

Study of lipid profile for psoriatic patients in AL-Anbar governorate.

Wajeeh Y. Mohammed* Abdullah S. Al-Hasan Ausama A. Faisal****

***University Of Anbar - College of science**

**** University Of Anbar - College of medicine**

Abstract: Psoriasis is a chronic inflammatory skin condition which affects approximately 1-3% of the world's population (1). It appears as red plaques covered with silvery scale that flakes away from the skin. Psoriatic plaques are often found on the elbows, scalp and knees but can also affect other parts of the body such as the face, feet and mucous membranes. Psoriasis is not contagious, nor is it caused by an allergy. However, the tendency to develop the condition can be genetically transmitted. Psoriasis causes itching in 60% to 70% of cases (2). However, the exact etiology of psoriasis is unknown. Abnormalities in lipid metabolism have been proposed because the abnormalities in lipids lead to abnormalities in skin formation which is made from lipids. To evaluate the serum lipids profile in psoriatic patients of Al-Anbar governorate and to compare the results with other external studies. The study group included 60 patients with psoriasis, and 30 healthy volunteers. Blood lipid profile was determined using commercial kits from reliable French and Spain companies. All patients had psoriasis involving less than 30% of body surface. Their ages ranged from 10 to 60 years with a mean of 32 years. Family history of psoriasis was positive in a percentage of (20%) of the patients. The mean levels of serum lipids (total cholesterol, triglyceride, low density lipoprotein, and very low density lipoprotein) in patients with psoriasis were found to be significantly higher than those of healthy individuals. The mean levels of high density lipoprotein were not significant. This study strengthens the relationship between the lipids intake, formation, and metabolism with the pathogenesis of psoriasis. Therefore it is concluded that psoriatic patients should be evaluated for hyperlipidemia and obstructive vascular diseases. Adminstrating lipid-lowering medicines for patients particularly cases with severe disease may be beneficial prognosis.

Key Words: Psoriasis, Triglyceride, VLDL, HDL, LDL, and Cholesterol.

Introduction:

Psoriasis is a common disease affecting, as presumed, approximately 120–180 million people worldwide(3). Around 150,000 new cases of psoriasis are reported annually. There are fewer reports on the incidence of psoriasis, but in recent studies an increasing trend over the last 3 decades was shown (3,4). The population prevalence of psoriasis has been reported to range from 2% to 3%. However, in some countries there is a higher prevalence rate for psoriasis, for example in Kazakhstan, Trinidad and Tobago, Paraguay, Kenya, Tanzania, Egypt, and Kuwait (5). Four hundred people die annually from psoriasis-related causes in the Unites States(3). Psoriasis prevalence in the population is affected by genetic, environmental,

viral, infectious, immunological, biochemical, endocrinological, and psychological (trauma, stress) factors as well as alcohol and drug abuse (6,7). Lipid metabolism research studies in psoriasis have been started at the beginning of the 20th century from the quantitative analysis of serum cholesterol in psoriatic patients (8). The abnormal fat metabolism was considered to be an important factor in the etiopathogenesis of psoriasis (3). Normal skin cells mature and replace dead skin every 28-30 days. Psoriasis causes skin cells to mature in less than a week. Because the body can't shed old skin as rapidly as new cells are rising to the surface, raised patches of dead skin develop on the arms, back, chest, elbows, legs, nails, folds between the buttocks, and scalp. Psoriasis is considered mild

if it affects less than 5% of the surface of the body; moderate, if 5-30% of the skin is involved, and severe, if the disease affects more than 30% of the body surface (figure 1) (5).

It was also suggested that continuous separation of psoriatic scales caused the permanent loss of lipids which might affect serum lipid abnormalities^(8,9). Lipid metabolism is a complex process which takes place in different human organs and peripheral blood⁽⁹⁾.

