Section: Dermatology

Original Research Article

CLINICOMYCOLGICAL PROFILE AND ANTIFUNGAL SUSCEPTIBILITY PATTERNS IN TINEA CORPORIS: CORRELATION OF MIC VALUES WITH CLINICAL OUTCOMES - A PROSPECTIVE STUDY

Priyanka Kamlesh Kanoje[1], Poonam Puri[2], Malini R. Capoor[3]

[1]Assistant Professor, Department of Dermatology and STD, Dr Shankarrao Chavhan Govt Medical College Vishnupri Nanded, India.
[2]Professor and Consultant, Department of Dermatology and STD, Vardhaman Mahavir Medical College and Safdarjung Hospital New Delhi, India.
[3]Professor and Senior Specialist, Department of Microbiology, Vardhaman Mahavir Medical College and Safdarjung Hospital New Delhi, India.

Received *: 01/01/2026*
Received in revised form *: 10/01/2026*
Accepted *: 19/01/2026*

Corresponding Author:
Dr. Priyanka Kamlesh Kanoje
Assistant Professor, Department of Dermatology And STD, Dr Shankarrao Chavhan Govt Medical College Vishnupri Nanded, India.
Email: priyanka.k1406@gmail.com

DOI:10.70034/ijmedph.2026.1.234

Source of Support: Nil,
Conflict of Interest: Nonedeclared

Int J Med Pub Health
2026; 16 (1); 1337-1346

ABSTRACT

Background: Tinea corporis is one of the most prevalent dermatophytic infections worldwide, with increasing reports of treatment failure and recurrence due to changing fungal epidemiology and emerging antifungal resistance. Antifungal susceptibility testing using minimum inhibitory concentration (MIC) values has gained importance in guiding effective therapy. This study was undertaken to evaluate the clinicomycolgical profile of tinea corporis and correlate antifungal susceptibility patterns with clinical outcomes.

Objectives: To study the clinical and mycological characteristics of tinea corporis, assess antifungal susceptibility patterns using MIC values, and correlate these findings with treatment response.

Materials and Methods: This prospective observational study included 52 clinically diagnosed cases of tinea corporis attending a tertiary care hospital. Clinical severity of pruritus, erythema, and scaling was assessed at baseline, 2 weeks, and 4 weeks. Skin scrapings were examined using KOH mount and fungal culture, followed by antifungal susceptibility testing using broth microdilution method as per CLSI guidelines. Patients were treated with different antifungal agents and followed up to assess clinical improvement, mycological clearance, adverse effects, and relapse. Statistical analysis was performed using appropriate tests, and a p-value <0.05 was considered significant.

Results: At baseline, the majority of patients presented with moderate to severe symptoms. At 4 weeks, itraconazole showed the highest pruritus resolution rate (81.82%) and mycological clearance (54.55%), followed by terbinafine. Percentage improvement in pruritus, erythema, and scaling was significantly higher with itraconazole and terbinafine compared to griseofulvin, ketoconazole, and fluconazole ($p<0.001$). Higher MIC values for fluconazole and ketoconazole correlated with poorer clinical outcomes and higher relapse rates. Adverse effects were more frequent in the ketoconazole group. Overall relapse was observed in 84.62% of patients, with the lowest relapse rate noted in the itraconazole group.

Conclusion: Itraconazole and terbinafine demonstrated superior clinical efficacy and lower MIC values, correlating well with improved treatment outcomes. Antifungal susceptibility testing plays a critical role in optimizing therapeutic strategies and addressing the growing problem of antifungal resistance in dermatophytosis.

Keywords: Tinea corporis. Antifungal susceptibility testing. Minimum inhibitory concentration (MIC).

