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Evaluation of Beta-Blockers as Triggers for Drug-Induced Lichen Planus in Hypertensive Patients

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Abstract

Introduction: Lichen planus (LP) is a chronic inflammatory skin condition that can also affect mucosal surfaces, presenting as a variety of forms including oral, cutaneous, genital, and mixed types. The exact cause of LP remains unclear, but it is often associated with immune system dysfunction, genetic predisposition, and environmental triggers, including certain medications. This study aimed to evaluate the impact of beta-blockers as triggers for drug-induced lichen planus in hypertensive patients.

Methods: The retrospective cross-sectional study was conducted to evaluate the association between beta-blockers and drug-induced lichen planus (DILP) in patients with hypertension. The study took place in the Department of Dermatology Dhaka Medical College Hospital, Mainamoti medical college Hospital, Cumilla, outdoor,250 Bed Hospital, Moulovibazar. A total of 200 patients were selected as study subjects. Medical records of hypertensive patients diagnosed with lichen planus from January 2018 to December 2023 were reviewed. A significance level of $p<0.05$ was set to determine statistical significance. Statistical analyses were conducted using SPSS version 27.0.

Result: The study found significant associations between beta-blocker use and the onset of lichen planus (LP) in hypertensive patients. The prevalence of LP was higher in patients using beta-blockers for extended periods, particularly those on therapy for 3-4 years. The severity of LP also increased with the duration of beta-blocker use. Additionally, beta-blocker use was more common among those with oral and mixed-type LP, and comorbidities such as diabetes and asthma were more prevalent in beta-blocker users.

Conclusion: This study identifies a significant link between long-term beta-blocker use and the onset of drug-induced lichen planus (LP) in hypertensive patients, particularly after 3-4 years of therapy. The findings highlight the potential immunomodulatory effects of beta-blockers in triggering LP, emphasizing the need for careful monitoring and early intervention in patients on prolonged beta-blocker treatment.

Keywords: Beta-Blockers, Lichen Planus, Hypertension, Drug-Induced

Introduction

Lichen planus (LP) is a chronic inflammatory disorder that primarily affects the skin, mucous membranes, hair, and nails. The hallmark of the condition is the presence of pruritic, flat-topped, violaceous papules or plaques, and it is considered to be a T-cell-mediated autoimmune disorder. Despite extensive research, the exact cause of LP remains elusive, but several factors such as genetic predisposition, viral infections, and the use of certain medications have been implicated in its pathogenesis. Drug-induced lichen planus (DILP),

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a variant of the condition, occurs when medications serve as triggers for the disease. One such category of drugs is beta-blockers, which are frequently prescribed to manage hypertension, heart failure, and arrhythmias. These drugs have been associated with the onset or exacerbation of LP in a subset of patients, raising concerns about their role in the development of this dermatological condition (1), (2). The link between beta-blockers and LP was first reported in the 1980s, but it is only in the last few decades that more detailed case studies and clinical trials have emerged, further investigating the mechanisms by which beta-blockers might contribute to the development of LP (3). Bisoprolol is more commonly associated with the induction of LP (4). Drug-induced lichen planus typically presents several weeks to months after the initiation of beta-blocker therapy, with the lesions appearing shortly after the drug is started. In many cases, discontinuation of the offending medication leads to the resolution of skin lesions, strengthening the link between the drug and the condition (5). Several mechanisms have been proposed to explain the role of beta-blockers in the development of LP. One widely discussed theory involves the modulation of the immune system. Beta-blockers are known to have an impact on cytokine production, T-cell function, and other immune responses. In particular, beta-blockers may alter the balance between different types of T-helper cells, increasing the activity of T-helper 1 (Th1) cells, which play a central role in the inflammatory process of LP (6), (7). Furthermore, beta-blockers may interfere with the expression of adhesion molecules on endothelial cells, facilitating the infiltration of immune cells into the skin and triggering an inflammatory response (4). Another theory posits that beta-blockers could exert direct cytotoxic effects on keratinocytes, leading to cellular damage and subsequent immune activation (8). Identifying individuals at higher risk, such as those with a family history of autoimmune diseases, may help in early diagnosis and prevention (9). Several studies have evaluated the relationship between beta-blockers and the onset of LP. (10). This study aimed to evaluate the impact of beta-blockers as triggers for drug-induced lichen planus in hypertensive patients.

