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Research Article

Does Lichen Planus Disease Increase Cardiovascular Risk?

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Abstract

Background and Aim: Lichen Planus (LP) is assumed to be closely related to dyslipidaemia. Several cytokines involved in LP pathogenesis, could explain its association with dyslipidaemia. Furthermore, chronic inflammation has been found to play an important role in the development of cardiovascular risk factors. The aim of our study is to evaluate the inflammation markers, Neutrophil/ Lymphocyte Ratio (NLR), and coagulation markers MPV, MPV/ platelet ratio and other laboratory parameters.

Materials and Methods: Fifty seven patients with diagnosis of LP according to clinical and histopathologic findings, as well as 40 age-sex matched controls were recruited to the study. The control group were selected from people without any systemic disease, medication or infection in the previous month. The laboratory parameters were retrospectively scanned. Differences between patients and controls were evaluated, depending on data type, using parametric or nonparametric tests, for independent or paired samples. A p-value < 0.05 was considered significant.

Results: There was not a statistically significant difference in NLR values between patient and control groups whereas difference was significant in MPV/ platelet rate (p=0.01).

Conclusion: The patients with LP were found to have higher MPV/ platelet ratio compared to the control group independent of age, sex, disease duration and clinical types.

Keywords: Lichen Planus; Cardiovascular Risk; MPV; MPV/Platelet Ratio

Introduction

Lichen planus (LP) is a chronic inflammatory disease that affects the skin, appendages and mucous membranes. LP occurs mostly over 45 years old, and is more common among women [1]. The pathogenesis of LP is considered multifactorial, especially the evidence indicates that an imbalance of immunologic cellular reactivity is central [2,3]. Although LP is an immune-mediated disease, the keratinocytes are known to release more cytokines (pro-inflammatory and anti-inflammatory) (TNF-α, IL-2, IL-4, IL-6 and IL-10) during the lymphocytotoxic process [3,4]. Additionally, the dyslipidemia seen in these subjects may be attributed to these proinflammatory cytokines. The recent studies reported an association between LP and dyslipidaemia [5-8]. They suggested chronic inflammation may result with dyslipidemia. Dyslipidaemia is a well-established cardiovascular risk factor for coronary artery disease, stroke, myocardial infarction and cardiovascular deaths [9,10]. Recent advances in clinical laboratory techniques have opened new horizons for a better understanding of the role of platelets in thrombosis, immunity, inflammation and angiogenesis. Blood platelets participate actively in immune-inflammatory processes [11,12]. Immune inflammatory processes associated with skin diseases could induce platelet activation, which, in turn, would contribute to acceleration and modulation of these processes. Mean Platelet Volume (MPV), an indicator of platelet activation, is a newly emerging risk factor for atherothrombosis. According to our knowledge, the MPV/platelet ratio is superior to the MPV alone in predicting long-term mortality [13,14]. As LP is a chronic inflammatory disease, LP patients may have increased cardiovascular risk factors in the long term. The aim of our study is to evaluate the inflammation markers, Neutrophil/ Lymphocyte Ratio (NLR), and coagulation markers MPV, MPV/ platelet ratio and other laboratory parameters.

Materials and Methods

The study was performed by the dermatology unit of Afyon Kocatepe University, after obtaining the approval of the local ethical committee (the committee's approval number of ethics: 2015/04-30). In our study, 57 patients with diagnosis of LP according to clinical and histopathologic findings, as well as 40 age-sex matched controls were recruited to the study. The control group was selected from people without any systemic disease, medication or infection in the previous month.

The laboratory parameters were retrospectively scanned. Differences between patients and controls were evaluated, depending on data type, using parametric or nonparametric tests, for independent or paired samples. A p-value < 0.05 was considered significant.

Results

Of the 57 patients, 35 were women and 22 were men. Of the 40

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Table 1: The laboratory parameters and demographic datas of patients and controls.

