International Journal of Basic & Clinical Pharmacology
Shah HJ et al. Int J Basic Clin Pharmacol. 2026 May;15(3):472-478
http://www.ijbcp.com
pISSN 2319-2003 | eISSN 2279-0780

DOI: https://dx.doi.org/10.18203/2319-2003.ijbcp20261111

Original Research Article

Efficacy and safety of combined lactoferrin and luliconazole therapy compared with luliconazole alone in fungal skin infections: a prospective, randomized and active-controlled study

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Received: 09 February 2026
Revised: 11 March 2026
Accepted: 12 March 2026

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ABSTRACT

Background: Fungal skin infections are a prevalent global health concern, affecting millions and often impairing quality of life. Standard antifungal therapies, including azoles, are increasingly limited by rising resistance and biofilm-mediated tolerance. Lactoferrin, an iron-chelating glycoprotein with antimicrobial and antifungal synergy, may enhance therapeutic outcomes. Sequential application of lactoferrin with luliconazole, a potent topical azole, offers a potential strategy to improve biofilm inhibition and accelerate clinical and mycological cure.
Methods: This prospective, randomized, open-label, single-center clinical trial included 60 adults with KOH-confirmed cutaneous candidiasis or dermatophytosis. Participants were randomized 1:1 to receive either sequential lactoferrin with luliconazole (Group A) or luliconazole alone (Group B) for 14 days. Outcomes included biofilm inhibition, mycological and clinical cure, and lesion size reduction. Assessments were performed at baseline, Day 7, and Day 14. Safety was evaluated through adverse event monitoring.
Results: Sixty patients were randomized and analyzed. Baseline demographic characteristics were comparable between groups. Biofilm inhibition was significantly greater in Group A at Day 7 (45.45% vs 19.64%) and Day 14 (90.90% vs 58.92%; $p<0.001$). Mycological cure reached 96.66% in both groups by Day 14, while clinical cure was higher in Group A (80% vs 40%; $p<0.05$). Lesion size reduction was greater in Group A (80.56% vs 65.44%; $p=0.004$). Adverse events were mild and fewer in Group A.
Conclusions: These findings indicate that lactoferrin, when used alongside standard antifungal treatment, showed beneficial effect on biofilm inhibition, supporting its use as a promising adjunctive therapy in superficial fungal infections.

Keywords: Superficial fungal infection, Lactoferrin, Luliconazole, Biofilm inhibition

INTRODUCTION

Fungal skin infections (FSI) represent a major public health issue, creating a substantial burden on patients worldwide. In 2021, global cases of FSI's prevalence and incidence were reported at approximately 616.5 million and 1.73 billion, respectively.[1] Fungal infections can range from mild superficial infections (SFIs) to severe, life-threatening invasive infections. SFIs of the skin and nails are most commonly caused by Candida species (*Candida albicans*), Dermatophytes (the most common being *Trichophyton rubrum, T. mentagrophytes,* and *Epidermophyton floccosum)*, and Malassezia species (*M. globose*).[2] These SFIs interfere with daily activities and are

associated with a poor quality of life and increased healthcare expenditure.[3]

Standard antifungal therapy primarily involves the use of topical or systemic agents, such as azoles, which exert their fungicidal effect by inhibiting lanosterol 14α-demethylase in the ergosterol biosynthesis pathway. This inhibition results in ergosterol depletion and accumulation of lanosterol, ultimately compromising fungal cell membrane integrity.[4,5] However, despite the availability of these drugs, treatment of FSIs is increasingly restricted by antifungal resistance. Antifungal resistance can be multifactorial, including reduced drug uptake, overexpression of efflux pumps, decreased drug affinity for its target, and alterations in multiple metabolic pathways.[6] Furthermore, the biofilm mode of growth significantly impairs treatment efficacy, as the extracellular polymeric matrix limits antifungal drug penetration, thereby exposing only superficial cellular layers to therapeutic concentrations.[7,8] Collectively, biofilm persistence and the emergence of drug-resistant fungal strains highlight the need for innovative antifungal interventions capable of enhancing biofilm disruption and accelerating clinical resolution without compromising cutaneous safety.

