL-Cholesterol was calculated by formula: VLDL=TG÷5.

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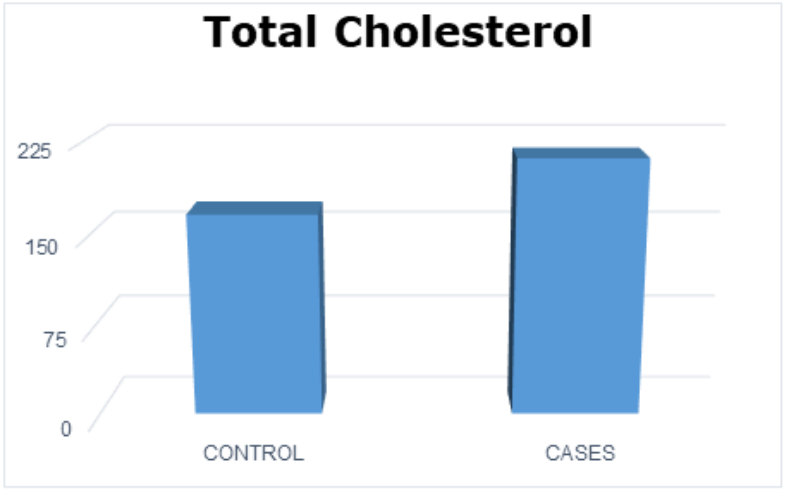
STATISTICS AND RESULTS

Results were expressed as mean ± standard deviation. A $p<0.05$ was considered as a statistically significant. Student's t test was employed for the statistical analysis of data.

Table 1: Comparisons of lipid profile between controls & cases

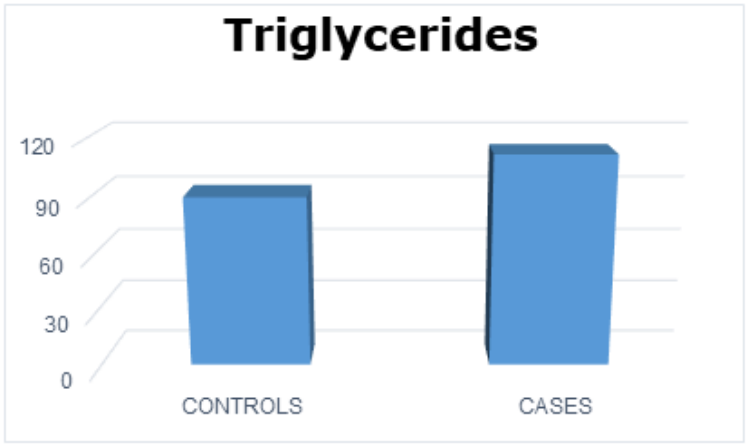
PARAMETERS	CONTROLS (N – 50)	CASES (N – 50)
Total cholesterol (mg/ dl)	167± 25	212 ±35
Triglycerides (mg/dl)	90± 21	112± 34
LDL – Cholesterol (mg/dl)	86± 20	156 ± 42
VLDL – cholesterol (mg/dl)	19.8 ± 4.0	25± 7.3
HDL – Cholesterol (mg/dl)	45 ±12	45± 9

Figure 1: Bar diagram showing comparison of Total Cholesterol in controls & cases.



The mean Total cholesterol in cases (212 ±35) was found to be significantly higher than in control groups(167± 25).

Figure 2: Bar diagram showing comparison of Triglycerides in controls & cases.



The mean Triglycerides in cases (112± 34) was found to be significantly higher than in control groups(90± 21)

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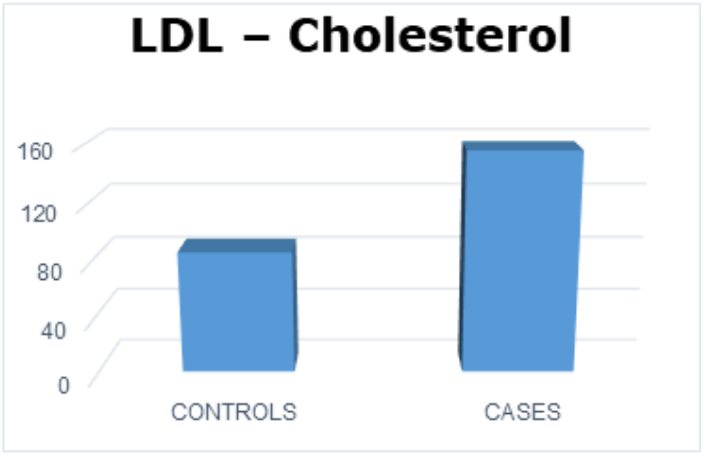
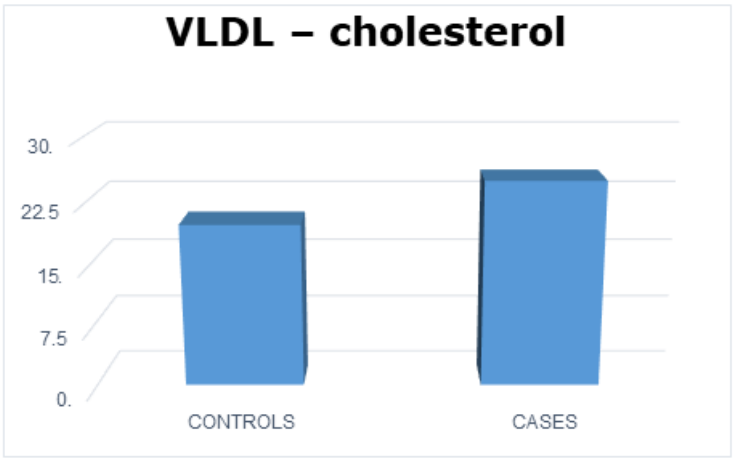
Figure 3: Bar diagram showing comparison of LDL-Cholesterol in controls & cases