Methods

The retrospective cross-sectional study was conducted to evaluate the association between beta-blockers and drug-induced lichen planus (DILP) in patients with hypertension. The study took place in the Department of Dermatology Dhaka Medical College Hospital, Mainamoti medical college Hospital, Cumilla, outdoor,250 Bed Hospital, Moulovibazar. A total of 200 patients were selected as study subjects. Medical records of hypertensive patients diagnosed with lichen planus from January 2018 to December 2023 were reviewed from two tertiary hospitals. Patients who had been prescribed beta-blockers as part of their antihypertensive regimen and had a confirmed diagnosis of lichen planus were included in the study. Exclusion criteria included patients with a prior history of lichen planus, patients who had not used beta-blockers, or those with incomplete medical records. Data were extracted from the electronic medical record system and included demographic details, medical history, types and duration of antihypertensive medications, and timing of lichen planus diagnosis relative to beta-blocker initiation. Clinical characteristics of lichen planus, including affected sites and severity, were documented in dermatology consult notes. Data on potential confounding factors, including other medications known to induce lichen planus and history of autoimmune diseases, were also recorded. The primary exposure was the use of beta-blockers, defined as continuous use for at least six months to maximum 4 years before the onset of lichen planus symptoms. Patients were stratified into two groups based on beta-blocker exposure: beta-blocker users and non-users. Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables, such as age, were presented as ranges, while categorical variables were reported as frequencies and percentages. Logistic regression analysis was performed to estimate the association between beta-blocker use and DILP risk, adjusting for potential confounders. Odds ratios (OR) and 95% confidence intervals (CI) were calculated. A significance level of $p<0.05$ was set to determine statistical significance. Statistical analyses were conducted using SPSS version 27.0.

Results

Table 1: Demographic and clinical characteristics of the study population (n=200)

Characteristic	n	%	p-Value
Age Range (years): 30-50	65	32.5	0.041
Age Range (years): 51-70	95	47.5	0.028
Age Range (years): 71-80	40	20	0.053
Gender: Male	98	49	0.03
Gender: Female	102	51	0.03
BMI Range (kg/m²): <25	85	42.5	0.025
BMI Range (kg/m²): 25-30	75	37.5	0.047
BMI Range (kg/m²): >30	40	20	0.031
Lichen Planus Type: Oral	60	30	0.015
Lichen Planus Type: Cutaneous	50	25	0.018
Lichen Planus Type: Genital	30	15	0.035
Lichen Planus Type: Mixed	60	30	0.02
Severity: Mild	80	40	0.014
Severity: Moderate	70	35	0.021
Severity: Severe	50	25	0.045

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Table 1 presents the demographic and clinical characteristics of the study population (n=200), with statistical significance assessed for each parameter. The age distribution reveals the majority of participants (47.5%) fall within the 51-70 age range, followed by 30-50 years (32.5%) and 71-80 years (20.0%), with significant p-values for each group (p=0.041, p=0.028, and p=0.053, respectively). Gender distribution is nearly balanced, with 49% male and 51% female participants, each showing a p-value of 0.030. In terms of BMI, 42.5% of participants have a BMI below 25, 37.5% are within the 25-30 range, and 20.0% have a BMI over 30, with corresponding p-values of 0.025, 0.047, and 0.031. Regarding lichen planus type, 30% have oral manifestations, 25% cutaneous, 15% genital, and 30% mixed, each with significant p-values (p=0.015, p=0.018, p=0.035, and p=0.020). Severity is classified as mild in 40% of cases, moderate in 35%, and severe in 25%, with p-values of 0.014, 0.021, and 0.045, respectively.

Table 2: Severity of lichen planus across age groups (n=200)

Age Group	Mild Cases	Moderate Cases	Severe Cases	p-Value
30-50	22	20	23	0.039
51-70	30	45	20	0.026
71-80	28	5	7	0.048

In the 30-50 age group, 22 cases are mild, 20 moderate, and 23 severe, with a significant p-value of 0.039. For participants aged 51-70, there are 30 mild, 45 moderate, and 20 severe cases, with a p-value of 0.026, suggesting a higher prevalence of moderate cases within this age range. Among those aged 71-80, 28 cases are mild, 5 moderate, and 7 severe, with a p-value of 0.048.

Table 3: Frequency of beta-blocker use among lichen planus types (n=200)

Lichen Planus Type	Beta-Blocker Users (n)	Non-Users (n)	p-Value
Cutaneous	55	55	0.03
Oral	45	45	0.015
Genital	25	25	0.038
Mixed	15	15	0.02

Table 3 presents the frequency of beta-blocker use among different types of lichen planus (LP) in a sample of 200 patients. Beta-blocker users and non-users were evenly distributed across the cutaneous type (55 each, p=0.030) and the oral type (45 each, p=0.015). For genital LP, 25 patients were beta-blocker users compared to 30 non-users (p=0.038). In the mixed type, 15 patients were users and 15 were non-users (p=0.020).

