Study of Serum Leptin and Lipid Profile Concentration in Psoriatic Patients in Hilla - Iraq

Radwan A. Al-Asadi; Moaed E. Al-Gazally and Mohammed K. Al-Hattab

Department of Biochemistry-College of Medicine - Babylon University / Hilla - Iraq

Abstract

Background: To study the role of biochemical markers in sera of psoriatic patients.
Aim: Evaluation and study of the correlation between lipid profiles and Leptin in sera of psoriatic patients in Hilla province – Iraq.
Patients and Methods: The study was conducted on sixty psoriatic patients and thirty apparently healthy persons were taken as control group. Blood collected from the clinic of dermatology in Merjan Teaching Hospital and from the consultative center for allergy and asthma in Hilla city / Iraq. Sera obtained from the blood were used to determine the effect of psoriasis on leptin, total cholesterol, high density lipoprotein (HDL-C), triglycerides (TGs), very low density lipoprotein (VLDL), and low density lipoprotein cholesterol (LDL-C) concentration.
Results and Discussion: The results of the present study showed (significant increase in leptin, total cholesterol, triglycerides, very low density lipoprotein and low density lipoprotein cholesterol concentration), significant decrease in high density lipoprotein cholesterol in sera of psoriasis group compared to control group, significant increase in leptin concentration in sera of psoriasis females than males, significant increase in leptin concentration in sera of psoriasis patients as increase BMI (body mass index).

الخلاصة

الخلفية : لمتابعة المتغيرات الكيموحيوية في أمصال مرضى داء الصدفيّة.
الهدف: دراسة المتغيرات الكيموحيوية ومنها صورة الدهون واللبتين في أمصال مرضى داء الصدفيّة.
المرضى وطرائق العمل: أجريت الدراسة على ستين مريض بداء الصدفية وثلاثين شخص سوي أخذت كمجموعة سيطرة. تم الحصول على عينات الدم من مرضى الصدفية ومجموعة السيطرة في مستشفى مرجان التعليمي ومركز الحساسية والربو في مدينة الحلة. المصول تم الحصول عليها من الدم لقياس تأثير داء الصدفية على هرمون النحافة و الكولسترول الكلي و البروتينات الدهنية عالية الكثافة و الكليسيريدات الثلاثية والبروتينات الدهنية واطئة الكثافة جدا و البروتينات الدهنية واطئة الكثافة .
النتائج ومناقشتها: أظهرت نتائج هذه الدراسة (زيادة ملحوظة في تركيز هرمون النحافة و الكولسترول الكلي و الكليسيريدات الثلاثية والبروتينات الدهنية واطئة الكثافة جدا و البروتينات الدهنية واطئة الكثافة) و نقصان ملحوظ في البروتينات الدهنية عالية الكثافة ومقارنتها بمجموعة السيطرة. كذلك هذه الدراسة أظهرت زيادة ملحوظة في تركيز هرمون النحافة في مصول النساء المصابات بمرضى الصدفية مقارنة بالرجال المصابين بالصدفية وزيادة ملحوظة في هرمون النحافة تتزامن مع الزيادة في مؤشر كتلة الجسد.

Introduction

Psoriasis is a common chronic, immune-mediated, inflammatory disease of the skin [(1)]. It is relapsing, non-contagious disorder characterized by red patchy lesions, with grey or silvery-white, dry scales [(2)].

Lesions are typically distributed symmetrically on the scalp, elbows, knees and essentially any part of the body. It is a disease with an unpredictable course, prone for flare-

ups and remissions and can affect the joints and nails [3].

Psoriasis is generally categorised into one of three severities based on the extent of body surface covered. Where 2% of the body is affected, it is classified as mild, where 3-10% of the body is covered, it is classified as moderate and where more than 10% of the body is affected, the disease is classified as severe. Based on these criteria, approximately 25-30% patients have psoriasis, which is considered moderate to severe [4].

It can afflict both men and women, and usually begins in early adulthood although it has been reported at birth. There is a bimodal distribution in the age of onset. Type I or early onset psoriasis typically appears in individuals between ages 15 to 20 years and shows a tendency to disseminate, greater number of relapses, and higher frequency of familiar history of psoriasis when compared with Type II or late onset psoriasis during or after the fifth decade of life [5, 6, 7].