Lactoferrin (LF), a non-heme iron-binding glycoprotein belonging to the transferrin family, is abundantly present in mammalian exocrine secretions, particularly milk and colostrum.[9] LF exhibits broad-spectrum antimicrobial activity, primarily through iron sequestration, which restricts microbial growth. Both LF and its derivative peptide lactoferricin have demonstrated significant fungistatic activity against Candida and Trichophyton species and exhibit synergistic interactions with azole and polyene antifungal agents under in vitro conditions.[10] These observations support the potential role of LF as an adjuvant to conventional antifungal therapy. Among azoles, luliconazole demonstrates potent in vitro and clinical activity against Candida and Trichophyton species at low therapeutic concentrations and is effective with once-daily topical administration.[4]

Evidence suggests that the ideal topical antifungal for SFIs, should possess broad-spectrum activity, rapid mycological and clinical cure, convenient dosing, and low resistance rates.[4] Although the antifungal efficacy of LF has been well documented, its role in biofilm inhibition remains insufficiently characterized, with limited evidence available. Considering LF's broad antimicrobial profile, reported synergism with azole antifungals, and the established potent antifungal activity of Luliconazole, a LF-Luliconazole treatment regimen may confer enhanced biofilm disruption and improved therapeutic effectiveness.

Given the current scarcity of clinical data investigating an LF–Luliconazole treatment regimen, the present study aims to evaluate whether Lactoferrin cream sequential application with Luliconazole provides superior biofilm inhibition, mycological clearance, and clinical improvement in patients with cutaneous candidiasis and dermatophytosis compared with Luliconazole monotherapy.

METHODS

Study design

This was a prospective, randomized, open-label, single-center, parallel-group clinical study conducted at a tertiary care center. The primary objective was to evaluate and compare the clinical efficacy and safety of sequential administration of a Lactoferrin-containing cream followed by Luliconazole with that of Luliconazole monotherapy in patients diagnosed with cutaneous candidiasis or dermatophytosis. The study was conducted over the period from May 2024 to September 2024, encompassing patient recruitment, treatment, and follow-up assessments.

Study population

Patients of either sex, aged 18-65 years, presenting to the outpatient department of the tertiary care center with clinically suspected SFI specifically, candidiasis or dermatophytosis were screened for eligibility and confirmed by the presence of fungal hyphae on potassium hydroxide (KOH) microscopy prior to enrollment. Patients were required to abstain from using any topical products on the affected site, except an investigator-approved non-medicated cleanser and sunscreen, for the duration of the study. Women of childbearing age were required to present a negative urine pregnancy test at screening and to use acceptable contraception throughout the study.

Patients were excluded if they had a known hypersensitivity to Lactoferrin, azoles, or related compounds; concomitant dermatological conditions that could hinder clinical assessment; prior systemic antifungal therapy within 30 days of baseline; or concurrent medications that, in the investigator's judgment, might interfere with study outcomes. Pregnant or lactating women, patients with uncontrolled hypertension, serious or chronic systemic disease, history of significant irritation with topical medications, substance dependence, or participation in another clinical study within 90 days prior to screening were also excluded. A total of 65 patients were screened, five did not meet the criteria and were excluded as screen failure. Consequently, 60 patients were proceeding for randomization.

Randomization and allocation

Eligible patients attended a screening visit (Visit 1) and, following confirmation of eligibility, were randomized at Visit 2 (Baseline) in a 1:1 ratio to either the Group A (Lactoferrin cream with Luliconazole cream) or the Group B (Luliconazole cream alone) with 30 patients in each group. Randomization was performed using a centralized, computer-generated allocation sequence. The study flow is presented in Figure 1.