The stratum corneum consists of corneocytes and intracellular lipids, mainly ceramides, sterols, and free fatty acids which form the barrier for diffusion of substances into the skin⁽¹⁰⁻¹³⁾. The lipids are organized into multilamellar intercellular membranes derived from glycerophospholipids, glucocerebrosides, sphingomyelin of the stratum granulosum-stratum corneum interface^(13,14). Then the precursors are converted to ceramides and free fatty acids by the hydrolytic enzymes^(15,16). In psoriasis, alterations in ceramide content have been demonstrated⁽¹⁷⁾, and abnormal lipid structures reported⁽¹⁸⁾. Total lipids, phospholipids, triacylglycerols, and cholesterol were found to increase both in blood and in epidermis of psoriatic patients^(19,20).

Patients and Methods

A total 60 patient with psoriasis were enrolled. Half number of healthy individuals with matching ages were included as controls. The samples were collected from the patients during their visiting to dermatological clinic of Dr. Abdullah Salih Alhasan in Al- Anbar governorate. The ages of the patients ranging between 10-60 years old from both sexes. Many questions were asked to the patients about his name, age, accommodation, occupation, chronic diseases, family history, time of infection, the presence of psychological disturbances, smoking, most common diet, most common drinks, spiritual questions (prayer), time of disease exacerbation, and the factors that exacerbate psoriasis to avoid the interferences with the other diseases, and to find a cause for this disease. All of patients and healthy individuals were not smokers, have no any chronic diseases, and not alcoholic drinkers. After fasting of 14 hours, 12 ml of venous blood was drawn in sterile syringe and centrifuged to separate the serum and then stored at -45°C until being used. The estimation of total cholesterol, high density lipoprotein (HDL) cholesterol, and triglyceride levels were done by using the

enzymatic colorimetric method^(21, 22) from Spin react company (made in Spain). The serum concentration of high density lipoprotein was determined using a colorimetric test kit, a product of BIOLAB REAGENTS, France. VLDL was determined by dividing of triglycerides by 5. LDL was determined by using the following formula:

$$\text{LDL} = \text{Total cholesterol} - (\text{VLDL} + \text{HDL})$$

Results

The study included a total of 90 persons. Among them 60 had psoriasis (35 male and 25 female) and 30 were healthy controls (18 male and 12 female). Their ages ranged from 10 to 60 years with a mean of 32 years. All had psoriatic lesions that involved less than 30% of body surface. Family history of psoriasis was positive in a percentage of (20%) of the patients. The majority of patients (n= 60, 100%) had plaque type psoriasis. The duration of disease ranged between 1 month to 30 years with a mean of 6.9 years. History of seasonal variation of disease was positive in (58.3%) patients. Out of these (15%) noticed exacerbation of disease in winter while (43.3%) in summer season. Bad emotional state exacerbates of about (66.66%) of psoriatic patients, while the other (33.34%) does not affected. This study showed that there is no relationship between the occupation, accommodation, most common diet, most common drinks, and spiritual side and psoriasis. In the patient's group serum cholesterol, triglycerides, low density lipoprotein cholesterol (LDL-cholesterol) and very low density lipoprotein cholesterol (VLDL-cholesterol) were significantly higher than those in control group. While the difference in means of high density lipoprotein cholesterol (HDL-cholesterol) between two groups was not significant statistically. The results are depicted in Table 1.

The values are reported as mean \pm SD and 95% confidence interval. For statistical analysis between groups paired t test was used. Pearson test was used for correlation analysis. The levels of each marker were compared between the study groups and control group, using SPSS computer package. P values of < 0.05 were considered significant. The table above shows that total cholesterol, triglycerides, LDL, and VLDL are higher in patients in a comparison with controls in a significant difference ($P < 0.05$). While there is no significant difference in HDL between patients and controls ($P > 0.05$). Figure 1 and 2.

Table 1: the mean value, S.D, t-value, and p-value of the parameters were tested.