Psoriasis is distributed worldwide but its prevalence varies among different geographical areas and ethnic groups [8]. In Iraq, approximately 2.3% of the population is affected [9]. The immune system has been strongly implicated in the pathogenesis of psoriasis that resembles a T cell-mediated disease [10]. T cells are found in the dermis and epidermis and are accompanied by increased numbers of dermal dendritic cells, macrophages and mast cells [11].

Several environmental factors are recognised as triggers and exacerbators for psoriasis among which: infections [12], alcohol and smoking [13], family history (genetics) [14], trauma [15], stress [16], drugs [17] and diet [18].

Clinical types of psoriasis can be classified according to phenotype-based classification that intended for use in both clinical practice and researches [19]: plaque psoriasis, guttate (Eruptive) psoriasis, generalized pustular psoriasis, palmoplantar pustular psoriasis, psoriatic arthropathy, erythrodermic psoriasis, scalp psoriasis, nail psoriasis.

Leptin is a 16 kD (kilo Dalton) protein hormone. The primary amino acid sequence indicated that leptin may adopt a helical cytokine structure similar to interleukin-2 (IL-2) and growth hormone, and this led to the early indication that its receptor might be a member of the hemopoietin receptor family [20].

Human leptin is a protein of 167 amino acids. It is manufactured primarily in the adipocytes of white adipose tissue, and the level of circulating leptin is directly proportional to the total amount of fat in the body. White adipose tissue is the major source of leptin in addition to it can also be produced by brown adipose tissue, placenta, ovaries, skeletal muscle, stomach (lower part of fundic glands), mammary epithelial cells, bone marrow, pituitary gland ,liver and gastric chief cells [21,22].

Leptin plays a key role in regulating energy intake and energy expenditure, appetite and metabolism. Leptin acts on receptors in the hypothalamus of the brain, it works by inhibiting the activity of neurons that contain neuropeptide Y (NPY) and agouti-related peptide (AgRP), and by increasing the activity of neurons expressing α-melanocyte-stimulating hormone (α-MSH) [23,24].

Leptin promote angiogenesis by increasing vascular endothelial growth factor (VEGF) levels. In some epidemiological studies, hyperleptinemia is considered as a risk factor for atherosclerosis [25, 26].

The major lipids present in the plasma are fatty acids, TGs, cholesterol and phospholipids, are all transported

in plasma as lipoprotein particles [chylomicrons (CM), very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), low density lipoprotein (LDL) and high density lipoprotein (HDL)] [27].

Materials and Methods

Materials

Subjects

The study was conducted over a period of eleven months from October 2010 till August 2011. Samples collected from clinic of dermatology in Merjan Teaching Hospital and consultative center for allergy and asthma in Hilla-Iraq. The practical side of the study was performed at the laboratory of biochemistry department in College of Medicine/Babylon University.

This study included sixty psoriatic patients and forty healthy were taken as control group. A questionnaire was designed to obtain the information from psoriasis patients and control group. It contained the name, age, weight, height and smoking.

The general criteria for all subjects in this study included not those suffering from any disease (e.g. hypertension, diabetes mellitus, asthma etc.), not given any medication (e.g. methotrexate, diuretics, steroid, etc.) for at least one month, not drinking alcohol, not smoking and not pregnant women. Subjects that had these criteria were excluded from this study.

The psoriasis group comprised sixty adults (27 men and 33 women), aged 18-77 year with mean ± SD of 46.18 ± 15.23year. All patients had not received any treatment for psoriasis for at least one month prior to blood collection and not received any medication for other diseases. The control group includes forty apparently healthy individuals (18 men and 22 women) aged 16-76 year with mean ± SD 43.43 ± 18.6 year.

Blood Sampling:

Venous blood samples were drawn from psoriasis and control subjects by using disposable syringes (5mL) in the sitting position. Five mls of blood were obtained from each subject by vein puncture and pushed slowly into plain disposable tubes. Blood was allowed to clot at 37˚C for 10-15 minutes, and then centrifuged at 1800 Xg for approximately 10-15 minutes then the sera were obtained and stored at -20˚C until analysis (measure leptin, total cholesterol, HDL-C, TGs, VLDL and LDL-C).

Methods

Serum leptin concentration was determined by Mediagnost (Germany) kit. Serum total cholesterol, triglycerides and HDL-C concentration were determined by Biolabo SA (France) kit. VLDL-C concentration was calculated by dividing triglycerides value by 2.22 [28]. LDL-C concentration was calculated by using Friedewald equation [29].

