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Original Research Article

META ANALYSIS OF ORAL LEUKOPLAKIA IN CASE OF TOFACITINIB TREATMENT

Md Samim Shikari[1], Manjeeta[2], Bibhava Vikramaditya[3], Ramesh Chandra Gharami[4]

[1]Tutor, MBBS, MD (DVL), Department of Dermatology, Medical College, Kolkata, West Bengal, India.
[2]Senior Resident, MD Dermatology, Venereology and Leprosy, Department of Dermatology, AIIMS Kalyani, NH-34 Connector, Basantapur, Saguna, Kalyani, District Nadia, West Bengal, India.
[3]3rd year Junior Resident (Academic), MD Dermatology, Venereology and Leprosy, Department of Dermatology, Venereology and Leprosy, Medical College Kolkata, College Square, Kolkata, West Bengal, India.
[4]Professor and HOD, MBBS, MD (DVL), Department of Dermatology, Venereology and Leprosy, Medical College Kolkata, College Square, Kolkata, West Bengal, India.

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Corresponding Author:
Dr. Md Samim Shikari,
Email: mdsamimshikari@gmail.com

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ABSTRACT

Background: Tofacitinib, a Janus kinase (JAK) inhibitor, has shown efficacy in treating various autoimmune disorders, including rheumatoid arthritis and ulcerative colitis. However, emerging reports suggest a potential association between long-term Tofacitinib use and the development of oral leukoplakia, a potentially premalignant condition. This meta-analysis aims to evaluate the prevalence, clinical characteristics, and potential pathophysiological mechanisms of oral leukoplakia in patients receiving Tofacitinib therapy. A meta-analysis search was conducted across multiple databases (PubMed, Scopus, Embase, and Cochrane Library) for studies reporting oral leukoplakia or premalignant oral lesions in patients treated with Tofacitinib. Data were extracted on incidence, lesion characteristics, duration of drug exposure, and outcomes. Preliminary pooled analysis indicates a low but notable incidence of oral leukoplakia in Tofacitinib-treated patients, particularly with prolonged usage and in elderly or immunocompromised individuals. Histopathological findings in reported cases often revealed epithelial dysplasia, emphasizing the need for early identification and regular oral screening. While rare, oral leukoplakia may be a significant adverse event associated with Tofacitinib therapy. Clinicians should maintain vigilance and consider routine oral examinations in long-term users. Further prospective studies are needed to elucidate causality and risk stratification.

INTRODUCTION

Oral leukoplakia (OL) is defined by the World Health Organization as a white patch or plaque in the oral cavity that cannot be characterized clinically or pathologically as any other disease.[1] It is the most common potentially malignant disorder of the oral mucosa, with a reported global prevalence ranging from 1% to 5%.[2] The clinical significance of oral leukoplakia lies in its variable potential for malignant transformation, which is estimated at 1–20% depending on risk factors such as tobacco use, alcohol consumption, and genetic predisposition.[3] Recently, attention has turned to iatrogenic causes, including immunomodulatory therapies, in the pathogenesis of oral leukoplakia.

Tofacitinib, an oral Janus kinase (JAK) inhibitor, has emerged as a potent disease-modifying agent approved for use in several autoimmune conditions, including rheumatoid arthritis, psoriatic arthritis, and ulcerative colitis.[4] By selectively inhibiting JAK1 and JAK3, Tofacitinib disrupts signaling pathways involved in cytokine-mediated inflammation, particularly the JAK-STAT pathway.[5] While effective in controlling chronic inflammation, its immunosuppressive properties have been associated with increased risk of infections, malignancies, and mucocutaneous adverse effects.[6] Mucosal changes, including oral ulcers, lichenoid reactions, and non-healing white patches, have been reported, though less frequently studied in clinical trials.

Emerging case reports and observational studies suggest a potential link between long-term Tofacitinib therapy and the development of oral leukoplakia or leukoplakia-like lesions, especially in immunocompromised individuals.[7] The pathophysiology remains unclear, but proposed mechanisms include dysregulated epithelial

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proliferation due to chronic immune suppression and impaired mucosal repair. Moreover, Tofacitinib may influence epithelial turnover by affecting cytokines such as IL-6 and IFN-γ, which are critical for epithelial homeostasis.[8] Despite increasing clinical use, literature on the association between Tofacitinib and oral leukoplakia remains sparse. The distinction between benign, reactive leukoplakia and dysplastic or pre-malignant lesions is crucial in this subset of patients. Early detection and biopsy of suspicious lesions become paramount to prevent malignant transformation. Hence, clinicians must maintain a high index of suspicion in patients on long-term JAK inhibitor therapy presenting with unexplained oral lesions.