No.	parameters	factor	Mean mg/dl	Std. deviation	t- value	P-value
1	Total cholesterol	patient	227.63	40.839	8.152	0.000
		control	161.10	25.493		
2	Triglycerides	patient	188.62	125.468	3.283	0.001
		control	111.83	34.069		
3	HDL	patient	60.65	14.242	1.703	0.090
		control	55.70	9.990		
4	LDL	patient	129.26	40.653	5.652	0.000
		control	83.03	26.402		
5	VLDL	patient	37.72	25.049	3.283	0.001
		control	22.37	6.814		

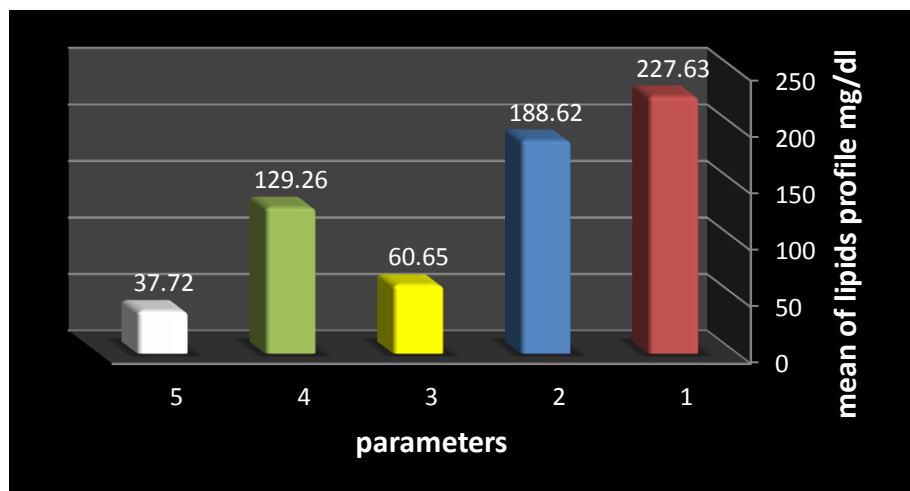


Figure 1: the mean values of patients parameters. 1- cholesterol, 2- triglycerides, 3- HDL, 4- LDL, and 5- VLDL.

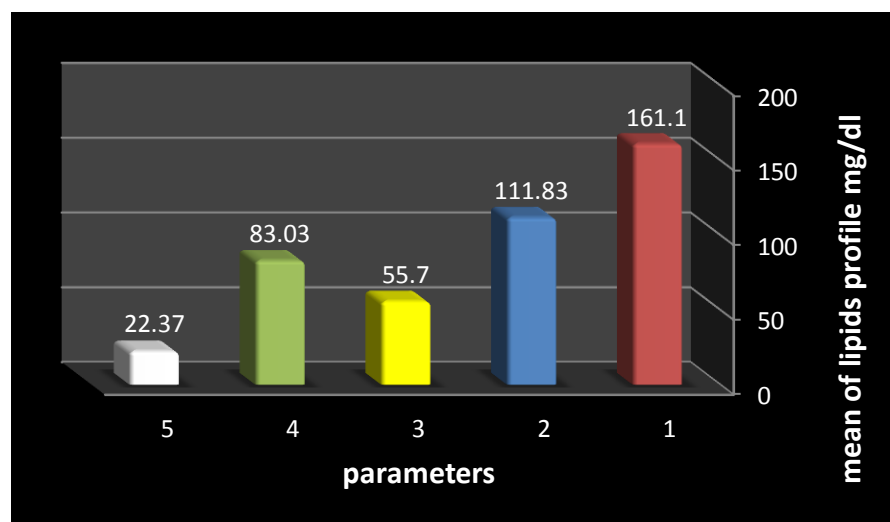


Figure 2: the mean values of controls parameters. 1- cholesterol, 2- triglycerides, 3- HDL, 4- LDL, and 5- VLDL.