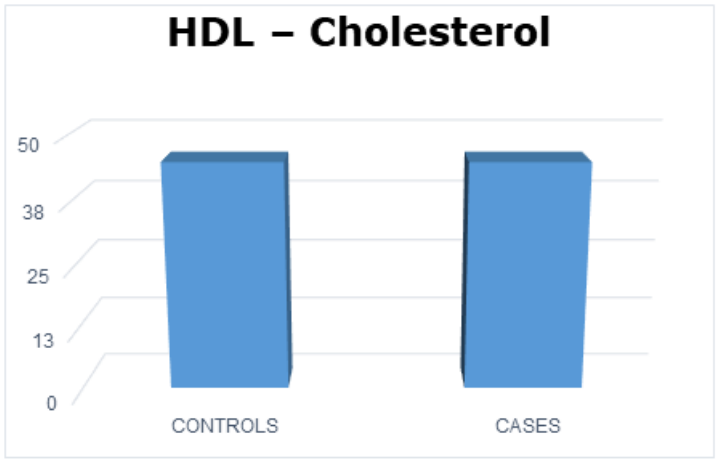
The mean LDL -cholesterol in cases (156 ± 42) was found to be significantly higher than in control groups(86± 20).

Figure 4: Bar diagram showing comparison of VLDL Cholesterol in controls & cases.



The mean VLDL -cholesterol in cases (25± 7.3) was found to be significantly higher than in control groups(19.8 ± 4.0).

Figure 5: Bar diagram showing comparison of HDL Cholesterol in controls & cases.



No significant differences in HDL levels between cases and controls.

DISCUSSION

Psoriasis is the recurrent inflammatory skin disorder, characterized by marked increase in keratinocyte proliferation as well as abnormal differentiation [13]. Lea Jr. et al, (1958) reported increased serum lipid levels in psoriasis patients [14]. Akhyani et al mentioned in their study significant increase in serum total cholesterol, triglyceride, and LDLc in psoriasis [15].

Aberrant lipoprotein composition at disease onset observed in a study indicates predisposition of psoriasis patients to develop lipid abnormalities . The lipids present in the scales of psoriasis have shown increased levels of cholesterol and low free fatty acids [16]. During exfoliation there is loss of cholesterol from the scales. This could be the reason for increased synthesis of serum cholesterol causing dyslipidemia. Functional and structural abnormalities have been seen in various segments of gastrointestinal tract [17]. Intestines play an important role in the absorption, composition, and degradation of lipoproteins. Thus there is a possibility that the structural abnormalities in the gastrointestinal tract can adversely affect the lipid levels.

Psoriasis is a chronic inflammatory state and chronic inflammation has been suggested as part of the metabolic syndrome. Both Psoriasis and the metabolic syndrome are characterized by increases in the immunological activity of type 1 helper T cells. Psoriasis is a T helper cell 1 response, leading to increased levels of TNF .TNF is also shown to cause insulin resistance which is suggested to interfere negatively with lipid metabolism. Macrophages activated by engulfing LDL immune complexes release large quantities of tumor necrosis factor (TNF-α) and IL-1β [18]. Cytokine driven inflammation and tissue destruction is a common theme of chronic inflammatory disease. That is why, in psoriasis, the association between lipid and immunologic abnormalities was observed, so the disease could be described as an immunometabolic syndrome. The significant role of cytokines, such as TNF-α, IL-6, IL-8, IFN-gamma, IL-1, and IL-17 in the generation of proatheromatous abnormalities (dyslipidemia, insulin resistance, endothelial dysfunction, clotting system activation and pro-oxidative stress) was reported. The lipid abnormalities seen in psoriasis might facilitate and maintain the inflammatory reaction in the skin.

The skin is a potential target for oxidative injury, as it is continuously exposed to UV radiation and other environmental stresses generating reactive oxygen species . Increased ROS production in patients of psoriasis [19] and decreased concentration of antioxidants leads to oxidative stress, which causes lipid peroxidation and this may lead to cell damage by continuous chain reactions. In addition, it may be responsible for activation of phospholipase A2 and production of many mediators by arachidonate, deactivation of adenylate cyclase and activation of guanylate cyclase leading to decrease in the cAMP/cGMP ratio responsible for epidermal proliferation in patients of psoriasis [20].