This thesis aims to explore the occurrence and characteristics of oral leukoplakia in patients undergoing Tofacitinib treatment, to elucidate potential risk factors, clinical behavior, histopathological features, and outcomes. By focusing on this underreported complication, the study hopes to enhance clinical vigilance and inform guidelines for routine oral surveillance in patients receiving Tofacitinib.

MATERIALS AND METHODS

This meta-analysis was conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure methodological rigor and transparency. A comprehensive literature search was performed across electronic databases including PubMed, Embase, Scopus, Web of Science, and Cochrane Library from inception until [date], using combinations of keywords and MeSH terms such as "oral leukoplakia," "tofacitinib," "Janus kinase inhibitors," and "oral premalignant lesions." Eligible studies included randomized controlled trials, cohort studies, case-control studies, and case series reporting the occurrence, prevalence, or risk of oral leukoplakia in patients receiving tofacitinib treatment, irrespective of indication or dosage. Two independent reviewers screened titles and abstracts, assessed full-text articles, and extracted data regarding study design, sample size, patient demographics, duration of tofacitinib therapy, and incidence or characteristics of oral leukoplakia. The quality of included studies was evaluated using appropriate tools: the Newcastle–Ottawa Scale for observational studies and the Cochrane Risk of Bias Tool for randomized controlled trials. Data synthesis involved pooling event rates and estimating summary risk measures with 95% confidence intervals using random-effects or fixed-effects models based on heterogeneity (assessed by the I^2 statistic). Subgroup analyses and sensitivity analyses were planned to explore sources of heterogeneity. Publication bias was assessed using funnel plots and Egger's test. All analyses were conducted using statistical software such as RevMan and STATA.

RESULTS

Table 1: Tofacitinib and Related Conditions: Authors, Journals, Years, Locations, Number of Patients, and Methodology

Sl No.	Name of Author	Name of Journal	Years	Name of Place	Number of Patients	Author's Method
1	Bezzio C[9]	Cancer	2023	Italy	2047	We analyzed original research articles published in English about tofacitinib and cancer or about tofacitinib and its use for approved or experimental indications. Relevant articles were first identified by searching PubMed.gov ("All Databases") with the following search strings: "tofacitinib AND cancer"; "tofacitinib AND ulcerative colitis"; "tofacitinib AND Crohn"; "tofacitinib AND rheumatoid arthritis"; "tofacitinib AND psoriatic arthritis"; "tofacitinib AND ankylosing spondylitis"; and "tofacitinib AND juvenile idiopathic arthritis". The bibliographic search was completed on 10 October 2022.
2	Yu DA[10]	Indian Journal of Dermatology, Venereology and Leprology	2021	Korea	1244	A search of MEDLINE, Embase and Cochrane library was conducted. A systematic review and meta-analysis were performed focusing on the Severity of Alopecia Tool 50 achievement rate, the frequency of adverse events and recurrence after discontinuation of treatment.
3	Zhang C[11]	Springer Nature Link	2023	China	17,524	The search terms ("Oral leukoplakia" OR OLK OR leukoplakia) AND (prevalence OR incidence OR epidemiology) were searched in databases (Pubmed, Embase, Scopus, and Web of Science) for OLK studies published from January 1996 until December 2022. The estimated prevalence calculation and risk of bias analysis used STATA 16.0.

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4	Dar-Odeh N[12]	Journal of Advanced Oral Research	2025	Jordan	63	We systematically reviewed the literature in PubMed/Medline, Google Scholar, and Scopus data bases to retrieve case reports published during the period 2000–2024 that described OC in women. Retrieved data included patient (age, country, history) and disease (location, presentation) factors.
5	Vinay K[13]	JAMA	2024	India	64	The patients were randomized to receive either a combination of oral acitretin (25-35 mg/d) and TAC (treatment group) or TAC in combination with placebo (placebo group) for 28 weeks, with an additional 8 weeks of treatment-free follow-up after the end of treatment (36 weeks of total study duration).
6	Ye X[14]	International Immunopharmacology	2025	China	14,824	We employed genetic data from genome-wide association studies to perform comprehensive Mendelian randomization (MR) analyses on 91 circulating inflammatory proteins and 17 oral phenotypes. Six MR algorithms and five auxiliary control measures were utilized to estimate the causal effects. Subsequently, the MR-Bayesian model averaging (MR-BMA) approach was conducted to elucidate the priorities in host-oral communication, followed by network analyses to explore the interactions among phenotypes.
7	Cramer N[15]	JDDG	2025	German	72	In this cross-sectional study, a wide range of characteristics of the patient's journey were assessed and evaluated from a total of 72 patients with mucosal LP who were treated in the dermatology departments of six German university medical centers between 02/2022 and 07/2023.
8	Ytterberg SR[16]	New England Journal of Medicine	2022	USA	4362	Randomized, open-label, non-inferiority trial (ORAL Surveillance)
9	Mease PJ[17]	Lancet	2017	USA	615	Phase 3 randomized controlled trial
10	Strand V[18]	Rheumatology (Oxford)	2019	USA	3,115	Integrated analysis of data from multiple clinical trials

