Original

Lichen planus autoimmune comorbidities: A retrospective case-control study

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Abstract

Purpose: To assess whether patients with lichen planus (LP) show increased prevalence and odds of autoimmune and viral comorbidities compared with matched controls using real-world data. **Methods:** We analyzed TriNetX Linked Network electronic health records and claims data from 2016 to 2024 (more than 112 million patients). LP was defined by at least 2 International Classification of Diseases, Tenth Revision, Clinical Modification codes (excluding drug-induced variants) and matched 1:1 to controls on age, sex, and race. We calculated odds ratios (ORs), 95% confidence intervals, prevalence, and absolute risk differences (ARDs). **Results:** The matched cohort included 43 458 LP and control patients (mean age, 53.8 years; 64.4% female). LP was associated with higher prevalence of autoimmune conditions, including psoriasis (6.59% versus 2.54%; ARD, 4.05%; OR, 2.71), lupus erythematosus (2.87% versus 1.13%; ARD, 1.74%; OR, 2.58), and autoimmune thyroiditis (3.09% versus 1.61%; ARD, 1.48%; OR, 1.95). Associations were also seen for Sjögren syndrome, alopecia areata, localized scleroderma, and vitiligo. Hepatitis C virus was more common in LP (2.95% versus 2.03%; ARD, 0.92%) but with a lower OR (1.47). **Conclusion:** LP was associated with substantially higher odds and prevalence of several autoimmune comorbidities, exceeding those for viral infections such as hepatitis C. These findings inform LP pathogenesis and comorbidity patterns but do not support changes to clinical management.

Introduction

Lichen planus (LP) is a rare, chronic, T cell–mediated inflammatory disorder that affects the skin and mucous membranes.[1] LP has been linked to several systemic conditions, including autoimmune diseases and viral infections,[2] but these associations remain incompletely defined. Most prior studies have been limited by small sample sizes, single-center designs, or homogenous populations, leaving uncertainty about the broader comorbidity profile of LP. Using the TriNetX database, which aggregates real-world data from a large and diverse patient population, we sought to determine whether patients with LP have an increased risk of autoimmune comorbidities compared with age-, sex-, and race-matched controls.

Methods

We used data from the TriNetX Linked Network, which integrates electronic health records and claims data from more than 112 million patients, to evaluate comorbidity risk among individuals with LP from July 1, 2016, to December 9, 2024. LP was defined by the presence of at least 2 International Classification of Diseases, Tenth Revision, Clinical Modification codes for LP (L43), excluding code L43.2 (lichenoid drug reaction). Cases were matched 1:1 to controls using propensity score matching on age, sex, and race via a nearest-neighbor algorithm. For each comorbidity, we calculated odds ratios (ORs) with 95% confidence intervals, absolute prevalence, and absolute risk differences (ARDs) to facilitate clinical interpretation.

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Table 1. *Odds of Comorbidities in LP Versus Matched Controls.*

Diagnosis	ICD-10 Code(s)	LP Prevalence, %	Control Prevalence, %	ARD	OR (95% CI)	*P* Value
Psoriasis	L40, L40.9	6.59	2.54	4.05	2.710 (2.524–2.909)	< .0001
Lupus erythematosus	L93, L93.0, L93.2, M32, M32.9	2.87	1.13	1.74	2.583 (2.325–2.870)	< .0001
Autoimmune thyroiditis	E06.3	3.09	1.61	1.48	1.949 (1.777–2.138)	< .0001
Sjögren syndrome	M35.0, M35.00	2.41	0.96	1.45	2.542 (2.268–2.849)	< .0001
Alopecia areata	L63, L63.9	1.74	0.45	1.29	3.948 (3.370–4.625)	< .0001
Localized scleroderma (morphea)	L94.0	1.10	0.17	0.93	6.520 (5.102–8.332)	< .0001
HCV infection	B17.1, B19.2, B18.2, B19.2, B18.2	2.95	2.03	0.92	1.467 (1.345–1.600)	< .0001
Vitiligo	L80	0.97	0.26	0.71	3.829 (3.106–4.722)	< .0001
Autoimmune hepatitis	K75.4	0.25	0.11	0.14	2.232 (1.588–3.138)	3.82×10^{-6}

Abbreviations: ARD, absolute risk difference; CI, confidence interval; HCV, hepatitis C virus; ICD-10, International Classification of Diseases, Tenth Revision, Clinical Modification; LP, lichen planus; OR, odds ratio.