Some medications used to treat psoriasis, such as oral retinoids and cyclosporine, may induce dyslipidaemia in some patients [21].

In our study, we found Serum Total cholesterol, Triglycerides, LDL-cholesterol &VLDL cholesterol levels in psoriasis group were significantly higher than in control group ($p<0.001$). No significant difference in HDL levels between the two groups.

CONCLUSION

Our data suggest that psoriasis patients has been associated with abnormal lipid profile and they must be considered as a group at high risk for cardiovascular disease. We suggest early screening with serum lipid profile assay in psoriatic patients at the time of presentation and follow-up to evaluate the risk for cardiovascular diseases.

REFERENCES

[1] Davidson. Psoriasis. In : Principles and Practice of Medicine. 18 ed. 2002. p. 900.

[2] Davidovici BB, Sattar N, Prinz J, Puig L, Emery P, Barker JN, et al. Psoriasis and systemic inflammatory diseases: Potential mechanistic links between skin disease and co-morbid conditions. J Invest Dermatol 2010;130:1785-96.

[3] Raychaudari SP, Farher EM. The prevalence of psoriasis in the world. J Eur Acad Dermatol Venereol 2001;15:16-7.
[4] Aliya Nusrath, Prathibha K., Rajeshwari A. Evaluation of Cardiovascular Risk Factors in Psoriasis Patients: International Journal of Recent Trends in Science And Technology. 2014, 9(3): 328- 331.
[5] Briganti S, Picardo M. Antioxidant activity, lipid peroxidation and skin diseases. What's new? J Eur Acad Dermatol Venereol. 2003; 17:663–669.
[6] Luty-Frackiwicz A, Gorka IM, Januszewska L. Influence of smoking and alcohol consumption on total antioxidant status in patients with psoriasis. Adv Clin Exp Med. 2006; 15(3): 463–69.
[7] Mallbris L, Granath F, Hamsten A, Stahle M. Psoriasis is associated with lipid abnormalities at the onset of skin disease. J Am Acad Dermatol 2006;54:614-21.
[8] Gottlieb AB, Chao C, Dann F. Psoriasis comorbidities. J Dermatolog Treat 2008;19:5-21.
[9] Choi WJ, Park EJ, Kwon IH, Kim KH, Kim KJ. Association between psoriasis and cardiovascular risk factors in Korean patients. Ann Dermatol 2010;22:300-6.
[10] Di Lisi D, Macaione F, Corrado E, Bonura F, Novo G, Peritore A, et al. Cardiovascular risk profile of patients with psoriasis. Recenti Prog Med 2013;104:102-5.
[11] Goldstein JL, Ho YK, Basu SK, Brown MS. Binding site on macrophages that mediates uptake and degradation of acetylated low density lipoprotein, producing massive cholesterol deposition. Proc Natl Acad Sci U S A 1979;76:333-7.
[12] Steinberg D, Parthasarathy S, Carew TE, Khoo JC, Witztum JL. Beyond cholesterol. Modifications of low-density lipoprotein that increase its atherogenicity. N Engl J Med 1989;320:915-24.
[13] Rocha P, Silva AS Rebelo, Figuniredo A, Quinitanilha A, Texeira F. The Inflammatory response in mild and in severe psoriasis. British J of Dermatol. 2004; 150(5):917–28.
[14] Lea Jr., H. H. Cornish, and W.D. Block,; Studies on serum lipids, proteins, and lipoproteins in psoriasis, The Journal of Investigative Dermatology. 1958; 30(4):181–185.
[15] Akhyani M, Ehsani AH, Robati RM, Robati AM. The lipid profile in psoriasis: a controlled study: JEADV. 2007; 21:1330-1332.
[16] Wilkinson DI. "Psoriasis and dietary fat: the fatty acid composition of surface and scale (ether-soluble) lipids," The Journal of Investigative Dermatology, 1966; 47(3):185–192.
[17] Piskin S, Gurkok F, Ekuklu G, and Senol M, "Serum lipid levels in psoriasis," YonseiMedical Journal. 2003; 44(1):24–26.
[18] Takeda H, Okuho M, Hoga M, Aizawa K. Lipid analysis of peripheral blood monocytes in psoriatic patients using fourier transform infrared microspectroscopy. J Dermal 2001;28:303-11.
[19] Maccarrone M, Catani MV, Iraci S, Melino G, Agro AF. A survey of reactive oxygen species and their role in dermatology J Eur Acad Dermatol Venerol. 1997; 8:185-202.
[20] Popov I, Lewing G. A deficient function of the antioxi- dative system of the organism as an aetiopathogenetic factor in psoriasis. Med Hypothesis. 1991; 35:229-36.
[21] Neimann AL, Shin DB, Wang X, Margolis DJ, Troxel AB, Gelfand JM. Prevalence of cardiovascular risk factors in patients with psoriasis. J Am Acad Dermatol 2006; 55: 829–835.