DISCUSSION

Psoriasis is a common, chronic inflammatory skin disease characterized by a marked increase in keratinocyte proliferation, abnormal differentiation of keratinocytes, prominent alterations in dermal capillary vasculature and the presence of dermal and epidermal mononuclear leucocytes and neutrophils(23). Several reported studies demonstrate an association of psoriasis with dyslipidemia(24). The present study demonstrated increased total cholesterol, LDL, triglyceride, and VLDL serum levels in patients with psoriasis compared to controls. A cross sectional study indicated increased TC and triglyceride, decreased HDL and no alteration in LDL in psoriasis patients compared to controls (25). In a hospital based cross sectional study in Tikreet, their study demonstrated increased total cholesterol, LDL, triglyceride, and VLDL serum levels and a reduction in serum HDL in patients with psoriasis compared to controls (26). In a hospital based matching study in Iran, psoriasis patients were shown to have significantly higher mean levels of triglyceride, TC, VLDL, LDL and no alteration in HDL (27). A cross sectional study of 84 psoriatic patients attending an outpatient hospital based study in Turkey compared with 40 age and sex matched healthy controls from the community, demonstrated higher mean triglyceride, TC, LDL and lower mean HDL for psoriatic patients versus controls (28). The lipid disturbances are recognized as a very important part in the pathogenesis of psoriasis. The results of the majority of the studies are coherent and indicate that the increased total cholesterol, LDL cholesterol and triglycerides in psoriatic patients' serum are features of the metabolic syndrome. These factors have also a great impact on some features observed in psoriatic patients especially on cardiovascular diseases. These lipid disturbances are also connected with immunological abnormalities, that is why psoriasis could be classified as an immunometabolic disease.

In the patient's group serum cholesterol, triglycerides, low density lipoprotein cholesterol (LDL-cholesterol) and very low density lipoprotein cholesterol (VLDL-cholesterol) were significantly higher than those in control group ($P < 0.05$). While the difference in means of high density lipoprotein cholesterol (HDL-cholesterol) between the two groups was not significant statistically ($P > 0.05$). This may be explained that the stratum corneum consists of corneocytes and intracellular lipids, mainly

ceramides, sterols, and free fatty acids which form the barrier for diffusion of substances into the skin (10-13). The lipids are organized into multilamellar intercellular membranes derived from glycerophospholipids, glucocerebrosides, sphingomyelin of the stratum granulosum-stratum corneum interface (13,14). Then the precursors are converted to ceramides and free fatty acids by the hydrolytic enzymes (15,16). In psoriasis, alterations in ceramide content have been demonstrated (17). we chose this local study to compare the results with other external studies, our results differ with many studies despite the similarity with others, this means that the pathogenesis of psoriasis differs from country to other, therefore we have not to rely on external studies in the treatment and the prognosis of psoriasis. The use of lipids lowering drugs may improves the conditions of psoriasis. Eventually our study strengthens the relationship between psoriasis and atherosclerosis.

References:

- (1) Schon, N 2005 psoriasis general concepts, Engl J Med.
- (2) Sampogna, Br 2004 psoriasis etiopathogenesis, J dermatol.
- (3) Icen M., Crowson C. S., McEvoy M. T., et al. 2009 "Trends in incidence of adult-onset psoriasis over three decades: a population-based study," Journal of the American Academy of Dermatology, vol. 60, no. 3, 394–401.
- (4) Wilson F. C., Icen M., Kremers H. M., 2009 "Incidence and clinical predictors of psoriatic arthritis in patients with psoriasis: a population-based study," Arthritis Care and Research, vol. 61, no. 2, 233–239.
- (5) Chandran V. and Raychaudhuri S. P., 2010 "Geoeidemiology and environmental factors of psoriasis and psoriatic arthritis," Journal of Autoimmunity, vol. 34, no. 3, 314–321.
- (6) Romanowska M., and Al Yacoub, N. 2008 "PPAR δ enhances keratinocyte proliferation in psoriasis and induces heparin-binding EGF-like growth factor," Journal of Investigative Dermatology, vol. 128, no. 1, 110–124.
- (7) Chen Y.-J., Shen J.-L., and Lee F.-Y., 2009 "Elevated plasma osteopontin level is associated with occurrence of psoriasis and is an unfavorable cardiovascular risk factor in patients with psoriasis," *Journal of the American Academy of Dermatology*, vol. 60, no. 2, 225–230.