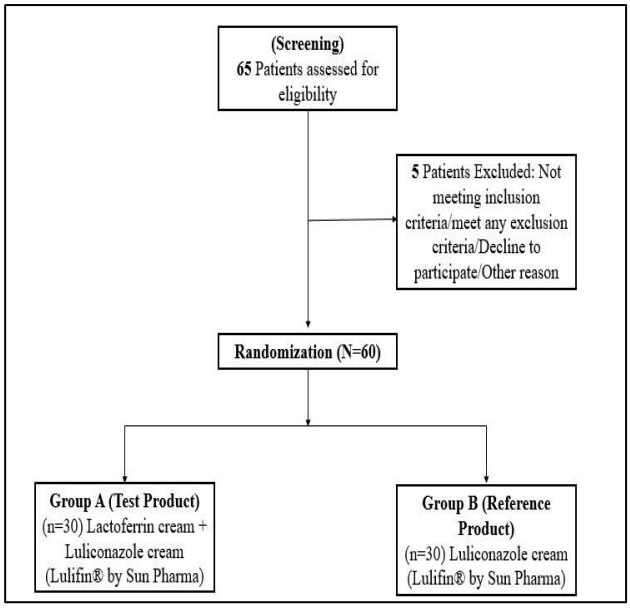

Figure 1: Study flow.

Treatment protocol

Patients were randomized into two treatment arms. Group A (Test group) received Lactoferrin 2% w/w cream (Siracilt FUBI®, Enavant Research LLP) followed by Luliconazole 1% w/w cream (Lulifin®, Sun Pharma), Whereas Group B (reference group) received Luliconazole 1% w/w cream alone.

All patients applied the assigned treatment twice daily to the affected area for 14 days. In Group A, patients applied Lactoferrin cream first, followed by Luliconazole cream in a sequential manner. In contrast, Group B received Luliconazole cream as a monotherapy. Follow-up assessments were conducted at Visit 3 (Day 07±1) and Visit 4 (Day 14±2). Concomitant antifungal or investigational treatment was not permitted.

Outcome measures

Primary endpoint

The primary efficacy endpoint was the rate of biofilm inhibition at Day 7 and Day 14.

Secondary endpoints

The key study outcomes included the proportion of patients achieving mycological cure at Day 7 and Day 14, defined as the absence of fungal elements on potassium hydroxide (KOH) microscopy. Clinical cure, characterized by complete resolution of visible lesions, was also assessed at the same time points. Additionally, safety assessments included recording the incidence of adverse events (AEs) and serious adverse events (SAEs), along with evaluating changes in laboratory safety parameters from baseline to Day 14.

Study assessments

Assessment of biofilm formation inhibition for antifungal resistance

Clinical isolates of Candida or Dermatophyte species were collected using sterile swabs or skin scrapings during Visits 2,3 and 4. Specimens were transported in sterile Stuart's transport medium tubes and stored at 2-8 °C until processing. All samples were labeled and processed within one week of collection.

The isolates were cultured in 96-well microtiter plates containing Yeast Nitrogen Base with 1% glucose and incubated at 37 °C for 24 hours to facilitate biofilm formation. Biofilm biomass was quantified using the Crystal Violet staining method. Absorbance was measured at 595 nm wavelength using a microtiter plate reader to quantify biofilm mass. In addition, structural biofilm morphology and surface adherence were visualized using a Confocal Scanning Laser Microscope equipped with a 63x, 1.2 numerical aperture (NA) water-immersion objective lens. Intra- and inter-visit variations in biofilm formation were statistically compared to assess inhibition across treatment groups.

Mycological cure assessment

Direct microscopic examination using a 10% KOH mount was performed at Visits 2,3 and 4. Clinical specimens collected from the infected area using sterile swabs or skin scrapings were placed on glass slides, treated with 10% KOH, and examined microscopically for fungal elements. The presence or absence of hyphae, pseudohyphae, or spores was recorded, and samples demonstrating a complete absence of fungal structures were classified as mycologically cured.