Results

Total cholesterol, HDL-C, TG, VLDL and LDL-C concentration were measured in sera of sixty psoriatic patients and thirty healthy (control group) as shown in table (1).

The results in table (1) show significant increase in (total cholesterol, triglycerides, VLDL and LDL-C concentration), significant decrease in HDL-C concentration in sera psoriasis group compared with those of the control group.

Leptin concentration were measured in sera of sixty psoriatic patients and thirty healthy (control group) as shown in table (2).

Also this study showed significant increase in leptin concentration in sera

of psoriasis group compared with those of the control group, significant increase in leptin concentration in sera of males in psoriasis group compared to those of control group, significant increase in leptin concentration in sera of females in psoriasis group compared to those of control group

Table 1: Serum total cholesterol, HDL-C, TG, VLDL and LDL-C concentration in psoriasis and control group. (P-value of < 0.05 was considered to be statistically significant)

Parameter	Subjects	No.	Mean ± SD	P-value
Total-Cholesterol (mmol/L)	Psoriasis group	60	5.38 ± 0.88	P< 0.001
	Control group	40	4.58 ± 0.83	
HDL-C (mmol/L)	Psoriasis group	60	1.1 ± 0.28	P< 0.001
	Control group	40	1.48 ± 0.44	
Triglycerides (mmol/L)	Psoriasis group	60	1.61 ± 0.91	P< 0.001
VLDL-Cholesterol (mmol/L)	Control group	40	1.05 ± 0.93	
	Psoriasis group	60	0.72 ± 0.41	P< 0.001
LDL-C (mmol/L)	Control group	40	0.47 ± 0.24	
	Psoriasis group	60	3.43 ± 0.93	P< 0.001
	Control group	40	2.73 ± 0.94	

Table 2: Serum leptin concentration in psoriasis and control group. (P-value of < 0.05 was considered to be statistically significant)

	Subjects	No.	Mean ± SD	P-value
Leptin (ng /ml)	Psoriasis group , male	27	11.25 ± 2.57	P< 0.001
	Psoriasis group ,female	33	16.07± 2.82	
	Control group , male	18	5.7± 1.09	P< 0.001
	Control group, female	22	10.76± 2.1	

Significant increase in leptin concentration also found in sera of females in psoriasis group compared to males of the same group, also significant increase in leptin concentration found in sera of females in control group compared to males of the same group.

Discussion

Leptin:

One of the major findings of the present study was the significant increase in serum leptin concentration observed in psoriasis group compared

to the control group. The results of the present study were in agreement with Amira *et al*. [30] study in which they found people with psoriasis to have higher levels of the obesity-related hormone (leptin) than those without psoriasis and disagree with Aktan, *et al*. [31] study in which they found there was non significant difference between serum leptin levels of psoriasis and control group.

There are two possible mechanisms by which plasma leptin level increased in psoriatic patients:

First mechanism plasma leptin levels are strongly correlated with adiposity. Tissue adiposity along with gender is the main determinant of leptin gene expression and release [32].

Second mechanism is inflammation process, the proinflammatory mediators in psoriasis stimulate leptin expression, which eventually leads to metabolic dysregulation. Different inflammatory stimuli, including IL-1, IL-6, and lipopolysaccharides, regulate leptin messenger RNA expression and circulating leptin levels [33].

Studies have been performed to investigate the possible role of tissue leptin levels in the pathogenesis of psoriasis. This can be explained in view of previous studies Bernotiene *et al* [34] that showed the leptin modulates T-helper cell activity. In those studies leptin was found to activate monocytes and macrophages, potentiating the production of various proinflammatory cytokines TNF- α, IL-6 (Interleukin) and directing T- cell differentiation to Th1 phenotype [35].

Leptin has been shown to stimulate keratinocyte proliferation, expression of adhesion molecules and angiogenesis which is characteristic for psoriasis [36].

There are several causes lead to significant increase in serum leptin levels in women than in men for both psoriasis and control group:

First: Women in general have more body fat than men and different fat distribution. The serum leptin concentration is closely positively related to fat mass. Women have more subcutaneous fat than visceral fat, while the opposite condition is present in men. It has been shown that subcutaneous fat expresses more leptin mRNA than abdominal fat, this may partially explain the gender differences in leptin levels between the sexes [37].

Second: In women, estrogen was positively associated with leptin in agreement with previous report, Shimizu H. *et al* [38] they found estrogen increases in vivo leptin production. Androgens are known to play an important role in normal fat distribution [39].

