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Risk of Latent Tuberculosis and Hepatitis B Virus Reactivation in Patients with Psoriasis Treated with Biologics: A Retrospective Single-center Cohort Study in South Korea

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Psoriasis is a chronic inflammatory skin disease that often requires systemic immunomodulatory therapy[1]. Although the introduction of biologic agents, including tumor necrosis factor (TNF)-α inhibitors, interleukin (IL)-17, and IL-23 inhibitors, has significantly improved disease management, concerns have been raised regarding opportunistic infections, particularly latent tuberculosis infection (LTBI) and hepatitis B virus (HBV) reactivation[1,2]. Such risks are particularly relevant in regions where LTBI and chronic HBV infection remain prevalent, such as Korea. Although infection screening and prophylactic guidelines are well established, real-world data on infection reactivation during biologic therapy remain limited, particularly in East Asian populations. To fill this literature gap, this study aimed to evaluate the incidence of LTBI and HBV reactivation in Korean patients with psoriasis treated with IL-12/23, IL-23, and IL-17 inhibitors, providing long-term real-world data.

Medical records of 296 patients with moderate-to-severe psoriasis who received biologic agents at Kyungpook National University Hospital between January 2013 and August 2025 were retrospectively reviewed. Biologics included IL-12/23 (ustekinumab), IL-23 (guselkumab, risankizumab), and IL-17 inhibitors (secukinumab, ixekizumab). Eligible patients had baseline and at least one follow-up test for LTBI or HBV during treatment. LTBI was evaluated using an interferon-γ release assay (IGRA) and chest radiography, and HBV was assessed using serologic markers (HBsAg, HBsAb, HBcAb), with HBV DNA testing performed when indicated. Patients lacking sufficient data for LTBI or HBV assessment were excluded. Baseline values were defined as results obtained closest to biologic initiation; follow-up tests were conducted every 6-12 months. For patients with IGRA conversion or HBV reactivation, data at the time of the event were analyzed; for all others, the most recent available results were used for comparison. LTBI was defined as IGRA positivity without clinical or radiographic evidence of active tuberculosis, and HBV reactivation as newly detectable HBV DNA or a ≥1 log10 IU/mL increase from baseline. The study was approved by the Institutional Review Board of Kyungpook National University Hospital (IRB no. KNUH 2025-04-039) and conducted in accordance with the Declaration of Helsinki.

Baseline characteristics of the 296 patients included in the analysis are summarized in Table 1. Among them, 287 underwent LTBI monitoring; 61 (21.3%) were IGRA positive and 226 (78.7%) were IGRA-negative at baseline. All patients who were IGRA positive before biologic initiation received prophylactic therapy for LTBI. Among the 226 patients with negative baseline results, IGRA conversion (from negative to

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Table 1. Baseline demographic and clinical characteristics of patients treated with biologics (*n* = 296)

Clinical characteristics	Total patients† (*n* = 296)	LTBI monitoring (*n* = 287)	HBV monitoring (*n* = 269)
Sex, Male:Female	200:96 (2.1:1)	193:94 (2.1:1)	181:88 (2.1:1)
Age at biologic initiation, mean ± SD (yrs)	42.8±13.9	42.8±14.0	43.3±13.7
Age at analysis, mean ± SD (yrs)	N/A	47.0±14.1	47.4±14.0
Comorbidities, *n* (%)			
None	197 (66.6%)	192 (66.9%)	204 (75.8%)
Hypertension	40 (13.5%)	15 (5.2%)	17 (6.3%)
Diabetes mellitus	26 (8.8%)	10 (3.5%)	10 (3.7%)
Dyslipidemia	19 (6.4%)	7 (2.4%)	8 (3.0%)
Biologic treatment duration, mean ± SD (wks)	N/A	237.6±124.2	234.2±127.4
Patients with biologic switch, *n* (%)	55 (18.6%)	53 (18.5%)	51 (19.0%)
Initial biologic agent used, *n* (%)			
Ustekinumab	94 (31.8%)	93 (32.4%)	89 (33.1%)
Guselkumab	85 (28.7%)	81 (28.2%)	81 (30.1%)
Risankizumab	58 (19.6%)	55 (19.2%)	49 (18.2%)
Secukinumab	29 (9.8%)	29 (10.1%)	21 (7.8%)
Ixekizumab	30 (10.1%)	29 (10.1%)	29 (10.8%)
Biologic agent at analysis, *n* (%)			
Ustekinumab	N/A	50 (17.4%)	48 (17.8%)
Guselkumab	N/A	105 (36.6%)	104 (38.7%)
Risankizumab	N/A	70 (24.4%)	62 (23.0%)
Secukinumab	N/A	31 (10.8%)	24 (8.9%)
Ixekizumab	N/A	31 (10.8%)	31 (11.5%)

*Abbreviation: N/A, not applicable; SD, standard deviation; yrs, years; wks, weeks
†Among the 296 patients, 260 underwent both LTBI and HBV monitoring. Given that the follow-up time points for LTBI and HBV evaluations were not identical, parameters requiring a single unified endpoint could not be calculated and are presented as N/A

positive) occurred in 10 patients (4.4%). All patients with IGRA conversion received prophylactic treatment with a 3-month combination regimen of isoniazid and rifampin, and none progressed to active disease. The mean duration from biologic initiation to IGRA conversion was 32.9 ± 16.9 months (range, 13-63 months). Detailed clinical characteristics of the 10 patients who experienced IGRA conversion are presented in Table 2.