INTRODUCTION

Dermatophytosis is one of the most common superficial fungal infections worldwide, affecting keratinized tissues such as skin, hair, and nails. Among its clinical variants, tinea corporis is a frequent presentation characterized by annular, erythematous, scaly lesions with central clearing and active borders. The global burden of dermatophytosis has shown a rising trend, particularly in tropical and subtropical regions, including India, where warm and humid climatic conditions, overcrowding, poor hygiene, and widespread misuse of topical steroid combinations have contributed to increasing prevalence and chronicity of infections.[1]

In recent years, the epidemiology of dermatophytosis has undergone significant changes. Traditionally predominant species such as Trichophyton rubrum and Trichophyton mentagrophytes complex have demonstrated altered distribution patterns and emerging resistance to commonly used antifungal agents. This shift has resulted in higher rates of treatment failure, recurrence, and atypical clinical presentations, posing diagnostic and therapeutic challenges to clinicians.[2] The increasing incidence of recalcitrant dermatophytosis highlights the need for laboratory confirmation and antifungal susceptibility testing to guide rational therapy.

Antifungal susceptibility testing, particularly the determination of Minimum Inhibitory Concentration (MIC) values, has gained importance as a tool to assess the in vitro effectiveness of antifungal agents against dermatophytes. MIC values provide quantitative data regarding fungal sensitivity and resistance patterns, allowing clinicians to optimize antifungal selection and dosing strategies. However, there remains limited correlation data between laboratory-derived MIC values and actual clinical outcomes in routine practice.[3]

The emergence of antifungal resistance, especially against azoles and allylamines such as terbinafine, has been reported in several studies across India and other countries. Genetic mutations in the squalene epoxidase gene and other resistance mechanisms have been implicated in reduced drug susceptibility. These developments emphasize the necessity for region-specific antifungal surveillance and susceptibility profiling.[4]

Aim

To study the clinicomycological profile and antifungal susceptibility patterns in patients with tinea corporis and correlate MIC values with clinical outcomes.

Objectives

1. To analyze the clinical and demographic characteristics of patients diagnosed with tinea corporis.
2. To identify dermatophyte species and determine their antifungal susceptibility patterns using MIC values.
3. To correlate MIC values with treatment response and clinical outcomes.

MATERIALS AND METHODS

Source of Data

The study data were obtained from patients clinically diagnosed with tinea corporis attending the Dermatology Outpatient Department of the tertiary care hospital. Laboratory data were collected from the Department of Microbiology/Mycology.

Study Design

This study was conducted as a prospective observational study.

Study Location

The study was carried out at the Department of Dermatology in collaboration with the Department of Microbiology/Mycology at a tertiary care teaching hospital.

Study Duration

The study was conducted over a period of 12 months.

Sample Size

A total of 52 patients clinically suspected of tinea corporis were enrolled in the study based on inclusion and exclusion criteria.

Inclusion Criteria

- Patients of either gender aged ≥18 years.
- Clinically diagnosed cases of tinea corporis.
- Patients who had not received systemic antifungal therapy in the preceding four weeks.
- Patients willing to provide informed written consent.

Exclusion Criteria

- Patients with mixed dermatophytosis involving multiple anatomical sites.
- Patients on prolonged topical steroid or immunosuppressive therapy.
- Pregnant and lactating women.
- Patients with known immunocompromised states such as HIV infection or malignancy.
- Patients unwilling to participate in the study.

Procedure and Methodology

After obtaining informed consent, detailed clinical history and demographic data were recorded using a structured proforma. Clinical examination was performed to document lesion morphology, distribution, duration of symptoms, and prior treatment history. Skin scrapings were collected aseptically from the active margins of lesions after cleaning the area with 70% alcohol.

Patients were initiated on standard antifungal therapy as per institutional protocol and followed up at regular intervals to assess clinical response. Treatment outcomes were categorized as complete cure, partial response, or treatment failure based on clinical improvement and symptom resolution.

Sample Processing

Collected skin scrapings were subjected to direct microscopic examination using 10% potassium hydroxide (KOH) mount for detection of fungal elements. Samples were cultured on Sabouraud Dextrose Agar (SDA) with and without cycloheximide and incubated at 25-28°C for up to four weeks. Fungal isolates were identified based on colony morphology, microscopic features using lactophenol cotton blue mount, and standard mycological techniques.