Third: Testosterone levels were inversely associated with leptin levels in men. In addition a negative association between testosterone and leptin was also observed in women [40]. In obese men increased BMI was associated with a substantial reduction in testosterone in men. In women, however, an association between testosterone and BMI was only found in overweight women. It is likely that testosterone plays a causal role in visceral fat accumulation [41].

Lower testosterone levels have been reported to predict visceral obesity and replacement doses of testosterone decreases abdominal fat mass in men with low testosterone levels [42,43].

The results of the present study were in agreement with Reidun O *et al* [40] they found that gender differences increase with increasing BMI and differences in life style parameters are predictors of serum leptin, serum leptin is especially low in men than women.

Lipid profile (Total cholesterol, HDL-C , TG, VLDL and LDL-C):

The results of present study show significant increase in (total cholest-

erol, triglycerides, VLDL and LDL-C concentration) in sera of psoriasis group compared to those control group and significant decrease in HDL-C concentration in sera of psoriasis group compared to control group.

The results of the present study were in agreement with other studies, Amina H. [44], Gurkok Fet al[45], Reynoso-von Drateln C. et al[46] they showed that lipid profile in psoriatic patients undergoes some considerable changes in which the levels of total cholesterol, LDL-C, VLDL and triglycerides were significantly increased and decreased HDL-C concentration.

In another assay Piskin S. et al [47] they found that total cholesterol and LDL-C levels were to be significantly higher in patient with psoriasis than those of controls, but no significant differences were found in the triglyceride and VLDL levels.

In a hospital based cross sectional study in Iran, psoriasis patients were shown to have significantly higher mean levels of triglyceride, total cholesterol, VLDL, LDL-C and no alteration in HDL-C [48].

A cross sectional study indicated increased total cholesterol and triglyceride, decreased HDL-C and no alteration in LDL-C in psoriasis patients compared to controls [49].

There is an increased interest in HDL-C concentration, because there is an inverse relationship between the level of HDL-C and the development of atherosclerosis. HDL-C is a very important factor in reverse cholesterol transport (RCT).

It takes part in the transport of cholesterol produced or accumulated in the peripheral tissues to the liver or other steroidogenic tissues and exerts the antioxidant, anti - inflammatory, antithrombotic and fibrinolytic action [50].

In psoriasis, a decrease of HDL synthesis and HDL structural changes can be observed, due to various biochemical disturbances, such as abnormalities of receptor function, changes of hepatic structure and function, activity changes of hepatocyte membranes, impaired RCT, esterification, and lipases [51].

The activation of the immune system in psoriasis may cause some changes in lipid profile of patients. However, these changes may be related to some abnormalities of the digestive system. The digestive system takes part in the decomposition, modification, and synthesis of many organic compounds, including lipids. In psoriatic patients, structural and functional abnormalities have been found in nearly all the segments of the gastrointestinal tract [52].

Blood lipid profile (total cholesterol, HDL-C, LDL-C and TGs) is a useful tool in determining the risks of cardiovascular diseases. LDL is bad cholesterol being associated in deposition of cholesterol on the walls of arteries and HDL is good cholesterol being associated in carrying cholesterol out of the blood system and is more compact than LDL-C [53].

Conclusions

The study concluded that psoriasis causes undesirable effects on leptin and lipid profile.

Recommendations

1. Evaluation of resistin in sera of psoriasis patients and correlates its concentration with disease severity.
2. Determination of insulin in sera of psoriasis patients and correlates its concentration with leptin concentration.

3. Determination of serum selenium concentration in sera of psoriasis patients.
4. Determination of serum testosterone concentration in sera of psoriasis patients.