Clinical cure assessment

Clinical cure was defined as complete lesion resolution or marked improvement. Clinical assessments included lesion evaluation and measurement using Vernier calipers. Lesion area was calculated using the formula: Area=$\pi\times$ (length/2)$\times$(width/2).

Safety assessments

Safety evaluations included monitoring of AEs, SAEs, vital signs, and clinical laboratory parameters. All safety events were categorized based on severity and causality using MedDRA terminology.

Statistical analysis

Efficacy and safety outcomes were summarized using descriptive and inferential statistics. Between-group comparisons were conducted using t-tests, ANOVA, or non-parametric equivalents as appropriate after normality

assessment. A p-value <0.05 was considered statistically significant.

Ethical considerations

The study was approved by the Institutional Ethics Committee and registered with the Clinical Trials Registry-India (CTRI/2024/03/063840; registered on 08 March 2024). Written informed consent was obtained from all patients. The study adhered to the declaration of Helsinki, International Council for Harmonisation (ICH) Good Clinical Practice guidelines, and applicable regulatory requirements.

RESULTS

Sixty patients were randomized and stratified into two groups: (I) Group A: Lactoferrin containing cream+ Luliconazole cream, and (II) Group B: Luliconazole cream monotherapy. The mean age of patients had no substantial age-related imbalance between groups (P>0.05). Gender distribution was comparable across groups, with a slight predominance of male participants observed in both cohorts (Table 1).

Inhibition of biofilm formation

The findings demonstrate a superior inhibitory effect on biofilm formation in Group A compared with Group B. The mean change from baseline to Visit 3 (Day 7) was significantly greater in Group A (−0.25±0.04) compared to Group B (−0.11±0.01) (p<0.001). This corresponded to a higher inhibition rate in Group A (45.45%) versus Group B (19.64%). By Visit 4 (Day 14), Group A demonstrated near-complete suppression of biofilm formation with a mean value of 0.05±0.04, reflecting a 90.90% reduction from baseline. In contrast, Group B showed continued but less pronounced improvement, with a mean biofilm score of 0.23±0.03 and a reduction rate of 58.92% (p<0.001) (Table 2).

Rate of mycological cure

Mycological outcomes also demonstrated a modestly faster clearance trend in Group A. At Visit 3, absence of fungal elements was noted in 63.33% of Group A participants compared with 56.66% in Group B. By Visit 4, both treatments resulted in near-complete eradication, with 96.66% clearance in both groups (Table 3).

Clinical cure analysis

Clinical cure rates revealed a more pronounced difference between interventions. At Visit 3, only one patient in each group (3.33%) achieved a complete clinical response. However, by Visit 4, Group A showed significantly superior outcomes, with 80% of patients attaining complete clinical resolution compared with 40% in Group B (P<0.05) (Table 4).

Change in average lesion area

At baseline (Visit 2), the mean lesion areas were comparable between the two groups. By Visit 4 (Day14), Group A demonstrated a mean lesion area reduction of 11366.19±6432.50 mm² (80.56% improvement), significantly greater than the reduction observed in Group B (7841.42±3548.49 mm²; 65.44% reduction) (p= 0.004) (Table 5).

Table 1: Baseline demographic and clinical characteristics of study participants in both treatment groups.

Parameters	Statistics	Group A (n=30)	Group B (n=30)
Age (years)	Mean±SD	32.13±9.73	34.73±10.50
Gender	Male, N (%)	16 (53.33)	18 (60.00)
	Female, N (%)	14 (46.66)	12 (40.00)

N: The number of subjects in the safety population for each group, n: Number of patients in the specified category.

Table 2: Rate of inhibition of biofilm formation assessed from visit 2 (baseline) to visit 3 (day 7) and visit 4 (day 14).