Antifungal susceptibility testing was performed using the broth microdilution method as per Clinical and Laboratory Standards Institute (CLSI) guidelines. MIC values for commonly used antifungal drugs were recorded.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using statistical software. Descriptive statistics were expressed as mean, standard deviation, frequencies, and percentages. The correlation between MIC values and clinical outcomes was assessed using appropriate statistical tests such as Chi-square test and Pearson or Spearman correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

Data Collection

Data were collected using a predesigned case record form that included demographic details, clinical findings, laboratory results, antifungal susceptibility data, and treatment outcomes. Follow-up data were recorded systematically to evaluate therapeutic response and correlate with MIC values.

RESULTS

Table 1. Comparison of pruritus, erythema and scaling between drugs T, I, G, K and F

A) Pruritus

Pruritus at 0 week

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Mild	1 (9.09%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1.92%)	0.952*
Moderate	2 (18.18%)	4 (36.36%)	3 (30%)	3 (30%)	2 (20%)	14 (26.92%)	
Severe	8 (72.73%)	7 (63.64%)	7 (70%)	7 (70%)	8 (80%)	37 (71.15%)	
Median (25th-75th)	3 (2.5-3)	3 (2-3)	3 (2.25-3)	3 (2.25-3)	3 (3-3)	3 (2-3)	0.957

Pruritus at 2 weeks

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	2 (18.18%)	2 (18.18%)	0 (0%)	0 (0%)	0 (0%)	4 (7.69%)	0.045*
Mild	2 (18.18%)	7 (63.64%)	5 (50%)	2 (20%)	2 (20%)	18 (34.62%)	
Moderate	7 (63.64%)	2 (18.18%)	4 (40%)	5 (50%)	7 (70%)	25 (48.08%)	
Severe	0 (0%)	0 (0%)	1 (10%)	3 (30%)	1 (10%)	5 (9.62%)	
Median (25th-75th)	2 (1-2)	1 (1-1)	1.5 (1-2)	2 (2-2.75)	2 (2-2)	2 (1-2)	0.013

Pruritus at 4 weeks

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	4 (36.36%)	9 (81.82%)	4 (40%)	0 (0%)	0 (0%)	17 (32.69%)	0.0001*
Mild	7 (63.64%)	2 (18.18%)	6 (60%)	6 (60%)	9 (90%)	30 (57.69%)	
Moderate	0 (0%)	0 (0%)	0 (0%)	2 (20%)	1 (10%)	3 (5.77%)	
Severe	0 (0%)	0 (0%)	0 (0%)	2 (20%)	0 (0%)	2 (3.85%)	
Median (25th-75th)	1 (0-1)	0 (0-0)	1 (0-1)	1 (1-2)	1 (1-1)	1 (0-1)	<0.0001

B) Erythema

Erythema at 0 week

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Moderate	5 (45.45%)	4 (36.36%)	4 (40%)	8 (80%)	5 (50%)	26 (50%)	0.297†
Severe	6 (54.55%)	7 (63.64%)	6 (60%)	2 (20%)	5 (50%)	26 (50%)	
Median (25th-75th)	3 (2-3)	3 (2-3)	3 (2-3)	2 (2-2)	2.5 (2-3)	2.5 (2-3)	0.307

Erythema at 2 weeks

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	0 (0%)	2 (18.18%)	0 (0%)	0 (0%)	0 (0%)	2 (3.85%)	0.0002*
Mild	5 (45.45%)	6 (54.55%)	0 (0%)	0 (0%)	0 (0%)	11 (21.15%)	
Moderate	6 (54.55%)	3 (27.27%)	9 (90%)	8 (80%)	7 (70%)	33 (63.46%)	
Severe	0 (0%)	0 (0%)	1 (10%)	2 (20%)	3 (30%)	6 (11.54%)	
Median (25th-75th)	2 (1-2)	1 (1-1.5)	2 (2-2)	2 (2-2)	2 (2-2.75)	2 (1.75-2)	<0.0001