References

1. Pariser D., Bagel J. and Gelfand J. (2007). National psoriasis foundation clinical consensus on disease severity .Arch Dermatology; 143:239–242.
2. Picardi A., Mazzotti E., Gaetano P., *et al*. (2005). Stress, social support, emotional regulation, and exacerbation of diffuse plaque psoriasis psychomatics; 46:556–564.
3. Campalani E. and Barker J. (2005). The clinical genetics of psoriasis; 6:51-60.
4. Gilliard S. and Finlay A. (2005). Current management of psoriasis in the United Kingdom: patterns of prescribing and resource use in primary care. Int J Clin Pract; 59 (11): 1260-1267.
5. Langley R., Krueger G. and Griffiths C. (2005). Psoriasis: epidemiology, clinical features, and quality of life. Ann Rheum Dis; 64 (Suppl. II): 18-23.
6. Kormeili T., Lowe N. and Yamauchi P. (2004) Psoriasis: immunopathogenesis and evolving immunomodulators and systemic therapies; U.S. experiences. Br J Dermatol; 151:3-15.
7. Christophers E. (2001). Psoriasis epidemiology and clinical spectrum. Clin Exp Dermatol; 26:314-20.
8. Raychaudhuri S. and Farber E. (2001). The prevalence of psoriasis in the world J Eu Acad Dermatol Venereol; 15:16-7.
9. Alsamarai A. (2008). Prevalence of skin diseases in Iraq. Int J Der.
10. Bowcock A. (2005). Getting under the skin: the immunogenetics of psoriasis. Nat Rev Immunol; 5:699–711.
11. Nickoloff B. and Nestle F. (2004). Recent insights into the immunopathogenesis of psoriasis provide new therapeutic opportunities. J Clin Invest; 113:1664–1675.
12. Naysmith L. and Rees L. (2003). Psoriasis and its management. J. R Coll Physicians Edinb ; 33: 104-113.
13. Cohen A. and Halevy S. (1999). Alcohol intake, immune response, on the skin. Clin Dermatol ; 17(4): 411-412.
14. Neimann A., Porter S. and Gelfand J. (2006). The epidemiology of psoriasis. Future Drugs Ltd.
15. Miller R. (1982).The Koebner phenomenon. Int J Dermatol; 21:192-197.
16. Mease P. and Menter M. (2006). Quality-of-life issues in psoriasis and psoriatic arthritis, outcome measures and therapies from a dermatological perspective. J Am Acad Dermatol; 54:685-704.
17. Hunter J., Savin J. and Dahi M. (1996). London Blackwell science Clinical dermatology 2nd edition: 51-54.
18. Horrobin D. (1987). Low prevalence of coronary heart disease, asthma, psoriasis, rheumatoid arthritis in Eskimos are caused by high dietary intake of eicosapentanoic acid, a genetic variation of essential fatty acids metabolism or a combination of both .Med hypothesis;22 :421-428.
19. Griffiths C., Christophers E., Barker J., *et al*. (2007). A classification of psoriasis vulgaris according to phenotype. Br J Dermatol; 156:258–262.
20. Metcalf D. (1993). Hematopoietic regulators: Redundancy or subtlety. Blood; 82:3515-3519.
21. Margetic S., Gazzola C., Pegg G., *et al*. (2002). Leptin: a review of its peripheral actions and interactions". Int. J. Obes. Relat. Metab. Disord. 26 (11): 1407–1433.
22. Bado A., Levasseur S., Attoub S., *et al*. (1998). The stomach is a source of leptin. Nature 394 (6695): 790–793.
23. Brennan A. and Mantzoros C. (2006). Drug Insight: the role of leptin in human physiology and pathophysiology--emerging clinical applications. Nat Clin Pract Endocrinol Metab 2 (6): 318–327.
24. Dubuc G., Phinney S., Stern J., *et al*. (1998). Changes of serum leptin and endocrine and metabolic parameters after 7 days of energy restriction in men and women". Metab. Clin. Exp. 47 (4): 429–434.
25. Zhang W., Telemaque S., Augustyniak R., *et al*. (2010). Adenovirus- mediated leptin expression normalises hypertension associated with diet-induced obesity". J Neuroendocrinol. 22 (3): 175–180.
26. Knight W., Seth R., Boron J., *et al*. (2009). Short-term physiological hyper-leptinemia decreases arterial blood pressure". Regul Pept. 154 (1-3): 60–68.
27. Smith A., Beckett G., Walker J., *et al.* (2000). Disorders of plasma lipids and lipoproteins. Lectures Notes on Clinical Biochemistry; 6th ed. Blackwell Science. 101–104.

