

Correlation between malondialdehyde and dyslipidemia in psoriatic patients

Fadhil Jawad Al-Tu'ma,^a Ali Tariq Abd Al-Hassan,^b Eman Musa Abed Al-Da'amy^a

^aDepartment of Biochemistry, College of Medicine, University of Karbala, Holy Karbala, Iraq.

^bCollege of Dentistry, University of Karbala, Holy Karbala, Iraq.

Correspondence to Fadhil Jawad Al-Tu'ma (email: f_altoma_56@yahoo.com).

(Submitted: 21 January 2016 – Revised version received: 3 March 2016 – Accepted: 13 May 2016 – Published online: 26 June 2016)

Objectives This study was designed to investigate the correlation between serum lipid profile and lipid peroxidation as malondialdehyde (MDA) in psoriatic patients.

Methods This case–control study was performed on 70 psoriatic patients and 30 healthy individuals as control, matched for age and sex. The blood samples were collected after 14 h fasting. The serum lipid profile was assayed using the standard kit and MDA was assayed using the ELISA kit.

Results The certain parameters, including serum triglyceride, cholesterol, low-density lipoprotein-cholesterol (LDL-C) and very low-density lipoprotein (VLDL), were significantly higher in the case group compared to the controls ($P < 0.05$), while high-density lipoprotein-cholesterol (HDL-C) remained within normal limit in the patients group compared to the control, while there was a significantly higher levels in MDA in case group as compared to that found in the controls ($P < 0.05$).

Conclusion These results have revealed the higher plasma level of lipid profiles in psoriatic patients. This may elevate the risk of atherosclerosis, particularly cardiovascular disorders. Therefore, from the epidemiological point of view, on screening the psoriatic patients, particularly those with severe psoriasis, is recommended.

Keywords psoriasis, lipid profile, malondialdehyde

Introduction

Psoriasis is a chronic inflammatory and autoimmune disorder with unknown etiology. The prevalence rate of psoriasis ranges from 1 to 4.8%. Although it may involve in all age groups, the mean age of its incidence is 17.8 years.¹ The disease is characterised by an increased keratinocyte proliferation and alteration in the dermal and epidermal T-cells, monocytes macrophages and neutrophils.² The increased antigen presentation by dendrites cells and their presentation to T-lymphocytes lead to the following changes: T-cell activation and secretion of type1 (TH1) cytokines like interferon, interleukin-2 and tumour necrosis factor alpha (TNF- α). These cytokines induce inflammatory changes in the epidermis, yielding thick, scaly plaques.³ Recently, the role of T-lymphocytes in pathogenesis of psoriasis and atherosclerosis has been clarified. The psoriasis has been associated with an abnormal plasma lipid metabolism and diabetes, probability related to alterations in insulin secretion and sensitivity.⁴ Furthermore, there is an increased oxidative stress which is accompanied by a high frequency of cardiovascular disease.⁵ The high rate of cardiovascular events is related to the severity of the disease which occurs more frequently in patients with large areas of the body affected by psoriasis lesions.⁶ Although hyperlipidemia is one of the cardiovascular risk factors, the findings of various studies are not consistent, and some researches even disagree with the role of hyperlipidemia in psoriasis (or the role of cardiovascular disorders in psoriasis).^{7–11} This study was designed to investigate the serum lipid profile of patients with various grades of psoriasis.

Materials and Methods

There is a cross-sectional study conducted over a period from March 2015 through March 2016. The samples were collected

from the dermatology clinic in Al-Hussein Teaching Hospital in Karbala city. The practical side of the study was performed at the laboratory of clinical chemistry department and the laboratory of the immunology department in Al-Hussein Teaching Hospital. This study included 70 patients, attending a dermatology clinic in Al-Hussein Teaching Hospital (50 males and 20 females) with an age range of 7–70 years. They were diagnosed by a dermatology specialist as having psoriasis. On the other hand, 30 apparently healthy persons (15 males and 15 females) with an age range of 9–60 years were chosen as a control group. A questionnaire was designed to obtain the information from psoriasis patients and control group. It contained the name, age, weight, height, duration of disease and site of lesion.