	Patients	*Controls*	*P values*
N	Cutaneous LP*: 40 Oral LP: 7 lichen planopilaris: 2 Oral+ Cutaneous LP: 8 Total: 57	40	-
Age	38.05 ± 12.28	36.44 ± 13.06	0.05>
Sex	F: 35, M: 22	F: 22, M: 18	0.05>
Disease Duration 0-1 month 2-6 month 6 month<	 n:1 n:11 n:45	-	-
MPV**	9.61 ± 0.22	8.65 ± 0.32	0.05>
MPV/ platelet	0.04 ± 0.02	0.03 ± 0.01	0.018
NLR***	2.21 ± 1.01	2.07 ± 0.67	0.05>

*LP: Lichen Planus; **MPV: Mean Platelet volume; ***NLR: Neutrophil lymphocyte ratio.

controls 22 were women and 18 were men. There were 40 cutaneous LP, 7 oral LP, 2 lichen planopilaris, 8 cutaneous plus oral LP. LP disease duration in Table 1. The five patients with LP were positive HBV antigen, and 2 patients with LP were positive HCV antigen. There was not a statistically significant difference in NLR values between patient and control groups whereas difference was significant in MPV/platelet rate (p=0.01). We reported results of laboratory parameters and demographic data of patients and controls in Table 1.

Discussion

LP presents with definite plasma lipid profile aberrations [5-8]. Additionally, LP was previously reported to be associated with an abnormal carbohydrate metabolism in epidermal cells and a higher prevalence of diabetes than in the general population [15]. However, there are only a few studies which evaluate relation between LP cardiovascular risk, emerging evidence suggests that severe psoriasis is associated with increased risk of cardiovascular disease [16].

Contemporary scientific literature emphasize a strong association between chronic inflammation and increased cardiovascular disease. The findings of recent studies are important in that it adds to the growing evidence of a relation between LP and cardiovascular risk factors like dyslipidemia and metabolic syndrome. Saleh et al. also evaluated homocysteine and other cardiovascular risk factors in patients with LP. They found that patients with LP were found to have higher makers of both metabolic and cardiovascular risk factors in relation to controls most probably due to long standing inflammation [17].

Recently, the N/L ratio and MPV/platelet have been proposed as an indicator for systemic inflammatory status in several diseases, including atherosclerosis, myocardial infarction, diabetes mellitus, hypertension, metabolic syndrome, psoriasis and coronary artery ectasia [11,14,18,19]. Using the differential WBC count this values can easily be calculated. Unlike novel inflammatory biomarkers, it is cost effective and readily available. The N/L ratio and MPV/ platelet have not been evaluated in patients with LP yet. To our knowledge, this is the first study which investigated the role of N/L ratio and MPV/platelet as a measurement of systemic inflammation in patients with LP.

As the study was retrospective in nature, lipid profiles of all patients could not be reached and we could not evaluate and comment on dyslipidemia in LP patients.

Conclusion

Consequently, patients with LP were found to have higher MPV/ platelet ratio compared to the control group independent of age, sex, disease duration and clinical types. LP is a systemic disease which may increase cardiovascular risk. Further studies are necessary to make a comprehensive comment on cardiovascular risk in patients with LP.