Erythema at 4 weeks

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	4 (36.36%)	6 (54.55%)	1 (10%)	0 (0%)	0 (0%)	11 (21.15%)	<0.0001*
Mild	7 (63.64%)	5 (45.45%)	4 (40%)	1 (10%)	4 (40%)	21 (40.38%)	
Moderate	0 (0%)	0 (0%)	5 (50%)	6 (60%)	6 (60%)	17 (32.69%)	
Severe	0 (0%)	0 (0%)	0 (0%)	3 (30%)	0 (0%)	3 (5.77%)	
Median (25th-75th)	1 (0-1)	0 (0-1)	1.5 (1-2)	2 (2-2.75)	2 (1-2)	1 (1-2)	<0.0001

C) Scaling

Scaling at 0 week

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Mild	2 (18.18%)	0 (0%)	1 (10%)	4 (40%)	2 (20%)	9 (17.31%)	0.491*
Moderate	3 (27.27%)	6 (54.55%)	5 (50%)	2 (20%)	4 (40%)	20 (38.46%)	
Severe	6 (54.55%)	5 (45.45%)	4 (40%)	4 (40%)	4 (40%)	23 (44.23%)	
Median (25th-75th)	3 (2-3)	2 (2-3)	2 (2-3)	2 (1-3)	2 (2-3)	2 (2-3)	0.797

Scaling at 2 weeks

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	2 (18.18%)	1 (9.09%)	0 (0%)	0 (0%)	0 (0%)	3 (5.77%)	0.377*
Mild	4 (36.36%)	8 (72.73%)	5 (50%)	4 (40%)	6 (60%)	27 (51.92%)	
Moderate	5 (45.45%)	2 (18.18%)	5 (50%)	4 (40%)	3 (30%)	19 (36.54%)	
Severe	0 (0%)	0 (0%)	0 (0%)	2 (20%)	1 (10%)	3 (5.77%)	
Median (25th-75th)	1 (1-2)	1 (1-1)	1.5 (1-2)	2 (1-2)	1 (1-2)	1 (1-2)	0.254

Scaling at 4 weeks

Severity	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	4 (36.36%)	7 (63.64%)	2 (20%)	0 (0%)	0 (0%)	13 (25%)	0.001*
Mild	7 (63.64%)	4 (36.36%)	7 (70%)	6 (60%)	10 (100%)	34 (65.38%)	
Moderate	0 (0%)	0 (0%)	1 (10%)	2 (20%)	0 (0%)	3 (5.77%)	
Severe	0 (0%)	0 (0%)	0 (0%)	2 (20%)	0 (0%)	2 (3.85%)	
Median (25th-75th)	1 (0-1)	0 (0-1)	1 (1-1)	1 (1-2)	1 (1-1)	1 (0.75-1)	0.0007

Table 1 shows the baseline severity and sequential changes in pruritus, erythema, and scaling among patients treated with drugs T, I, G, K, and F. At baseline (0 week), most patients in all groups had severe pruritus (overall 71.15%), moderate-to-severe erythema (100%), and moderate-to-severe scaling (82.69%), with no statistically significant difference between treatment groups ($p>0.05$). At 2 weeks, a significant reduction in pruritus was observed, particularly in the I group, where a higher proportion of patients shifted to mild or absent pruritus ($p=0.045$). Median pruritus scores differed significantly across groups ($p=0.013$). By 4 weeks, the I group demonstrated the best improvement, with 81.82% patients having absent pruritus, compared to persistent moderate-to-severe symptoms in K and F groups ($p=0.0001$). Similar trends were observed for erythema and scaling. At 4 weeks, erythema resolution was highest in the I group (54.55% absent) and T group (36.36% absent), while K and F groups continued to show moderate-to-severe erythema ($p<0.0001$). Scaling reduction was also significantly superior in the I and T groups at 4 weeks, with 63.64% and 36.36% patients showing absence of scaling respectively ($p=0.001$). Overall, itraconazole (I) and terbinafine (T) demonstrated superior clinical improvement compared to griseofulvin (G), ketoconazole (K), and fluconazole (F).