The exclusion criteria for both the groups were: diabetes, hypertension, cardiovascular disease, smoking, history of alcohol intake, liver obstructive disease, kidney problems, connective tissue diseases, hypothyroidism, family history of hyperlipidemia and using lipid-lowering drugs, cyclosporine, corticosteroids, β -blockers, thiazide, retinoid and methotrexate. The subjects who had high-fat foods during dinner were excluded. After explaining the purpose of the study and obtaining the consent letter, the data were recorded on questionnaires for each patient. After a 14 h fasting period, 5 mL venous blood was taken in a sterile syringe in the morning from all cases and submitted to the laboratory. The serum levels of total cholesterol, triglyceride, LDL-C, HDL-C and VLDL-C were measured by an enzymatic method with the standard kits made by Biolabo, SA, France. In addition, the serum levels of malondialdehyde (MDA) were measured by ELISA method with the standard kits made by Elabscience, China. The severity of psoriasis was evaluated based on the standard criteria of psoriasis. The clinical severity of the disease was determined according

to the PASI score. By estimating the extent of the body surface in involvement, the scaling in percentage and scoring the erythematous, thickening of the affected areas (scalp, trunk, the lower limb and upper limb), the severity of the disease was determined. The collected data were analysed with student's *t*-test to assess the difference between the two groups. A logistic regression was used for correlation, and the multi-varieties regression was used to investigate the effect of serum lipid level on the severity of psoriasis. The *P* values of <0.05 were considered statistically significant.

Results

In this case-control study, 70 psoriatic patients and 30 normal individuals as the control group were enrolled for investigation. As in Table 1, the total cholesterol, triglycerides, VLDL-C, HDL-C and LDL-C were significantly altered (*P* < 0.05).

Table 2 depicts the levels of MDA (*P* < 0.05) which were significantly elevated in psoriatic patients as compared to normal healthy controls.

In the patient group, the serum total cholesterol, triglyceride, VLDL-C, and LDL-C were significantly higher than the controls while the HDL-C level was no change (*P* < 0.05). In the patient group, the serum MDA level was significantly higher than the controls (*P* < 0.05).

Discussion

The coronary atherosclerosis is common and the prevalence is increasing. The disorders are mediated by the T-helper cytokines, such as psoriasis, are associated with an increased risk of atherosclerosis and cardiovascular events.¹² It seems that the prevalence of cardiovascular events is associated with the severity of the disease and the body surface area involvement.¹³ One of the causes of cardiovascular diseases in psoriatic patients may be the elevation of plasma lipid and other inflammatory

mediators.¹⁴ There is a contraindicating reports about the association between serum triglyceride, cholesterol, LDL-C, VLDL-C and HDL-C with psoriasis; the discrepancy goes farthest, some studies indicate normal^{11,15} higher^{13,16,17} or even lower serum triglyceride levels in psoriatic patients.¹⁶

In this study, the serum triglyceride level was significantly in patients with psoriasis compared to the controls (*P* < 0.05). There have been controversial results on serum cholesterol level in psoriatic patients; different studies report higher,¹⁸ lower¹⁷ or even normal levels.^{9,19} Our results indicate significantly higher serum cholesterol levels in psoriatic patients compared to controls (*P* < 0.05). In a numerous studies, the serum LDL-C levels in psoriatic patients are reported normal⁷ or higher.⁹ In our investigation, the serum LDL-C in the case group was higher than the control group. Also in our study, the VLDL-C level was higher which contrasts the other data that indicate normal range.^{17,19} Also, the HDL-C level was nonsignificant in psoriasis patients compared with control group (*P* > 0.05) and inconsistent with other studies.^{13,17,19} The differences in the results of various studies might reflect genetic factors, lifestyle, severity of disease, daily activity and diet in each region. The causes of dyslipidemia (abnormal amount of lipids) in psoriasis may be multiple; the immune mechanisms involving IL-6 and tumour necrosis factor, C-reactive protein and cellular oxidative stress may be responsible for the altered lipid metabolism.⁵

The increased leukocyte count is linked to cell activation, as the activated neutrophils are the important sources of oxygen metabolites, which may trigger oxidative modifications in plasma constituents and in cell membranes, which probably accounted for the significant rise in plasma lipid peroxidation along with a significantly reduced antioxidants, clearly reveals the development of an oxidative stress conditions in psoriasis.²⁰⁻²³

The results of this study show significantly increased plasma MDA levels in psoriatic patients as compared to controls and the levels were positively correlated to disease severity, a result which supports the involvement of oxidative damage in the etiopathogenesis of psoriasis. These results are in agreement with those of other investigators.^{24,25} These studies suggested that the increased production of ROS/RNS in patients with psoriasis results in an increased lipid peroxidation with a subsequent formation of high levels of MDA.