- (8) Chibowska M., 1970 "Role of serum lipids in psoriasis," *Przegląd Dermatologiczny*, vol. 57, no. 2, 255–260.
- (9) Pietrzak A., Toruniowa B., and J. Chwaluk, 1994 "Lipid profile in psoriatic patients according to sex and age," *Przegląd Dermatologiczny*, vol. 81, no. 5, 441–449.
- (10) Jungersted J.M., Hellgren L. I., and T. Agner, 2008 "Lipids and skin barrier function—a clinical perspective," *Contact Dermatitis*, vol. 58, no. 5, 255–262.
- (11) Bleck O., Abeck D., Ring J. et al., 1999 "Two ceramide subfractions detectable in Cer(AS) position by HPTLC in skin surface lipids of non-lesional skin of atopic eczema," *Journal of Investigative Dermatology*, vol. 113, no. 6, 894–900.
- (12) Landmann L., 1986 "Epidermal permeability barrier: transformation of lamellar granule-disks into intercellular sheets by a membrane-fusion process, a freeze-fracture study," *Journal of Investigative Dermatology*, vol. 87, no. 2, 202–209.
- (13) Elias P. M. and Menon G. K., 1991 "Structural and lipid biochemical correlates of the epidermal permeability barrier," *Advances in Lipid Research*, vol. 24, 1–26.
- (14) Grayson S. and Elias P. M., 1982 "Isolation and lipid biochemical characterization of stratum corneum membrane complexes: implications for the cutaneous permeability barrier," *Journal of Investigative Dermatology*, vol. 78, no. 2, 128–135.
- (15) Man M. Q. M., Feingold K. R., and P. M. Elias, 1996 "Optimization of physiological lipid mixtures for barrier repair," *Journal of Investigative Dermatology*, vol. 106, no. 5, 1096–1101.
- (16) Holleran W. M., Feingold K. and P. M. Elias, 1991 "Regulation of epidermal sphingolipid synthesis by permeability barrier function," *Journal of Lipid Research*, vol. 32, no. 7, 1151–1158.
- (17) Motta S., Monti and Caputo R. ., 1994 "Abnormality of water barrier function in psoriasis: role of ceramide fractions," *Archives of Dermatology*, vol. 130, no. 4, 452–456.
- (18) Ghadially R., Reed J. T., and Elias P. M., 1996 "Stratum corneum structure and function correlates with phenotype in psoriasis," *Journal of Investigative Dermatology*, vol. 107, no. 4, 558–564.
- (19) Khyshiktuev B. S. and Falko E. V., 2005 "Alterations in the parameters of lipid metabolism in different biological objects in psoriatic patients during exacerbation and remission," *Vestnik Dermatologii i Venerologii*, vol. 6, 40–43.
- (20) Ansidei V., Binazzi M., Cantelmi A., Gaiti A., and Porcellati G., 1981 "Phospholipid involvement in psoriatic epidermis," *Italian Journal of Biochemistry*, vol. 30, no. 1, 40–45.
- (21) Buccolo G. et al. 1973 Quantitative determination of serum triglycerides by use enzymes. *Clin. Chem*; 19(5): 476-482.
- (22) Naitoh H.K. Kaplan A et al. 1984 cholesterol , *clin chem. The C.V. Mosby Co. St Louis. Toronto. Princeton* 1194-11206 and 437.
- (23) OrtonneJP. 1999 Recent developments in the understanding of the pathogenesis of psoriasis. *Br J Dermatol*, 140:1-7.
- (24) Neimann AL, Shin DB, Wang X, et al. 2006 Prevalence of cardiovascular risk factors in patients with psoriasis. *J Amer Acad Dermatol*, 55:829-835.
- (25) Cohen AD, Sherf M, Vidavsky L, et al. 2008 Association between psoriasis and the metabolic syndrome. *Dermatol*, 216:152-155.
- (26) Alobaidi A. H. Ahmad, 2006 Biochemical Changes in Psoriasis Lipid Profile, Oxidant and Antioxidant Markers. *J med. Vol. 3 Issue 1*.
- (27) Akhyani M, Ehsani AH, Robati RM, Robati AM. 2007 The lipid profile in psoriasis: a controlled study. *J Eur Acad Dermatol Venereol*, 21:1330-1332.
- (28) Solak Tekin N, Tekin IO, Barut F, Sipahi EY. 2007 Accumulation of oxidized low-density lipoprotein in psoriatic skin and changes of plasma lipid levels in psoriatic patients. *Mediators Inflamm*, 1-5.