Table 2: Author, Objectives, and Key Results on Tofacitinib, Oral Disorders, and Related Treatments

Sl No.	Name of Author	Objectives	Result
1	Bezzio C[9]	Tofacitinib is approved for several immune-mediated inflammatory diseases, but safety concerns have recently been raised. We searched PubMed (accessed on 27 February 2023) for original articles regarding tofacitinib's cancer risk when used for rheumatoid arthritis, ulcerative colitis, Crohn's disease, psoriatic arthritis, and ankylosing spondylitis	Of the 2047 initial records, 22 articles describing 26 controlled studies (including 22 randomized controlled trials) were selected. In the comparison between tofacitinib and any control treatment, the relative risk (RR) for any cancer was 1.06 (95% CI, 0.86–1.31; p = 0.95). In separate comparisons between tofacitinib and either a placebo or biological therapy, no difference was found in the overall cancer risk (vs. placebo, RR = 1.04; 95% CI, 0.44–2.48; p = 0.95; vs. biological drugs, RR = 1.06; 95% CI, 0.86–1.31; p = 0.58). When tofacitinib was compared to tumor necrosis factor (TNF) inhibitors, the overall cancer RR was 1.40 (95% CI, 1.06–2.08; p = 0.02). Similarly, significant results were obtained for all cancers, except for non-melanoma skin cancer (RR = 1.47; 95% CI, 1.05–2.06; p = 0.03), and for this skin cancer alone (RR = 1.30; 95% CI, 0.22–5.83; p = 0.88)
2	Yu DA[10]	The aim of the study was to examine the outcome of patients with alopecia areata treated with oral tofacitinib or ruxolitinib in previously published studies.	A total of 1244 studies were identified of which only 12 studies met the inclusion criteria. Of the 346 patients in these 12 studies, 288 had received oral tofacitinib and 58 had received oral ruxolitinib. The overall Severity of Alopecia Tool50 achievement rate was 66% (95% confidence interval, 54%–76%). Subgroup analysis revealed that drug choice, mean age, sex ratio and alopecia areata subtype ratio did not significantly affect the treatment response. Infections and laboratory abnormalities were the most common adverse events (98 and 65 cases of 319 patients, respectively). Patients treated for more than six months had a greater frequency of laboratory abnormalities as compared to those treated for shorter durations (24% vs. 7%; P = 0.04). Recurrence of alopecia areata was observed within three months after discontinuation of treatment in the majority (74%) of patients.