Parameters	Statistics	Group A (n=30)	Group B (n=30)
Visit 2	Mean±SD	0.55±0.07	0.56±0.07
Visit 3	Mean±SD	0.30±0.03	0.45±0.08
Visit 4	Mean±SD	0.05±0.04	0.23±0.03
Change from visit 2 → visit 3	Mean±SD	-0.25±0.04	-0.11±0.01
	p-value (within group)	<0.001	<0.001
	p-value (inter group)	<0.001	
Rate of biofilm inhibition (%)		45.45	19.64
Change from visit 2 → visit 4	Mean±SD	-0.50±0.03	-0.33±0.04
	p-value (within group)	<0.001	<0.001
	p-value (inter group)	<0.001	
Rate of biofilm inhibition (%)		90.90	58.92

n: Number of patients in the specified category, %: Mean percentage change from baseline within the group at the specified visit.

Table 3: Proportion of patients with absence of fungal elements at visit 3 (day 7) and visit 4 (day 14).

Parameters	Statistics	Group A (n=30)	Group B (n=30)
Visit 3	N (%)	19 (63.33)	17 (56.66)
Visit 4	N (%)	29 (96.66)	29 (96.66)

N: The number of subjects in the safety population for each Group, n: Number of patients having a specified category, %: Calculated using the number of subjects in the safety population for each Group, or the safety population for the overall, as the Denominator (n/N*100).

Table 4: Proportion of patients achieving clinical cure at visit 2 (baseline), visit 3 (day 7), and visit 4 (day 14).

Parameters	Statistics	Group A (n=30)	Group B (n=30)
Visit 2	N (%)	0 (0.00)	0 (0.00)
Visit 3	N (%)	1 (3.33)	1 (3.33)
Visit 4	N (%)	24 (80.00)	12 (40.00)

N: The number of subjects in the safety population for each Group, n: Number of patients having at specified category, %: Calculated using the number of subjects in the safety population for each Group, or the safety population for the overall, as Denominator (n/N*100).

Table 5: Average lesion area at different visits and change in average lesion are from visit 2 (baseline) to visit 4 (day 14).

Visit	Statistics	Group A (n=30)	Group B (n=30)
Visit 2	Mean±SD	14108.80±8292.29	11982.14±7479.39
Visit 3	Mean±SD	5827.38±3252.96	7021.66±4507.28
Visit 4	Mean±SD	2742.60±1859.79	4140.71±3930.90
Mean reduction (visit 2 → 4)	Mean±SD	11366.19±6432.50	7841.42±3548.49
	p-value (within group)	<0.001	<0.001
	p-value (inter group)	0.004	
% reduction (visit 2 → 4)	%	80.56	65.44

N: The number of subjects in the safety population for each Group, n: Number of patients having at specified category, %: Mean percentage change from baseline within the group at the specified visit.

Table 6: Overall summary of adverse events in both treatment groups.

Observed AES in both treatment groups			
Variable	**Group A (n=30)**	**Group B (n=30)**	**Overall (n=60)**
Total number of AE	2	7	9
Total number of patients with at least one AE	2	7	9
Patient discontinued due to AE	0	0	0
AES categorized by severity grade in both treatment groups			
Severity	**Group A (n=30)**	**Group B (n=30)**	**Overall (n=60)**
Mild	2	7	9
Moderate	0	0	0
Severe	0	0	0

N: The number of subjects in the safety population for each Group, n: Number of subjects with adverse event.

Safety analysis

Safety assessments indicated that both regimens were well tolerated. A total of nine adverse events (AEs) were reported during the study period, with 6.67% (n=2) incidents in Group A and 23.33% (n=7) in Group B. All AEs were mild in severity, and no serious or treatment-related discontinuations occurred (Table 6).

DISCUSSION

This study evaluated the potential synergistic effect of sequentially administered Lactoferrin with Luliconazole in patients with candidiasis and dermatophytosis. Existing evidence suggests that Lactoferrin and its derivatives exhibit broad-spectrum antifungal activity against multiple Candida species,[11] and enhance the efficacy of azole antifungals by inducing fungal apoptosis through mechanisms involving chromatin condensation, DNA fragmentation, and reactive oxygen species accumulation.[12,13] Although these findings provide a strong mechanistic foundation, clinical data supporting Lactoferrin's antibiofilm activity (in-vivo) remain scarce. To the best of our knowledge, this is the first clinical evaluation of sequential Lactoferrin and Luliconazole application in superficial fungal infections; therefore,

direct comparison with previously published clinical outcomes is limited.