HBV monitoring was completed in 269 patients; 5 (1.9%) were HBV carriers (HBsAg positive) who received biologics under antiviral prophylaxis, and 75 (27.9%) had serologic profiles indicating past infection (HBsAg negative and HBcAb positive). Across all serologic groups, no cases of chronic HBV exacerbation or relapse of past HBV infection were detected during treatment.

In this retrospective single-center cohort, a low incidence of IGRA conversion was observed (4.4%), and no cases of active tuberculosis or HBV reactivation, including chronic HBV exacerbation or relapse, were detected among patients with moderate-to-severe psoriasis treated with IL-12/23, IL-23, or IL-17 inhibitors. All IGRA converters received prophylaxis and none developed active disease. These findings support the

Table 2. Clinical characteristics of patients with interferon-γ release assay conversion (n = 10)

Case	Sex	Age at biologic initiation (years)	Biologic agent at initiation	Biologic agent at conversion	Duration from initiation to conversion (weeks)	Comorbidities	Prophylactic regimen after conversion	Progression to active TB
1	M	42	Secukinumab	Secukinumab	57	None	3-month INH/RFP	No
2	M	45	Secukinumab	Secukinumab	89	None	3-month INH/RFP	No
3	M	56	Risankizumab	Risankizumab	97	None	3-month INH/RFP	No
4	F	48	Ixekizumab	Ixekizumab	103	None	3-month INH/RFP	No
5	M	50	Guselkumab	Guselkumab	103	None	3-month INH/RFP	No
6	F	62	Guselkumab	Guselkumab	111	None	3-month INH/RFP	No
7	F	41	Guselkumab	Guselkumab	136	None	3-month INH/RFP	No
8	M	34	Ustekinumab	Guselkumab	197	None	3-month INH/RFP	No
9	F	36	Ustekinumab	Risankizumab	248	None	3-month INH/RFP	No
10	M	62	Ustekinumab	Ustekinumab	272	None	3-month INH/RFP	No

M, Male; F, Female; TB, tuberculosis; INH, isoniazid; RFP, rifampin

safety of IL-17 and IL-23 inhibitors when adequate screening and prophylactic measures are implemented in endemic regions.

TNF inhibitors, the first biologics developed for psoriatic disease, may increase the risk of LTBI reactivation and *de novo* tuberculosis, particularly early in therapy[3]. Accordingly, current guidelines recommend LTBI screening and prophylaxis before initiating biologic therapy[4,5]. In contrast, IL-17 and IL-23 inhibitors pose a lower reactivation risk, as they do not interfere with granuloma maintenance[4]. Torres et al.[5] recently demonstrated no significant increase in tuberculosis reactivation among patients with psoriasis and LTBI treated with IL-17 or IL-23 inhibitors, even in the absence of chemoprophylaxis. Our findings are consistent with these findings, providing real-world evidence from a Korean cohort, where latent infections remain prevalent.

The 4.4% IGRA conversion rate observed in this study warrants careful interpretation. It may reflect either reactivation of latent bacilli or *de novo* infection during immunosuppression. As IL-17 and IL-23 inhibitors are not directly involved in granuloma disruption, *de novo* infection from environmental exposure appears more plausible. The wide temporal distribution of conversions over 1-5 years—without early clustering—also argues against predominant reactivation and supports *de novo* acquisition amid ongoing community transmission. In Korea, where tuberculosis remains at an intermediate-to-high endemic level, longer treatment duration likely reflects cumulative exposure rather than biologic therapy itself activating LTBI. Finally, although a greater number of conversions occurred among IL-23 inhibitor users, this likely reflects their greater representation within the cohort (Table 1) rather than a drug-specific effect.

Regarding HBV reactivation, no cases were observed in our cohort, including among 5 inactive HBsAg carriers who were managed with antiviral prophylaxis. This aligns with prior reports indicating that IL-17 and IL-23 inhibitors carry a relatively low HBV reactivation risk, especially with appropriate antiviral management[6]. Chiu et al.[7] reported a 15.2% reactivation rate among secukinumab-treated patients without prophylaxis, primarily in HBsAg-positive individuals, whereas no reactivation occurred in those who received prophylaxis. Gargiulo et al.[8] similarly observed no reactivation in patients seropositive for HBV treated with IL-17/IL-23 inhibitors. This study provides additional real-world evidence supporting the safe use of these biologics in HBV-endemic regions.

This study holds several limitations. Its retrospective design and single-center setting may limit the generalizability of the findings, and the relatively small number of IGRA converters (n = 10, 4.4%) and HBsAg carriers (n = 5, 1.9%) reduces the statistical power and restricts detailed subgroup analyses. Nevertheless, the long study duration (over 12 years) and comprehensive laboratory follow-up strengthen the reliability of the safety outcomes.

In conclusion, IL-12/23, IL-23, and IL-17 inhibitors can be

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safely administered in patients with psoriasis when appropriate infection screening and prophylactic interventions are implemented. Continuous vigilance through comprehensive baseline evaluation and long-term monitoring remains essential, particularly in regions with a high endemic burden of LTBI and chronic HBV infection.

Key Words: Biologics, Hepatitis B virus reactivation, Latent tuberculosis, Psoriasis

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CONFLICT OF INTEREST

In relation to this article, we declare that there is no conflict of interest.

DATA SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICAL APPROVAL STATEMENT

The study was approved by the Institutional Review Board of (IRB No. KNUH 2025-04-039). This study was conducted in accordance with the principles of the Declaration of Helsinki.

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