Table 2: Comparison of percentage improvement in pruritus, erythema and scaling between drugs T, I, G, K and F

Parameter	T (n=11) Mean±SD	I (n=11) Mean±SD	G (n=10) Mean±SD	K (n=10) Mean±SD	F (n=10) Mean±SD	Total Mean±SD	P value
% improvement in pruritus at 2 weeks	50 ± 26.87	63.64 ± 22.13	41.67 ± 19.64	23.33 ± 21.08	33.33 ± 13.61	42.95 ± 24.77	0.001‡
% improvement in pruritus at 4 weeks	78.79 ± 16.82	93.94 ± 13.48	76.67 ± 21.08	41.67 ± 25.15	60 ± 11.65	70.83 ± 25.11	<0.0001‡
% improvement in erythema at 2 weeks	39.39 ± 17.12	60.61 ± 23.89	16.67 ± 17.57	0 ± 0	6.67 ± 14.05	25.64 ± 27.9	<0.0001‡
% improvement in erythema at 4 weeks	77.27 ± 18.67	83.33 ± 19.72	43.33 ± 30.63	-3.33 ± 36.68	35 ± 21.44	48.4 ± 40.46	<0.0001‡
% improvement in scaling at 2 weeks	48.48 ± 30.23	54.55 ± 18.4	31.67 ± 24.15	6.67 ± 14.05	26.67 ± 29.61	34.29 ± 28.85	0.0003‡
% improvement in scaling at 4 weeks	71.21 ± 29.9	84.85 ± 21.67	55 ± 34.29	1.67 ± 76.36	46.67 ± 25.82	52.88 ± 49.37	0.0006‡

Table 2 compares the percentage improvement in pruritus, erythema, and scaling across treatment groups. At 2 weeks, the highest mean percentage improvement in pruritus was seen in the I group (63.64 ± 22.13%), followed by T (50 ± 26.87%), while the lowest improvement was observed in the K group (23.33 ± 21.08%) ($p=0.001$). At 4 weeks, itraconazole again showed maximum improvement in pruritus (93.94 ± 13.48%), followed by T (78.79 ± 16.82%) and G (76.67 ± 21.08%), with K showing the least improvement (41.67 ± 25.15%) ($p<0.0001$). Erythema improvement followed a similar pattern, with I and T groups demonstrating significantly higher improvement at both 2 and 4 weeks, whereas minimal or negative improvement was noted in the K group ($p<0.0001$). Scaling improvement was also significantly greater in I and T groups at both follow-ups, confirming superior clinical response with these drugs ($p<0.001$).

Table 3: Comparison of improvement in pruritus, erythema and scaling between drugs T, I, G, K and F

A) Pruritus

Improvement in pruritus at 2 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
No change	0 (0%)	0 (0%)	1 (10%)	4 (40%)	1 (10%)	6 (11.54%)	0.027*
Improved	11 (100%)	11 (100%)	9 (90%)	6 (60%)	9 (90%)	46 (88.46%)	

Improvement in pruritus at 4 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
No change	0 (0%)	0 (0%)	0 (0%)	2 (20%)	0 (0%)	2 (3.85%)	0.102*
Improved	11 (100%)	11 (100%)	10 (100%)	8 (80%)	10 (100%)	50 (96.15%)	

B) Erythema

Improvement in erythema at 2 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
No change	1 (9.09%)	0 (0%)	5 (50%)	10 (100%)	8 (80%)	24 (46.15%)	<0.0001*
Improved	10 (90.91%)	11 (100%)	5 (50%)	0 (0%)	2 (20%)	28 (53.85%)	

Improvement in erythema at 4 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
No change	0 (0%)	0 (0%)	2 (20%)	4 (40%)	2 (20%)	8 (15.38%)	0.0006*
Deteriorated	0 (0%)	0 (0%)	0 (0%)	3 (30%)	0 (0%)	3 (5.77%)	
Improved	11 (100%)	11 (100%)	8 (80%)	3 (30%)	8 (80%)	41 (78.85%)	

C) Scaling

Improvement in scaling at 2 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
No change	1 (9.09%)	0 (0%)	3 (30%)	8 (80%)	5 (50%)	17 (32.69%)	0.0002*
Improved	10 (90.91%)	11 (100%)	7 (70%)	2 (20%)	5 (50%)	35 (67.31%)	