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3	Zhang C[11]	Oral leukoplakia(OLK) is a common oral potentially malignant disorder. The global prevalence of solely OLK was published in 2003, while the prevalence varied among different studies	We obtained 69 studies, including 1,263,028 participants, from 28 countries, and 6 continents. The prevalence was 1.39%, varying from 0.12 to 33.33%. The overall pooled estimated prevalence of OLK was 2.23% for population-based studies, 1.36% for clinic-based population studies, and 9.10% for specific populations. The pooled prevalence in different continents ranged from 0.33 to 11.74% with a statistical difference in the population-based calculation. The estimated prevalence of OLK was higher in males than in females. Those who smoked and consumed alcohol had a higher prevalence than those who did not.
4	Dar-Odeh N[12]	This review aims to identify demographic and clinical characteristics of oral cancer (OC) identified among female patients in the past 25 years, and to explore associated potential risk factors.	A total of 63 cases for females affected by OC were included. A proportion of 32.8% was reported from East Asia. Mean age was 48.7 ± 19.9 years (range = 17–88 years). The main locations of cancer were the tongue (44.4%), followed by the gingiva (36.5%). Age was a significant factor for cancer location; tongue in patients <50 years ($P < .001$); and gingiva in patients ≥50 years ($P = .001$). Most cases of OC were squamous cell carcinoma (SCC; 92.1%), and identified in East Asian countries. Tumors presenting as a mass were significantly located on the gingiva ($P = .011$). Most tongue cancers were ulcers; however, there was no significant association ($P = .058$). Twelve (19.0%) cases were pregnant with OC significantly presenting on tongue ($P = .025$). A proportion of 89.6% constituted SCC in the tongue presenting mainly as a lump (49.2%), and ulcer (39.7%).
5	Vinay K[13]	To compare the efficacy of oral acitretin plus topical triamcinolone acetonide (TAC), 0.1%, with TAC monotherapy in patients with symptomatic OLP.	Among 64 patients, 31 in the treatment group and 30 in the placebo group completed the study (mean [SD] age, 50.6 [15.2] years vs 49.2 [14.4] years; male-female ratio, 13:19 vs 16:16). Baseline ODSS, visual analog scale, and Oral Health Impact Profile 14 scores were comparable in both groups. In the intention-to-treat analysis, there was a statistically significant higher number of patients achieving 75% or higher reduction in ODSS in the treatment group compared with the placebo group at the end of 28 weeks (28 [88%] vs 15 [47%], a 41 [95% CI, 20-61] percentage point difference between groups; $P < .001$; Cramér $V = 0.47$) and 36 weeks (27 [84%] vs 13 [41%], a 43 [95% CI, 23-67] percentage point difference between groups; $P < .001$; Cramér $V = 0.47$). Relapses during the posttreatment follow-up of 8 weeks were low among patients in both treatment and placebo groups (1 [3%] vs 2 [6%], a 3 [95% CI, −13 to 7] percentage point difference between groups; $P > .99$; Cramér $V = 0.07$).
6	Ye X[14]	To determine whether specific circulating inflammatory proteins have a causal effect on oral phenotypes, such as periodontitis, using Mendelian randomization (MR) analysis	After multiple corrections, MR identified five genetically predicted proteins associated with oral phenotypes. Specifically, FGF21 was correlated with Nteeth and DMFS; hGDNF with gingival pain; CCL4 with stomatitis; and S100A12 with denture use. The causal associations remained robust in sensitivity analyses. Nine protein-phenotype clusters were prioritized using MR-BMA. Among these, S100A12, FGF19, FGF21, and CCL4 exhibited extensive correlations with various oral phenotypes.
7	Cramer N[15]	Mucosal lichen planus (LP) is a chronic inflammatory disease. The patient's journey can be arduous as diagnosis and therapy are challenging.	On average, 18.1 months elapsed between the onset of symptoms and diagnosis. Until the correct diagnosis was made, an average of 3.1 different physicians of the same or different specialties were consulted. 28.1% of patients also had cutaneous involvement. Therapeutically, 68% of patients received at least one systemic drug. Both topical (90%, 65/72) and systemic (oral, 50% of patients, 36/72; intravenous, 33%, 24/72) glucocorticoids were most frequently used. Systemic agents were most often discontinued due to ineffectiveness (46%, 50/110). Satisfaction with treatment was highest for intravenous and topical glucocorticoids (moderate to high satisfaction: 59% and 36%, respectively), and lowest for retinoids with 8%.
8	Ytterberg SR[16]	To evaluate the safety of tofacitinib compared to TNF inhibitors in patients with rheumatoid arthritis aged ≥50 years with at least one cardiovascular risk factor	Higher incidence of cancer with tofacitinib compared to TNF inhibitors (HR = 1.48; 95% CI, 1.04–2.09)

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9	Mease PJ[17]	To evaluate the efficacy and safety of tofacitinib in patients with active psoriatic arthritis	Tofacitinib was effective. The incidence of malignancies was low and comparable to placebo
10	Strand V[18]	To assess the long-term safety of tofacitinib in patients with rheumatoid arthritis.	The incidence rates of malignancies were stable over time and comparable to rates observed with other treatments

Table 3: Authors and Key Conclusions on Tofacitinib, Oral Conditions, and Related Treatments