However, some other investigators²⁶ did not detect any difference in serum MDA levels in psoriatic patients as compared to controls in spite of the significantly increased levels in tissues.

Also found nonsignificant values of plasma MDA in patients with psoriasis in relation to controls, in spite of high significant values in erythrocytes.²⁷

Agreeing with our results, a higher platelet, erythrocyte, tissue, serum and plasma levels of MDA and a correlation with disease severity have been reported in previous studies.²⁸

Although we did not find a correlation between levels of MDA and severity of psoriasis, some previous studies of Attwa et al. (2011)²⁹ and Jyothi et al. (2011)³⁰ had reported a positive correlation between the levels of MDA and the disease severity. They suggested that their results might support the proposal that serum MDA level could be helpful in predicting the prognosis of psoriasis and add further support for the involvement of oxidative stress in the pathogenesis of psoriasis. ■

Table 1. Lipid profile of psoriatic and control

Parameters	Group	Mean ± SD	Range	P-value
TC (mg/dl)	Patient	212.5 ± 52.46	105.0–311.0	<0.05
	Control	127.3 ± 18.87	165.0–101.0	
TG (mg/dl)	Patient	170.8 ± 62.71	403.0–80.0	<0.05
	Control	108.2 ± 11.56	140.0–87.0	
HDL-C (mg/dl)	Patient	39.59 ± 7.58	68.0–25.0	>0.05
	Control	40.70 ± 4.10	51.0–35.0	
LDL-C (mg/dl)	Patient	137.6 ± 47.71	235.6–34.20	<0.05
	Control	66.55 ± 19.52	103.2–39.0	
VLDL-C (mg/dl)	Patient	34.33 ± 12.58	16.00–80.60	<0.05
	Control	21.63 ± 2.313	28.0–17.40	

Table 2. Malondialdehyde of psoriatic and control

Parameters	Group	Mean ± SD	Range	P-value
MDA ng/ml	Patient	354.9 ± 112.2	31.74–497.0	<0.05
	Control	232.0 ± 70.35	111.0–369.0	