28. Godkar P. (1994). Textbook of Medical Technology, Clinical Biochemistry: Principles and Practice, Bhalani publishing house, Bombay. India. 223–225.
29. Carl A. and Edward R. (2006). Tietz text book of clinical Biochemistery and Molecular Diagnostics; 4th ed. 948.
30. Amira A., Nabila A. and Halla M. (2010). Serum and Tissue Leptin Levels in Relation to Psoriasis vulgaris Severity. Journal of American Science; 6(10):412-422.
31. Aktan F., et al. (2007). A Role of Leptin in Psoriasis. Turk J Med Sci; 37 (3): 135-138.
32. Faggioni R., Feingold K. and Grunfeld C. (2001).Leptin regulation of the immune response and the immunodeficiency of malnutrition. FASEB J; 15(14):2565-2571.
33. Sanna V., Di Giacomo A., La Cava A., *et al*. (2003).Leptin surge precedes onset of autoimmune encephalomyelitis and correlates with development of pathogenic T cellresponses. *J Clin Invest*. ; 111(2) :241-250. Bernotiene E., Palmer G. and Gabay C. The role of leptin in innate and adaptive immune responses. Arthritis Res Ther 2006; 8: 217 - 220.
34. Cao R., Brakenhielm E., Wahlestedt C. *et al.* Leptin, induces vascular permeability and synergistically stimulates angiogenesis with FGF-2 and VEGF. Pro Acad Sci USA 2001; 98: 6390 -6395.
35. Havel P. (2000). Role of adipose tissue in body-weight regulation: mechanisms regulating leptin production and energy balance. Proceedings of the Nutrition Society; 59: 359–371.
36. Trayhurn P., Duncan J., Hoggard N., *et al.* (1998). Regulation of leptin production: a dominant role for the sympathetic nervous system? Proceedings of the Nutrition Society 57 413–419.
37. Lonnqvist F., Arner P., Nordfors L., *et al*. (1995).Overexpression of the obese (ob) gene in adipose tissue of human obese subjects. Nat Med; 1:950-953.
38. Shimizu H., Shimomura Y., Nakanishi Y., *et al*. (1997).Estrogen increases in vivo leptin production in rats and human subjects. J Endocrinol; 154: 285-292.
39. Björntorp P. (1992).Male fat distribution and cardiovascular risk. Blood Press Suppl; 4: 17-19.
40. Reidun O., Jon F., Johan S., *et al.*(2011). Leptin in the General Population, Differences in Sex Hormones, Blood Lipids, Gender and Life Style Characteristics. The Open Behavioral Science Journal; 5: 8-15.
41. Dua A., Hennes M., Hoffmann R., *et al*. (1996).Leptin: a significant indicator of total body fat but not of visceral fat and insulin insensitivity in African-American women. Diabetes; 45: 1635-1637.
42. Khaw K. and Barrett E. (1992).Lower endogenous androgens predict central adiposity in men. Ann Epidemiol ; 2: 675-682.
43. Rebuffe M., Marin P. and Björntorp P. (1991).Effect of testosterone on abdominal adipose tissue in men. Int J Obes; 15: 791-795.
44. Amina H. (2009).Biochemical changes in psoriasis: lipid profile, oxidant and antioxidant markers .Middle East Journal of Internal Medicin ; volum 2,issu3:27-34.
45. Gurkok F., Piskin S., Ekuklu G. (1999). Serum lipid and lipoprotein levels in psoriasis. Bull Leprosy; 30: 105–111.
46. Reynoso-von Drateln C., Martinez-Abundis E., Balcazar-Munoz B.,*et al*. (2003).Lipid profile, insulin secretion, and insulin sensitivity in psoriasis. J Am Acad Dermatol; 48: 882–885.
47. Piskin S., Gurkok F., Ekuklu G., *et al*. (2003).Serum lipid levels in psoriasis. Yonsei Med J; 44: 24–26.
48. Akhyani M., Ehsani A., Robati R.*et al*. (2007) .The lipid profile in psoriasis: a controlled study. European Academy of Dermatology and Venereology; 21:1330-1332.
49. Cohen A., Sherf M., Vidavsky L., *et al*. (2008).Association between psoriasis and the metabolic syndrome. Dermatol; 216:152-155.
50. Kuliszkiewicz-Janus M., Mohamed A. and Abod N. (2006).The biology of HDL lipoprotein and its antisclerotic activity, Postepy Higieny i Medycyny Do´ swiadczalnej; vol. 60, pp: 307– 315.
51. Wakkee M. (2010).Psoriasis: comorbidity and treatment, Doctoral thesis, Erasmus University Rotterdam, Rotterdam, The Netherlands.
52. Pietrzak A., Lecewicz-Torun B. and Kadziela-Wypyska G. (1998).Changes in the digestive system in patients suffering from psoriasis. Ann Univ Mariae Cruie Sklodowska [Med]; 53 187–194.
53. Faiqa F., Muhammad N., Saman B., *et al.* (2006). Dietary effects on hemo-lipid profile; in university students of middle income group.Professional Med J; 13 (4): 621–626.