Improvement in scaling at 4 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
No change	1 (9.09%)	0 (0%)	2 (20%)	5 (50%)	2 (20%)	10 (19.23%)	0.027*
Deteriorated	0 (0%)	0 (0%)	0 (0%)	1 (10%)	0 (0%)	1 (1.92%)	
Improved	10 (90.91%)	11 (100%)	8 (80%)	4 (40%)	8 (80%)	41 (78.85%)	

Table 3 depicts categorical improvement patterns in symptoms. At 2 weeks, improvement in pruritus was observed in 100% of patients in T and I groups, compared to only 60% in the K group (p=0.027). By 4 weeks, nearly all patients showed improvement except for 20% in the K group (p=0.102). Improvement in erythema at 2 weeks was highest in the I (100%) and T (90.91%) groups, whereas no improvement was observed in the K group (p<0.0001). At 4 weeks, deterioration was noted in 30% of patients receiving K, while complete improvement was achieved in T and I groups (p=0.0006). Scaling improvement also favored T and I groups significantly at both 2 and 4 weeks, whereas poor response and deterioration were predominantly seen in the K group (p<0.05).

Table 4: Comparison of clinical cure rate between drugs T, I, G, K and F

At 2 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Markedly improved	4 (36.36%)	7 (63.64%)	1 (10%)	0 (0%)	0 (0%)	12 (23.08%)	0.0001*
Considerable residual lesions	7 (63.64%)	4 (36.36%)	5 (50%)	3 (30%)	6 (60%)	25 (48.08%)	
No change	0 (0%)	0 (0%)	4 (40%)	7 (70%)	4 (40%)	15 (28.85%)	

At 4 weeks

Outcome	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Healed	4 (36.36%)	6 (54.55%)	1 (10%)	0 (0%)	0 (0%)	11 (21.15%)	<0.0001*
Markedly improved	7 (63.64%)	5 (45.45%)	3 (30%)	0 (0%)	1 (10%)	16 (30.77%)	
Considerable residual lesions	0 (0%)	0 (0%)	6 (60%)	7 (70%)	9 (90%)	22 (42.31%)	
Worse	0 (0%)	0 (0%)	0 (0%)	3 (30%)	0 (0%)	3 (5.77%)	

Table 4 highlights the clinical cure rates among different treatment groups. At 2 weeks, the I group demonstrated the highest proportion of markedly improved patients (63.64%), followed by T (36.36%), while no marked improvement was seen in K and F groups (p=0.0001). At 4 weeks, complete healing was observed in 54.55% of patients in the I group and 36.36% in the T group, whereas none of the patients in K and F groups achieved complete cure. Additionally, worsening of lesions was observed only in the K group (30%) (p<0.0001), indicating inferior therapeutic efficacy of ketoconazole in this cohort.

Table 5: Comparison of KOH status between drugs T, I, G, K and F

KOH status	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
At 2 weeks							
Positive	11 (100%)	11 (100%)	10 (100%)	10 (100%)	10 (100%)	52 (100%)	NA
At 4 weeks							
Negative	4 (36.36%)	6 (54.55%)	1 (10%)	0 (0%)	0 (0%)	11 (21.15%)	0.003*
Positive	7 (63.64%)	5 (45.45%)	9 (90%)	10 (100%)	10 (100%)	41 (78.85%)	

Table 5 compares KOH microscopy status across treatment groups. At 2 weeks, all patients in all groups remained KOH positive (100%). However, at 4 weeks, conversion to KOH negativity was highest in the I group (54.55%) followed by T (36.36%), whereas none of the patients in K and F groups achieved mycological cure (p=0.003). This finding further supports better antifungal efficacy of itraconazole and terbinafine.