The present findings demonstrated that the addition of Lactoferrin to Luliconazole resulted in a significantly greater and more rapid reduction in biofilm formation compared with Luliconazole monotherapy. This observation aligns with existing in vitro data showing that Lactoferrin-derived peptides inhibit Candida biofilm development and exert cytotoxic effects against fluconazole-resistant strains.[14,15] Taken together, these results support the mechanistic rationale for adjunctive Lactoferrin use in enhancing antibiofilm response, particularly in cases where biofilm-associated resistance may contribute to treatment failure.

Patients treated with Lactoferrin followed by Luliconazole exhibited a higher mycological cure rate at Week 1, indicating an accelerated therapeutic response. This outcome aligns with proposed mechanisms of Lactoferrin activity, including iron sequestration, inhibition of Fe^{3+} uptake, disruption of fungal cell membrane integrity, and suppression of biofilm formation. Notably, previous studies indicate that this synergistic effect attenuated in the presence of high iron concentrations, further highlighting the relevance of iron metabolism in the antifungal response.[12-16] By Week 2, both treatment groups demonstrated substantial improvement, achieving comparable mycological clearance rates of 96.66%. Previously reported mycological cure rates for Luliconazole monotherapy range between 76.1% and 82.5%, suggesting a possible clinical advantage conferred by the LF-Luliconazole treatment regimen.[17-19] Clinical cure was achieved in 80% of patients receiving the Lactoferrin with Luliconazole treatment regimen compared with 40% among those receiving Luliconazole alone. Given that reported clinical cure rates for Luliconazole vary widely across studies, the enhanced response observed in this study suggests that adjunctive Lactoferrin may improve both therapeutic consistency and outcome predictability.[4-18] Lactoferrin's multifunctional antimicrobial, antioxidant, anti-inflammatory, and immunomodulatory properties may contribute to this enhanced clinical effect.[20]

The Lactoferrin with Luliconazole treatment group also demonstrated greater reduction in lesion size compared with monotherapy, supporting the therapeutic benefit of adjunctive Lactoferrin. While Luliconazole remains an established and effective topical antifungal agent, emerging data suggest that Lactoferrin exhibits additional antifungal pathways, including direct interaction with fungal cell surfaces resulting in membrane destabilization and cellular leakage.[16] Such multifaceted activity may contribute to enhanced lesion resolution.

Treatment-related adverse events were infrequent, mild in severity, and did not lead to treatment discontinuation. Interestingly, adverse events were lower in the Lactoferrin with Luliconazole treatment group than in patients receiving Luliconazole monotherapy. Existing evidence indicates that Lactoferrin reduces the incidence of invasive fungal infections. Mechanistically, Lactoferrin interacts directly with fungal cell membranes, leading to membrane disruption and subsequent cellular damage.[21]

Limitations

This study has several limitations, this was a single-center, open-label study, which may limit generalizability and introduce potential bias. The relatively small sample size and short follow-up duration restrict the ability to assess long-term outcomes or recurrence. Therefore, larger multi-center, blinded trials with extended follow-up and detailed microbiological evaluation are needed to confirm and expand these results.

CONCLUSION

These findings indicate that Lactoferrin, when used alongside standard antifungal treatment, showed beneficial effect on biofilm inhibition, supporting its use as a promising adjunctive therapy in superficial fungal infections.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee CTRI/2024/03/063840

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Cite this article as: Shah HJ, Pariyani JK, Shinde KV, Pandya DP, Dave BA, Lokhande MM. Efficacy and safety of combined lactoferrin and luliconazole therapy compared with luliconazole alone in fungal skin infections: a prospective, randomized and active-controlled study. Int J Basic Clin Pharmacol 2026;15:472-8.