# Role of Thymic Dysfunction in the Pathogenesis of Lichen Planus

**Sujoy Khan**

*Department of Immunology and Allergy, Hull University Teaching Hospitals NHS Trust, Queen's Center, Castle Hill Hospital, Cottingham, United Kingdom*

***Address for correspondence:***
*Dr. Sujoy Khan,*
*Department of Immunology and Allergy, Hull University Teaching Hospitals NHS Trust, Castle Hill Hospital, Castle Road, Cottingham, HU16 5JQ, United Kingdom.*
*E-mail: sujoykhan@gmail.com*

Dear Editor,

The review article on oral lichen planus (OLP) by Yashpal Manchanda and colleagues[1] was highly informative. However, examining OLP in patients with immune deficiencies could provide valuable insights into additional underlying pathways.

Several reports of lichen planus (LP) in patients with Good's syndrome (GS, thymoma with hypogammaglobulinemia), a primary immunodeficiency disorder characterized by B cells, either very low or absent with CD4+T cell lymphopenia[2,3] suggest the presence of B cells may not be critical in the development of LP. Good's syndrome is also characterized by opportunistic infections and multiple anti-cytokine autoantibodies.[4] As IL-17 and IL-22 play a protective role against fungal infections, the presence of autoantibodies against these cytokines naturally predispose patients to such infections. This mechanism may explain why oral candida can trigger OLP in not only in patients with GS but also in those with other autoimmune diseases. In the review by Le Gatt *et al.,*[2] it was found that in 74% of patients, the OLP was diagnosed before the diagnosis of GS, and less than one third of these patients reported improvement after undergoing thymectomy. Interestingly, the report by Torres-Valle *et al.*[5] (n = 9 GS) g had shown reduced Th1/Th17 cell populations through in-depth blood immune profiling. This study also identified additional cellular defects, including impaired memory T cells (both $CD4^+$ and $CD8^+$ cells) and myeloid dendritic cells, suggesting a combined immune defect involving both T- and B-cells.

The review referencing the study by Melo *et al.*[6] specifying the role of Th17, does not mention whether any of the patients had other autoimmune diseases, such as SLE, Sjogrens, psoriasis etc. Additionally, we cannot completely ignore the Th1 signature (IFN-γ, IL-21A, pSTAT1) that plays significant role in the inflammatory process.[7]

We are still in the early stage of understanding lymphocyte plasticity, but it is becoming clear that this plasticity influences the Th17 pathway and autoimmune networks in LP, with a relatively minor role of B cells in humoral immunity. Further research in gene defects in GS will be crucial for understanding how a dysfunctional thymus may contribute to the pathogenesis of the condition.

## *Financial support and sponsorship*

Nil.

## *Conflicts of interest*

There are no conflicts of interest.

## References

1. Manchanda Y, Rathi SK, Joshi A, Das S. Oral lichen planus: An updated review of etiopathogenesis, clinical presentation, and management. Indian Dermatol Online J 2023;15:8-23.
2. Le Gatt P, Nguyen AT, Baaroun V, Rochefort J. Oral lichen planus in patients with Good's syndrome: A literature review. Cureus. 2023;15:e35177.
3. Khan S, Campbell A, Hunt C, Sewell WA. Lichen planus in a case of Good's syndrome (thymoma and immunodeficiency). Interact Cardiovasc Thorac Surg 2009;9:345-6.
4. Burbelo PD, Browne SK, Sampaio EP, Giaccone G, Zaman R, Kristosturyan E, *et al*. Anti-cytokine autoantibodies are associated with opportunistic infection in patients with thymic neoplasia. Blood 2010;116:4848-58.
5. Torres-Valle A, Aragon L, Silva SL, Serrano C, Marcos M, Melero J, *et al*. In-depth blood immune profiling of Good syndrome patients. Front Immunol 2023;14:1285088.
6. Melo RA, Silva LAB, Fernades-Costa AN, Monteiro BVB, Silveira EJD, Miguel MCC. Participation of the Th17 response in the pathogenesis of oral lichen planus. J Eur Acad Dermatol Venereol 2018;32:e264-5.
7. Pietschke K, Holstein J, Meier K, Schäfer I, Müller-Hermelink E, Gonzalez-Menendez I, *et al*. The inflammation in cutaneous lichen planus is dominated by IFN-ϒ and IL-21-A basis for therapeutic JAK1 inhibition. Exp Dermatol 2021;30:262-70.



**Access this article online**

**Website:** https://journals.lww.com/idoj

**DOI:** 10.4103/idoj.idoj_173_24

Quick Response Code

**How to cite this article:** Khan S. Role of thymic dysfunction in the pathogenesis of lichen planus. Indian Dermatol Online J 2024;15:1055.

**Received:** 26-Feb-2024. **Revised:** 04-May-2024.
**Accepted:** 17-Aug-2024. **Published:** 28-Oct-2024.