Table 6: Comparison of side effects between drugs T, I, G, K and F

Side effects	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
At 2 weeks							
Nil	11 (100%)	11 (100%)	10 (100%)	10 (100%)	10 (100%)	52 (100%)	NA
At 4 weeks							
Nil	10 (90.91%)	9 (81.82%)	10 (100%)	2 (20%)	9 (90%)	40 (76.92%)	0.0002*
GIT	0 (0%)	1 (9.09%)	0 (0%)	3 (30%)	1 (10%)	5 (9.62%)	
KFT derangement	0 (0%)	0 (0%)	0 (0%)	3 (30%)	0 (0%)	3 (5.77%)	
LFT derangement	0 (0%)	1 (9.09%)	0 (0%)	2 (20%)	0 (0%)	3 (5.77%)	
Maculopapular rash	1 (9.09%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1.92%)	

Table 6 presents the adverse effect profile. At 2 weeks, no side effects were reported in any group. At 4 weeks, adverse effects were significantly more frequent in the K group, with gastrointestinal symptoms (30%), KFT derangement (30%), and LFT derangement (20%) (p=0.0002). The I group showed mild gastrointestinal and hepatic derangements in a small proportion of patients, while T and G groups demonstrated minimal side effects, indicating better tolerability.

Table 7: Comparison of relapse between drugs T, I, G, K and F

Relapse	T (n=11)	I (n=11)	G (n=10)	K (n=10)	F (n=10)	Total	P value
Absent	3 (27.27%)	5 (45.45%)	0 (0%)	0 (0%)	0 (0%)	8 (15.38%)	0.004*
Present	8 (72.73%)	6 (54.55%)	10 (100%)	10 (100%)	10 (100%)	44 (84.62%)	
Total	11 (100%)	11 (100%)	10 (100%)	10 (100%)	10 (100%)	52 (100%)	

Table 7 shows relapse rates among treatment groups. Relapse was significantly lower in the I group (54.55%) and T group (72.73%) compared to G, K, and F groups, where relapse was observed in 100% of patients (p=0.004). Overall, itraconazole showed the lowest relapse rate, suggesting superior sustained clinical response.

Table 8: MIC range, MIC50 and MIC90 for dermatophyte species (TR and TM)

Species	Measure	Fluconazole	Griseofulvin	Itraconazole	Ketoconazole	Terbinafine
TR	Range	1.625-32	0.125-8	0.125-1	0.25-16	0.125-8
	MIC50	16	2	0.125	8	2
	MIC90	32	8	0.5	16	8
TM	Range	16-16	0.125-4	0.0625-2	2-16	0.125-2
	MIC50	16	1	0.06	4	0.5
	MIC90	16	2	0.5	16	1

Table 8 compares MIC ranges, MIC50, and MIC90 values for different antifungal agents against *Trichophyton rubrum* (TR) and *Trichophyton mentagrophytes* (TM). For TR isolates, terbinafine and itraconazole demonstrated lower MIC50 values (2 µg/mL and 0.125 µg/mL respectively), indicating higher in vitro susceptibility, whereas fluconazole showed higher MIC50 and MIC90 values (16 µg/mL and 32 µg/mL). Similarly, TM isolates exhibited lower MIC50 values for itraconazole (0.06 µg/mL) and terbinafine (0.5 µg/mL), while fluconazole showed poor susceptibility with MIC50 of 16 µg/mL. These findings correlate with clinical outcomes, where itraconazole and terbinafine demonstrated superior efficacy and lower relapse rates.

DISCUSSION

Antifungal treatment failure has been reported with increasing frequency, largely due to the emergence of resistant dermatophyte strains.[4] Despite this concern, published data on antifungal susceptibility patterns from India remain sparse.[5] Antifungal susceptibility can be assessed using several standardized in-vitro techniques, including broth macro- and microdilution, agar dilution, and disc diffusion methods.[5]

This study presents a combined epidemiological and mycological assessment of tinea corporis, with particular focus on clinicomycological correlation and antifungal susceptibility profiles.

Dermatophytosis affected individuals across all age groups, with maximum cases observed in the 21–30-year age bracket, a trend similar to that reported in other Indian studies.[6-8].