References

- Bijlmakers MJ, Kanneganti SK, Barker JN, Trembath RC, Capon F. Functional analysis of the RNF114 psoriasis susceptibility gene implicates innate immune responses to double-stranded RNA in disease pathogenesis. *Hum Mol Genet.* 2011;15:3129–3137. doi: 10.1093/hmg/ddr215 PMID: 21571784
- Ortonne JP. Recent developments in the understanding of the pathogenesis of psoriasis. *Br J Dermatol.* 1999;140:1–7. PMID: 10731127
- Kraeger JG, Bowcock A. Psoriasis pathophysiology: current concepts of pathogenesis. *Ann Rheum Dis.* 2005;64(suppl 2):ii30–ii36. PMID: 15708932
- Shapiro J, Chohen AD, David M, Hotak E, Chodik G, Viner A, et al. The association between psoriasis, diabetes mellitus and atherosclerosis in Israel: a case control study. *J Am Acad Dermatol.* 2007;56:629–634. PMID: 17157411
- Gupta M, Charis S, Borkar M, Chandankhede M. Dyslipidemia and oxidative stress in patients of psoriasis. *Biomed Res.* 2011;22(2):221–224.
- Gelfand JM, Neiman AL, Shin DB, Wang X, Margolis DJ, Tormel AB. Risk of myocardial infarction in patients with psoriasis. *JAMA.* 2006;296:1733–1741. PMID: 17032986
- Seckin D, Tokgozoglul L, Akkoya S. Are lipoprotein profile and lipoprotein (a) levels altered in men with psoriasis? *J Am Acad Dermatol.* 1994;31:445–449. PMID: 8077470
- Pietrzak A, Lecewicz-Torun B. Activity of serum lipase [EC300]. And the diversity of serum lipid profile in psoriasis. *J Mol Cat B Enzym.* 2006;40:144–154. PMID: 11782673
- Piskin S, Gurkok F, Ekuklu G, Senol M. Serum lipid levels in psoriasis. *Yousef Med J.* 2003;44:24–26. PMID: 12619171
- 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;54:614–621. PMID: 16546581
- Yvanik BS, Ari Z, Onur E, Gunduz K, Tanulka S, Durkan K. Serum lipids and apolipoproteins in patients with psoriasis. *Clin Chem Lab Med.* 2002;40:65–68. PMID: 11916273
- Frostegard J, Ulfgren AK, Nyberg P, Hedén U, Swedenborg J, Andersson U, et al. Cytokine expression in advance human atherosclerotic plaques: dominance of pro-inflammatory (Th1) and macrophage-stimulating cytokines. *Atherosclerosis.* 1999;145:33–43. PMID: 10428293
- Akhyani M, Ehsani AH, Robati RM, Robati AM. The lipid profile in psoriasis: a controlled study. *J Eur Acad Dermatol Venercol.* 2007;21:1330–1332.
- Javidi Z, TayyebiMeibodi N, Nahidi Y. Serum lipids abnormalities and psoriasis. *Indian J Dermatol.* 2007;52:89–92. PMID: 17958837
- Reynoso-von Drateln C, Martínez-Abundis E, Balcázar-Muñoz BR, Bustos-Saldaña R, González-Ortiz M. Lipid profile, insulin secretion, and insulin sensitivity in psoriasis. *J Am Acad Dermatol.* 2003;48:882–885. PMID: 12789179
- Yvanik BS, Ari Z, Onur E, Gündüz K, Tanülkü S, Durkan K. Serum lipids and apolipoproteins in patients with psoriasis. *Clin Chem Lab Med.* 2002;40:65–68. PMID: 11916273
- Bajaj DR, Mahesar SM, Devrajani BR, Iqbal MP. Lipid profile in patients with psoriasis presenting at Liaquat University Hospital Hyderabad. *J Pak Med Assoc.* 2009;59:512–515. PMID: 19757693
- Fortinskaia ES, Torkhovskaia TI, Sharapova Gl, Loginova TK, Kliuchnikova Zh, Khalilov EM. Features of distribution of free and esterified cholesterol in the epidermis, biological membranes and plasma lipoproteins in psoriasis. *Klin Lab Diagn.* 1996;38–43. PMID: 8963558
- Farshchian M, Zamanian A, Farshchian M, Monsef AR, Mahjub H. Serum lipid level in Iranian patients with psoriasis. *J Eur Acad Dermatol Venereol.* 2007;21:802–805. PMID: 17567311
- Gornicki A, Gutsze A. Erythrocyte membrane fluidity changes in psoriasis: an EPR study. *Dermatol Set.* 2001;27:27–30. PMID: 11457641
- Therond B, Gerbaud P, Dimon S. Antioxidant enzymes in psoriatic fibroblasts and erythrocytes. *J Invest Dermatol.* 1996;106:1325–8. PMID: 8752678
- Santos-Silva A, Rebelo I, Castro EMB. Erythrocyte damage and leukocyte activation in ischemic stroke. *Clin Chim Acta.* 2002;320:29–35. PMID: 11983197
- Weiss SJ. Neutrophil-mediated met hemoglobin formation in the erythrocyte: the role of superoxide and hydrogen peroxide. *Bio Chem.* 1982;257:2947–53. PMID: 6277918
- Baz K, Cimen MY, Kokturk A, Yazici AC, Eskandari G, Ikizoglu G, et al. Oxidant/antioxidant status in patients with psoriasis. *Yonsei Med J.* 2003;44:987–990. PMID: 14703605
- Wozniak A, Drewa G, Krzyzyska-Malinowska E, Czajkowski R, Protas-Drozd F, Mila-Kierzenkowska C, et al. Oxidant–antioxidant balance in patients with psoriasis. *Med Sci Monit.* 2007;13:CR30–CR33. PMID: 17179907
- Yildirim M, Inaloz HS, Baysal V, Delibas N. The role of oxidants and antioxidants in psoriasis. *J Eur Acad Dermatol Venereol.* 2003;17:34–36. PMID: 12602965
- Kökçam I, Naziroğlu M. Antioxidants and lipid peroxidation status in the blood of patients with psoriasis. *Clin Chim Acta.* 1999;289:23–31. PMID: 10556650
- Bariaa AJ. Thesis; Babylon University, College of Medicine. Oxidant and antioxidant status in psoriasis. 2010;26–28.
- Attwa E, Swelam E. Relationship between smoking-induced oxidative stress and the clinical severity of psoriasis. *J Eur Acad Dermatol Venereol.* 2011;25:782–787. doi: 10.1111/j.1468-3083.2010.03860.x PMID: 21039915
- Jyothi RS, Govindswamy KS, Gurupadappa K. Psoriasis: an oxidative stress condition. *J Clin Diagn Res.* 2011;5:120–121.