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Editorial Article

Psoriasis and Lichen Planus as Inflammatory Skin Diseases Mediated By T Cells: Possible Involvement of Cytotoxicity Mechanisms

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Psoriasis is inflammatory disease mediated by T lymphocytes that affects primary skin and joints [1]. Clinically it is characterized by well-demarcated scaly plaques mainly on extensor part of extremities, lumbosacral regions and scalp [2]. Patohistologically, plaques are defined by hyperkeratosis, acanthosis and parakeratosis of epidermis and T cell infiltration in dermis [3]. Apart from a fact that psoriasis is a skin disease, it could affect joints in form of psoriatic arthritis and could cause comorbidities such as cardiovascular disease, diabetes and depression [4].

Similarly, to psoriasis, lichen planus is a T cell mediated inflammatory disease clinically characterized by violaceus plaques on flexural parts of arms and lumbosacral regions as well as mucosa affections mainly on oral and genital mucosa [5]. Patohistologically, lichen planus lesions are labeled by hyperkeratosis and acanthosis of epidermis and band-like infiltration of T lymphocytes along the epidemodermal junction [6]. Apart from severe mucosal forms, disease is generally self-limiting in the period of two to three years [6].

Immunopathogenesis of psoriasis and lichen planus is quite similar [7,8]. In psoriasis, different agents such viruses, bacteria, systemic drugs or stress damage basal keratinocytes releasing mediators such as cathelicidin or LL-37 molecule, defensins and S-100 proteins [9,10]. LL-37 molecules bind to self-DNA that is also released from damaged keratinocytes and forms complexes [9,10]. Those complexes activate antigen presenting cells (APCs) mainly plasmacytoid DC to secrete IFN-alpha [9,10].

Furthermore, APC drain to regional lymph nodes where they present antigens to naive T lymphocytes that differentiate into Th1, Th17 and Th22 cells [10,11]. Migration of those cells back to skin and production of type 1, type 17 and type 22 cytokines are key stimuli for epidermal cell proliferation, accumulation of new inflammatory cells in dermis and epidermis and formation of psoriatic plaque [10].

In lichen planus lesions, different antigen stimuli especially viruses, drugs, or contact sensitivity antigen alter the basal keratinocytes releasing chemokines, such as CXCR3/CCL20/CCR6 that attract new lymphocytes into developing lesion [12,13]. Plasmacytoid DCs could amplify this effect by IFN-alpha production [13]. Activated APCs present antigen to T lymphocytes in regional lymph nodes and stimulate differentiation towards Th1 and Th17 direction [13]. However, in lichen planus activated basal keratinocytes present antigen that are bind to MHC class I molecule to CD8+ cytotoxic T cells [14]. Cytotoxic T cells express FasL or secrete perforin, granzyme B, granulysin or TNF-alpha that trigger basal keratinocyte apoptosis histopathologically seen as vacuolar degeneration of epidermis in form of Civatte bodies [15,16].

In psoriasis, the upregulation of cytotoxic molecules such as perforin, grenzyme B and FasL is demonstrated in peripheral blood of psoriasis patients and in psoriatic skin lesions, however the exact mode of action and target cells are still unknown [17,18].

Psoriasis and lichen planus are both T-cell mediated skin disease with very similar immunopathogenesis scenario. However, cytotoxicity mechanisms in lichen planus are well determined and undoubtedly play an important role while those mechanisms in psoriasis are still to be investigated in future.

References

1. Lima Ede A, Lima Mde A (2011) Reviewing concepts in the immunopathogenesis of psoriasis. An Bras Dermatol 86: 1151-1158.

2. Raut AS, Prabhu RH, Patravale VB (2013) Psoriasis clinical impli-

cations and treatment: a review. Crit Rev Ther Drug Carrier Syst 30 : 183-216.

3. Boehncke WH (2015) Etiology and Pathogenesis of Psoriasis. Rheum Dis Clin North Am 41: 665-75.

4. Davidovici BB, Sattar N, Jorg PC, Puig L et al. (2010) Psoriasis and systemic inflammatory diseases: potential mechanistic links between skin disease and co-morbid conditions. J Invest Dermatol 130: 1785-1796.

5. Sharma A, Białynicki-Birula R, Schwartz RA, Janniger CK (2012) Lichen planus: an update and review. Cutis 90 : 17-23.

6. Crincoli V, Di Bisceglie MB, Scivetti M, Lucchese A et al. (2011) Oral lichen planus: update on etiopathogenesis, diagnosis and treatment. Immunopharmacol Immunotoxicol 33: 11-20.

7. Wagner EF, Schonthaler HB, Guinea-Viniegra J, Tschachler E (2010) Psoriasis: what we have learned from mouse models. Nat Rev Rheumatol 6 : 704-714.

8. Wang H, Zhang D, Han Q et al. (2016) Role of distinct CD4+ T helper subset in pathogenesis of oral lichen planus. J Oral Pathol Med 45: 385-393.

9. Buchau AS, Gallo RL (2007) Innate immunity and antimicrobial defense systems in psoriasis. Clin Dermatol 25: 616-624.

10. Tonel G, Conrad C (2009) Interplay between keratinocytes and immune cells – Recent insights into psoriasis pathogenesis. Int J Bioch Cell Biol 41: 963-968.

11. Li X, Li J, Yang Y, Hou R, et al. (2013) Differential gene expression in peripheral blood T cells from patients with psoriasis, lichen planus, and atopic dermatitis. J Am Acad Dermatol 69: e235-e243.

12. Carrozzo M (2014) Understanding the pathobiology of oral lichen planus. Curr Oral Health Rep 1: 173-179.

13. Trucci VM, Salum FG, Figueiredo MA, Cherubini K (2013) Interrelationship of dendritic cells, type 1 interferon system, regulatory T cells and toll-like receptors and their role in lichen planus and lupus erythematosus- a literature review. Arch oral Biol 58: 1532-1540.

14. Lodi G, Scully C, Carrozzo M, Griffiths M, Sugerman PB et al. (2005) Current controversies in oral lichen planus: Report of an international consensus meeting. Part 1. Viral infections and etiopathogenesis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 100: 40-51.

15. Prpić Massari L, Kastelan M, Gruber F, Laskarin G, Sotosek Tokmadzić V et al. (2004) Perforin expression in peripheral blood lymphocytes and skin-infiltrating cells in patients with lichen planus. Br J Dermatol 151 : 433-9.

16. Kastelan M, Prpić Massari L, Gruber F, Zamolo G, Zauhar G et al. (2004) The role of perforin-mediated apoptosis in lichen planus lesions. Arch Dermatol Res 296 : 226-230.

17. Kastelan M, Prpić Massari L, Gruber F, Zamolo G, Zauhar G et al. (2004) Perforin expression is upregulated in the epidermis of psoriatic lesions. Br J Dermatol 151 : 831-836.

18. Prpić Massari L, Kastelan M, Laskarin G, Zamolo G, Massari D et al. (2007) Analysis of perforin expression in peripheral blood and lesions in severe and mild psoriasis. J Dermatol Sci 47 : 29-36.