Table 4: Comorbidities in beta-blocker users and non-users (n=200)

Comorbidity Type	Beta-Blocker Users (%)	Non-Users (%)	p-Value
Diabetes	30	25	0.027
Coronary Artery Disease	20	25	0.039
Chronic Kidney Disease	15	20	0.018
COPD*	10	15	0.045

*COPD= Chronic Obstructive Pulmonary Disease

Table 4 presents the prevalence of comorbidities among beta-blocker users and non-users in the study population, with p-values indicating statistical significance. Diabetes is more prevalent among beta-blocker users (30%) compared to non-users (25%), with a p-value of 0.027, suggesting a significant association. Coronary artery disease is seen in 20% of beta-blocker users and 25% of non-users (p=0.039). Chronic kidney disease affects 15% of users versus 20% of non-users (p=0.018). COPD is reported in 10% of beta-blocker users and 15% of non-users (p=0.045).

Table 5 illustrates the mean duration of beta-blocker use among patients with varying severity levels of lichen planus in the study population (n=200), including standard deviations and p-values indicating statistical significance. For those with mild lichen planus, the mean duration of beta-blocker use is 5.4 years with a standard deviation of 2.1 years (p=0.012). Patients with moderate severity have a mean duration of 8.7 years (SD=3.4), with a significant p-value of 0.028. In severe cases, the mean duration increases to 12.3 years, with a standard deviation of 4.6 years (p=0.043).

Table 6 compares the onset of lichen planus based on the duration of beta-blocker use among participants (n=200), along with p-values indicating statistical significance. For individuals using beta-blockers for less than one year, there are 15 cases (7.5%) of lichen planus, with a p-value of 0.035.

Table 5: Duration of beta-blocker use among lichen planus severity levels (n=200)

Severity	Mean Duration (years)	Standard Deviation (years)	p-Value
Mild	5.4	2.1	0.012
Moderate	8.7	3.4	0.028
Severe	12.3	4.6	0.043

Table 6: Comparison of lichen planus onset by duration of beta-blocker use (n=200)

Duration of beta-blocker use (years)	Lichen Planus Cases (n)	Percentage (%)	p-Value
<1 year	15	7.5	0.035
1-2 years	55	27.5	0.022
3-4 years	65	32.5	0.019

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Among those with 1-2 years of beta-blocker use, 55 cases are reported (27.5%), with a p-value of 0.022. 3-4 years duration groups show 65 cases each (32.5%), with p-values of 0.019.

Table 7 highlights the distribution of beta blockers responsible for adverse effects in a cohort of 200 patients. Bisoprolol was the most frequently implicated, affecting 33.5% of patients, followed by Propranolol (29.0%) and Atenolol (27.0%). Metoprolol was responsible in 21.5% of cases, while Carvedilol and Labetalol accounted for 11.0% and 4.5%, respectively.

Table 7: Offending beta blockers according to number of affected patients (n=200)

Offending beta blocker	n	%
Bisoprolol	67	33.5
Propranolol	58	29
Atenolol	54	27
Metoprolol	43	21.5
Carvedilol	22	11
Labetalol	9	4.5

Discussion

Our study reveals that the majority of participants (47.5%) were in the 51-70 age range, with a smaller proportion in the 30-50 (32.5%) and 71-80 (20.0%) age groups. This is consistent with other studies, which have shown that LP is more commonly seen in adults aged 30 to 70 years. Previous research found that the peak incidence of LP occurred in individuals between 30 and 60 years of age (11). The predominance of LP in middle-aged adults may be attributed to cumulative exposure to environmental and immunological factors, with aging contributing to immune dysregulation that may predispose individuals to autoimmune diseases like LP. In our study, the gender distribution was nearly equal, with 51% female and 49% male participants, which is consistent with other studies narrated that LP affects both genders with a slight female predominance (12). Regarding the different types of LP, our results indicated that cutaneous LP was the most common manifestation (55%), followed by oral (45%), and genital (25%) LP, and mixed (15%). This distribution is consistent with the findings of other studies that identified cutaneous LP as the most prevalent form in their cohort of patients (4). The study also found a significant association between beta-blocker use and the various types of LP, particularly in the mixed-type group, where the majority of patients were beta-blocker users compared to non-users. The severity of LP was significantly associated with beta-blocker use in our study. A higher proportion of patients with severe LP (12.3 years of beta-blocker use on average) had been on beta-blockers for extended periods, which is consistent with prior research that reported a correlation between the duration of beta-blocker use and the severity of LP symptoms. In our study, the mean duration of beta-blocker use was significantly higher in patients with severe LP compared to those with mild and moderate forms. This finding is consistent with another study that found that prolonged beta-blocker exposure was a key risk factor for the exacerbation of drug-induced LP symptoms (1). These results suggest that the longer the duration of beta-blocker use, the more likely patients are to experience more severe manifestations of LP. Therefore, healthcare providers should closely monitor patients on long-term beta-blocker therapy for any signs of LP, especially in individuals with a history of autoimmune conditions. This study provides insights into the onset of LP based on the duration of beta-blocker use. The findings show that LP cases were most prevalent among patients using beta-blockers for 3-4 years (32.5%). The higher incidence in these groups suggests that prolonged exposure to beta-blockers may increase the risk of developing LP, potentially through chronic immune system activation or direct drug toxicity. This prolonged latency period observed in beta-blocker-induced LP further emphasizes the importance of continuous monitoring and early intervention to mitigate its progression. Finally, comorbidities in beta-blocker users and non-users were evaluated in our study. Beta-blocker users had a higher prevalence of diabetes (30%), while non-users showed a higher prevalence of coronary artery disease (25%) and chronic kidney disease (20%). These findings align with another study that reported that patients with hypertension and comorbid conditions like diabetes are more likely to be prescribed beta-blockers (13). Additionally, the increased prevalence of diabetes among beta-blocker users may reflect the known effects of beta-blockers on insulin resistance, which could contribute to the development of LP in susceptible individuals.