Results

A total of 44 352 patients with LP and 8 393 177 patients without LP were identified. After 1:1 propensity score matching, 43 458 patients were included in each cohort. The mean age was 53.8 ± 15.7 years; most participants were female (64.4%) and non-Hispanic White (61.4%). Comorbidities most strongly associated with LP, based on ORs, absolute prevalence, and ARDs, are summarized in Table 1.

Patients with LP demonstrated higher prevalence and odds of multiple autoimmune conditions compared with matched controls. The most common autoimmune comorbidities in the LP cohort were psoriasis (6.59% versus 2.54%; ARD, 4.05%), lupus erythematosus (2.87% versus 1.13%; ARD, 1.74%), and autoimmune thyroiditis (3.09% versus 1.61%; ARD, 1.48%). Additional autoimmune diseases showing notable associations included Sjögren syndrome (2.41% versus 0.96%; ARD, 1.45%), alopecia areata (1.74% versus 0.45%; ARD, 1.29%), and localized scleroderma (1.10% versus 0.17%; ARD, 0.93%).

Hepatitis C virus (HCV) infection was also more prevalent in LP patients (2.95% versus 2.03%; ARD, 0.92%); however, the absolute difference and OR were lower relative to autoimmune conditions (OR, 1.47 [1.35–1.60]). Less common yet positively associated conditions included vitiligo (0.97% versus 0.26%; ARD, 0.71%) and autoimmune hepatitis (0.25% versus 0.11%; ARD, 0.14%).

Discussion

In this large, matched cohort study, patients with LP demonstrated a higher prevalence of several autoimmune comorbidities compared with the general population. The most common associations included psoriasis (6.59% versus 2.54%), lupus erythematosus (2.87% versus 1.13%), and autoimmune thyroiditis (3.09% versus 1.61%). Overall, autoimmune conditions exhibited larger ORs and ARDs than HCV infection, a comorbidity historically emphasized in the LP literature.

These findings are consistent with prior retrospective studies that have reported elevated autoimmune disease burden among patients with LP. A United States retrospective case-control study found increased odds of autoimmune comorbidities overall (OR, 2.88; $P < .001$), including autoimmune thyroiditis (OR, 3.42; $P < .001$) and systemic lupus erythematosus (OR, 3.26; $P < .001$).[3] Our effect sizes for these conditions (ORs of 1.95 and 2.58, respectively) are directionally similar, though lower, which may reflect differences in population size, coding algorithms, or comorbidity definitions. Likewise, a German claims analysis reported markedly elevated odds of alopecia areata (OR, 7.8; $P < .001$) and psoriasis vulgaris (OR, 2.7; $P < .001$).[4] We similarly observed positive associations—alopecia areata (OR, 3.95) and psoriasis (OR, 2.71)—though with smaller magnitudes, again suggesting potential variability introduced by diagnostic coding practices or healthcare utilization patterns.

Notably, the German study reported a stronger association with HCV infection (OR, 3.00) than observed in our cohort (OR, 1.47).[4] This difference may reflect regional variation in HCV prevalence, differences in screening practices, or population-level risk factors.[5] Such discrepancies underscore the importance of contextualizing odds ratios, which are influenced by background prevalence, health system characteristics, and patterns of diagnostic testing.

This study has several limitations. We were unable to determine the specialty of diagnosing clinicians, which raises the possibility of diagnostic misclassification. Additionally, the geographic and institutional composition of the TriNetX network may not fully capture all patient populations, introducing potential selection bias. Because LP patients are more likely to be evaluated by dermatologists than other specialists, dermatologic comorbidities may be identified more readily than conditions involving other organ systems.

Conclusion

Patients with LP exhibit a higher prevalence of autoimmune comorbidities compared with matched controls, as demonstrated by both relative and absolute risk measures. These findings indicate that autoimmune conditions may co-occur with LP more frequently than previously recognized. Although the results highlight potential shared pathogenic pathways, they do not imply causality and do not support changes to current clinical management. Further research is warranted to clarify underlying mechanisms and to determine whether specific subgroups of LP patients may benefit from targeted comorbidity screening.

Potential conflicts of interest

The authors report no conflicts of interest. This work was supported by the CTSA Program Grant (award number UL1TR003163).

Author contributions

Grant funding was secured by L.M. and R.D. The study was designed by A.L.R., A.W., L.D., and B.B. The table creation was performed by A.M. The data analysis was conducted by A.L.R., D.R., M.M., J.M., and K.N. A.L.R. drafted the initial manuscript, and all authors contributed to manuscript revision and approved the final version for submission.

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