References

1. Daoud MS, Freedberg IM, Eisen AZ, Wolff K, et al. Pittelkow MR: Lichen planus. In: Fitzpatrick's Dermatology in General Medicine. Ed. 5th ed. Philedelphia: McGraw-Hill Co.1999: 561-577.
2. Sezer E, Ozugurlu F, Ozyurt H, Sahin S, Etikan I. Lipid peroxidation and antioxidant status in lichen planus. Clin Exp Dermatol. 2007; 32: 430-434.
3. Meller S, Gilliet M, Homey B. Chemokines in the pathogenesis of lichenoid tissue reactions. J Invest Dermatol. 2009; 129: 315-319.
4. Simark-Mattssona C, Bergenholtza G, Jontell M, Eklund C, Seymour GJ, Sugerman PB, et al. Distribution of interleukin-2, -4, -10, tumour necrosis factor-a and transforming growth factor-b mRNAs in oral lichen planus. Arch Oral Biol. 1999; 44: 499-507.
5. Lppez-Jornet P, Camacho-Alonso F, Rodriguez-Martines MA. Alterations in serum lipid profile patterns in oral lichen planus: a cross-sectional study. Am J Clin Dermatol. 2012; 13: 399-404.
6. Krishnamoorthy B, Gn S, N S M, M B S, Garlapati K. Garlapati K. Lipid profile and metabolic syndrome status in patients with oral lichen planus, oral lichenoid reaction and healthy individuals attending a dental college in northern India - a descriptive study. J Clin Diagn Res. 2014; 8: 92-95.
7. Dreiher J, Shapiro J, Cohen AD. Lichen planus and dyslipidemia: a case control study. Br J Dermatol. 2009; 161: 626-629.
8. Arias-Santiago S, Eisman AB, Fernandez JA, Giron-Prieto MS, Gutierrez-Salmeron MT, Mellado VG, et al. Cardiovascular risk factors in patients with lichen planus. Am J Med. 2011; 124: 543-548.
9. Manninen V, Tenkanen L, Koskinen P, Huttunen JK, Manttari M, Heinonen OP, et al. Joint effects of serum triglyceride and LDL cholesterol and HDL cholesterol concentrations on coronary heart disease risk in the Helsinki Heart Study. Implications for treatment. Circulation. 1992; 85: 37-45.
10. Wilson PW. Established risk factors and coronary artery disease: the Framingham Study. Am J Hypertens. 1994; 7: 7-12.
11. Imtiaz F, Shafique K, Mirza SS, Ayoob Z, Vart P, Rao S. Neutrophil lymphocyte ratio as a measure of systemic inflammation in prevalent chronic diseases in Asian population. Int Arch Med. 2012; 5: 2.
12. Bessman JD, Williams LJ, Gilmer Jr PR. Mean platelet volume. The inverse relation of platelet size and count in normal subjects, and an artifact of other particles. Am J Clin Pathol. 1981; 76: 289-293.
13. Jaremo P, Hansson G, Nilsson O. Elevated inflammatory parameters are associated with lower platelet density in acute myocardial infarctions with ST-elevation. Thromb Res. 2000; 100: 471-478.
14. Nunez J, Nunez E, Bodi V, Sanchis J, Minana G, Mainar L, et al. Usefulness of the neutrophil to lymphocyte ratio in predicting long-term mortality in ST segment elevation myocardial infarction. Am J Cardiol. 2008; 101: 747-752.
15. Seyhan M, Ozcan H, Sahin I, Bayram N, Karincaoglu Y. High prevalence of glucose metabolism disturbance in patients with lichen planus. Diabetes Res Clin Pract. 2007; 77: 198-202.
16. Ma C, Harskamp CT, Armstrong EJ, Armstrong AW. The association between psoriasis and dyslipidaemia: a systematic review. Br J Dermatol. 2013; 168: 486-495.
17. Saleh N, Samir N, Megahed H, Farid E. Homocysteine and other

cardiovascular risk factors in patients with lichen planus. J Eur Acad Dermatol Venereol. 2014; 28: 1507-1513.

18. Lee GK, Lee LC, Chong E, Lee CH, Teo SG, Chia BL, et al. The long-term predictive value of the neutrophil-to-lymphocyte ratio in Type 2 diabetic patients presenting with acute myocardial infarction. QJM. 2012; 105: 1075-1082.

19. Sen BB, Rifaioglu EN, Ekiz O, Inan MU, Sen T, Sen N. Neutrophil to lymphocyte ratio as a measure of systemic inflammation in psoriasis. Cutan Ocul Toxicol. 2014; 33: 223-227.

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