دراسة مرتسم الدهون لمرضى الصدفية في محافظة الانبار

وجيه يونس العاني عبد الله صالح حسن اسامة عباس فيصل

E.mail: dean_coll.science@uoanbar.edu.iq

الخلاصة:

الصدفية هي التهاب جلدي مزمن منتشر بنسبة 1-3% من سكان العالم، يظهر بشكل بقع حمراء مغطاة بطبقة من القشور الفضية التي تنتشر من الجلد. تظهر بقع الصدفية على عدة أجزاء من الجسم مثل المرفق، فروة الرأس، الركب، وأماكن أخرى مثل الوجه، الإقدام، والاعشبة المخاطية. الصدفية مرض غير معدي ولا يتسبب بواسطة الحساسية ولكن قابلية الإصابة بالصدفية يمكن أن تنتقل وراثيا. تسبب الصدفية حكة في مكان الإصابة بنسبة 60-70% من المصابين أما النسبة المتبقية فلا يعانون من حكة. السبب المرضي للصدفية غير معروف، ولكن لوحظ اضطراب في أيض الدهون مصاحب للمرض ويفسر ذلك بأن اضطراب الدهون يؤدي إلى خلل في تكوين طبقات الجلد التي تتكون من الدهون. هدفت هذه الدراسة إلى تقييم صورة الدهون عند المرضى المصابين بداء الصدفية في محافظة الانبار ومقارنة النتائج مع دراسات أخرى خارجية. تضمنت الدراسة 60 مريض و 30 متبرع سليم. تم قياس صورة دهون الدم لهم باستخدام الكتات التجارية من شركات معتمدة إسبانية وفرنسية. نسبة الإصابة بداء الصدفية لجميع المرضى كانت أقل من 30% من مساحة الجلد، وتتراوح أعمارهم بين (10-60) سنة وبمعدل 32 سنة. تاريخ العائلة مع الصدفية كان ايجابيا بنسبة 20% من مجموع المرضى. مستويات دهون المصل (الكولسترول الكلي، الدهون الثلاثية، الدهون البروتينية واطئة الكثافة، الدهون البروتينية واطئة الكثافة جدا) عند مرضى الصدفية كانت مرتفعة مقارنة مع الأشخاص الأصحاء، أما الدهون البروتينية عالية الكثافة عند مرضى الصدفية لم تسجل فرق معنوي مقارنة مع الأشخاص الأصحاء. هذه الدراسة تشدد على وجود علاقة بين تناول، تكوين، وإيض الدهون مع سبب المرض. لذلك نستنتج أن مرضى الصدفية يكونون عرضة لارتفاع دهون الدم وبالتالي انسداد الاوعية الدموية. استخدام الادوية الخافضة لدهون الدم للمرضى المصابين بداء الصدفية وخصوصا الحالات الشديدة يكون مفيد للمرضى ولا بد من متابعة صورة الدهون لديهم.