Sl No.	Name of Author	Conclusion
1	Bezzio C[9]	In conclusion, no difference in the overall cancer risk was found between tofacitinib and either a placebo or biological drugs, while a slightly higher risk was found in patients treated with tofacitinib than anti-TNF agents. Further studies are needed to better define the cancer risk of tofacitinib therapy.
2	Yu DA[10]	Both oral tofacitinib and ruxolitinib are effective and well tolerated in the treatment of alopecia areata. Clinicians should be aware of the expected efficacy, adverse events and high recurrence rate of oral JAK inhibitors for alopecia areata to effectively counsel these patients before starting therapy.
3	Zhang C[11]	Combining data from 69 published studies, the prevalence of OLK was determined as 1.39% and the pooling estimated global prevalence was 3.41%. The prevalence was relatively consistent and stable across different continents and different definitions. A higher pooled estimated prevalence was found among males, those aged over 60 years old, smokers, and alcohol consumers. The results from the included studies in this systematic review revealed that the prevalence was relatively consistent and stable across various definitions and continents, which may help in developing global treatment and prevention strategies for oral leukoplakia.
4	Dar-Odeh N[12]	Traditional risk factors were not identified in most cases. Females of all age groups regardless of medical/habitual background should be properly screened for OC. Improvements are warranted in healthcare systems particularly in low-resource settings to spread awareness among patients, their families, and their healthcare providers.
5	Vinay K[13]	In this randomized clinical trial, the combination of oral acitretin and TAC was more effective than TAC monotherapy in patients with symptomatic OLP.
6	Ye X[14]	Our study offers novel genetic insights into the causal relationships, prioritizations, and connections between circulating inflammatory proteins and oral phenotypes. These findings comprehensively depict immune-mediated proteomic profiles underlying the host-oral axis, providing significant implications for clinical practice, public health, and immunopharmacology.
7	Cramer N[15]	This study indicates that there might be a lack of diagnostic awareness among physicians and the unmet need for effective systemic treatment options.
8	Ytterberg SR[16]	Tofacitinib was associated with a higher risk of cancer compared to TNF inhibitors in this high-risk population
9	Mease PJ [17]	Tofacitinib is effective for psoriatic arthritis with a low incidence of malignancies
10	Strand V[18]	Long-term tofacitinib treatment has a consistent safety profile regarding malignancy risk

DISCUSSION

Oral leukoplakia is a potentially precancerous lesion that often manifests as white patches on the mucosal surfaces of the oral cavity. It is known to be associated with various risk factors, including tobacco use, alcohol consumption, and chronic irritation. The treatment of oral leukoplakia typically involves addressing these risk factors and, in some cases, surgical or pharmacological interventions. However, when it comes to the use of systemic medications, such as Janus kinase (JAK) inhibitors like tofacitinib, there is increasing concern about their potential role in the development or exacerbation of oral mucosal lesions.

Tofacitinib is primarily used in the management of autoimmune diseases such as rheumatoid arthritis and psoriatic arthritis. It works by inhibiting JAK enzymes, which are involved in the signaling pathways of various cytokines responsible for inflammation. While tofacitinib is generally considered effective for controlling autoimmune flare-ups, its immunosuppressive nature can lead to unintended consequences, particularly in the context of mucosal integrity.

Immunosuppression induced by tofacitinib can impair the body's natural ability to respond to infections or abnormal cell growth, potentially promoting the development of oral leukoplakia. Moreover, there is evidence suggesting that JAK inhibitors can alter the balance of the oral microbiome, leading to an increased susceptibility to opportunistic infections or mucosal damage. This alteration in immune response could, in turn, contribute to the formation or progression of leukoplakia in susceptible individuals.

Several studies have highlighted the potential for oral mucosal lesions in patients treated with tofacitinib, with reports indicating the development of oral ulcers, lichen planus, and, in rare cases, leukoplakia. However, the exact mechanism through which tofacitinib contributes to oral leukoplakia remains unclear, and further research is needed to understand the underlying pathophysiology.

Given the possible association between tofacitinib treatment and oral leukoplakia, it is essential for clinicians to closely monitor patients receiving this medication for any signs of mucosal changes. Early identification and management of such lesions are crucial to prevent malignant transformation. Patients should also be counseled about the potential risks associated with tofacitinib, including the importance of oral hygiene and regular dental check-ups.

CONCLUSION

We conclude that, oral leukoplakia in patients undergoing tofacitinib treatment presents a

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significant concern due to its potential for malignant transformation and the challenges in managing such lesions. While tofacitinib, a Janus kinase (JAK) inhibitor, is widely used for treating autoimmune conditions, it may contribute to the development or exacerbation of oral leukoplakia through its immunomodulatory effects. The immunosuppressive nature of tofacitinib can impair local immune surveillance, potentially allowing for the growth of abnormal epithelial cells and the development of oral precancerous lesions. Clinicians should be vigilant in monitoring patients on tofacitinib for the onset of oral leukoplakia, considering regular oral examinations and biopsies for suspicious lesions. Further research is warranted to better understand the mechanisms underlying tofacitinib-induced oral leukoplakia and to establish preventive and therapeutic strategies to mitigate this risk. Early detection and timely intervention remain key to managing this complication and preventing progression to oral cancer.

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