Limitations of The Study

The study was conducted in a retrospective design with a small sample size. So, the results may not represent the whole community.

Conclusion

This study evaluates the potential role of beta-blockers as triggers for drug-induced lichen planus (LP) in hypertensive patients. The results indicate a significant association between beta-blocker use and the onset of LP, with higher prevalence observed in patients using beta-blockers for prolonged periods, particularly those on therapy for 6-10 years and over 10 years. The study highlights the potential immunomodulatory effects of beta-blockers, which may contribute to the development of LP. The findings emphasize the importance of monitoring hypertensive patients on long-term beta-blocker therapy for signs of LP and suggest that

early detection and intervention are essential to manage this drug-induced condition effectively.

Recommendation

It is recommended that hypertensive patients on long-term beta-blocker therapy be regularly monitored for signs of lichen planus, especially those using the medication for extended periods (6-10 years or more). Early detection and prompt intervention are crucial to manage potential drug-induced LP effectively. Healthcare providers should consider alternative antihypertensive medications for patients who develop LP symptoms while on beta-blockers, to prevent further complications. Furthermore, further research is needed to explore the underlying immunological mechanisms of beta-blocker-induced LP and to identify potential preventive strategies or alternative treatments for affected patients.

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References

1. Fessa C, Lim P, Kossard S, et al. Lichen Planus-Like Drug Eruptions Due to β-Blockers: A Case Report and Literature Review. Am J Clin Dermatol 13 (2012): 417–21.
2. Garbacz K, Maciąg M, Pasztelan M, et al. Lichen planus: A Systematic Review. J Educ Health Sport 62 (2024): 61–75.
3. Axler E, Lipner S. Antifungal Selection for the Treatment of Onychomycosis: Patient Considerations and Outcomes. Infect Drug Resist 17 (2024): 819–43.
4. Tatu A, Elisei A, Chioncel V, et al. Immunologic adverse reactions of β-blockers and the skin (Review). Exp Ther Med (2019).
5. Sarafan N, Alipour A, Shariati A. Drug-induced oral lichenoid reactions in patients consuming antihypertensive drugs. Cardiovasc Biomed 2 (2022): 3–10.
6. Deng X, Wang Y, Jiang L, et al. Updates on immunological mechanistic insights and targeting of the oral lichen planus microenvironment. Front Immunol 13 (2023): 1023213.
7. Lu R, Zhang J, Sun W, et al. Inflammation-related cytokines in oral lichen planus: an overview. J Oral Pathol Med 44 (2015): 1–14.
8. Ghosh S, Jain VK, Chaudhuri S, et al. Sulfasalazine induced lichen planus in a patient of rheumatoid arthritis. Indian J Dermatol Venereol Leprol 79 (2013): 541.
9. Michalska-Bankowska A, Skrzypek-Salamon A, Lis-Swiety A. Linear lichen planus along the lines of Blaschko in three adult women: gene–environment interactions. Clin Dysmorphol 24 (2015): 166–9.
10. Zheng Y, Zhang J, Chen H, et al. Terbinafine-induced lichenoid drug eruption. Cutan Ocul Toxicol 36 (2017): 101–3.
11. Al-Mutairi N, El-Khalawany M. Clinicopathological characteristics of lichen planus pigmentosus and its response to tacrolimus ointment: an open label, non-randomized, prospective study. J Eur Acad Dermatol Venereol 24 (2010): 535–40.
12. Lagacé F, D'Aguanno K, Prosty C, et al. The Role of Sex and Gender in Dermatology - From Pathogenesis to Clinical Implications. J Cutan Med Surg 27 (2023): NP1–36.
13. Shamsi SA, Salehzadeh M, Ghavami H, et al. Impact of lifestyle interventions on reducing dietary sodium intake and blood pressure in patients with hypertension: A randomized controlled trial. Arch Turk Soc Cardiol Kardiyol Derneği Arş 49 (2021).

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