Increased physical activity, frequent sweating, and the use of occlusive synthetic garments may predispose this age group to infection. A male preponderance was observed, consistent with earlier reports,[6-8] although recent literature indicates a gradual increase in female cases, possibly reflecting changing lifestyle patterns and improved access to dermatological care.[9,10]

Recurrent disease was documented in one-fourth of patients, comparable to the observations of Kaur et al.,[11]but markedly lower than the high recurrence rates described by Tahiliani et al.[8]A positive family history was recorded in 78.85% of cases, exceeding proportions reported in previous studies.[9,11-13]This finding emphasizes the importance of intrafamilial transmission, which is often facilitated by overcrowding, sharing of clothing, and exposure to contaminated fomites. Notably, a large majority of patients in the present study reported sharing clothes within the household, further supporting this mode of transmission.

Only patients demonstrating thin, septate hyphae on potassium hydroxide (KOH) microscopy were included in the analysis. Reported KOH positivity rates in the literature vary between 80% and 92%, with comparable findings documented by multiple Indian authors.[8,14-17]

Historically, Trichophyton rubrum has been regarded as the most common causative organism of dermatophytosis in India.[3,8,18-20] In contrast, our study identified T. mentagrophytes as the predominant species, accounting for 59.62% of isolates, while T. rubrum comprised 40.38%. This changing etiological pattern aligns with recent reports from different regions of the country.[15,21,22]

The observed shift in species dominance may be attributed to multiple factors, including widespread misuse of topical corticosteroid combinations, indiscriminate antifungal consumption, environmental influences, host immune variations, and differences in keratin affinity among dermatophyte species.[8]

Among the five antifungal agents tested, fluconazole and ketoconazole demonstrated higher MIC values. In our study, fluconazole showed the highest MIC geometric mean, MIC_{50}, and MIC_{90} against Trichophyton rubrum, suggesting an increased risk of clinical failure and relapse. Similar findings have been reported in previous studies.[23.24]

Several studies, including those by Barros et al., Ghannoum et al., Santosh et al., Das et al., Vanapalli et al., and Maurya et al., have reported elevated minimum inhibitory concentration (MIC) ranges (1–64 µg/mL) for fluconazole against Trichophyton rubrum and Trichophyton mentagrophytes.[17,25-29]

In India, unregulated availability and misuse of antifungal agents appear to be major drivers of fluconazole resistance. Enhanced ABC transporter–mediated efflux is a key mechanism contributing to reduced azole susceptibility. Resistance to triazoles, particularly fluconazole, has been observed in up to 35.4% of clinical isolates, often resulting in poor treatment outcomes.[28]

On the contrary, the MIC-90 values for ketoconazole in the present study were higher than those reported in earlier studies.[23,27-29]

In the present study, the MIC range of griseofulvin against Trichophyton mentagrophytes (0.125–4 µg/mL) was lower than that observed for T. rubrum, findings that are comparable with previous reports.[28] Griseofulvin remained the first-line therapy for dermatophytosis for several decades prior to the introduction of azoles, and its prolonged use may have contributed to the higher MIC values reported in recent years. Increasing treatment failure with griseofulvin subsequently led to the preferential use of allylamines.[28] However, Poojary et al. reported lower MIC ranges for griseofulvin against T. rubrum (0.03–1 µg/mL) and T. mentagrophytes (0.06–1 µg/mL).[23] Similarly, Tahiliani et al. observed low MIC values for griseofulvin (0.25–3.0 µg/mL).[8]

Itraconazole demonstrated the lowest MIC values in the present study, with slightly higher MICs for T. mentagrophytes (0.0625–2 µg/mL) compared with T. rubrum (0.125–1 µg/mL), similar to the findings of Poojary et al.[23] However, itraconazole MIC ranges in our study were higher than those reported by Kumar et al.(0.03–0.125 µg/mL).[30] , Budhiraja et al.[31] (0.03–0.125 µg/mL), and Das et al.[28](0.03–1 µg/mL), indicating a rising trend of in-vitro resistance to itraconazole. In contrast, Badali et al.[32] and Deng et al.[33] reported higher itraconazole MIC values than those observed in the present study. Substitution of squalene epoxidase at Ala448Thr has been associated with elevated itraconazole MICs and the development of triazole insensitivity.[34]

In the present study, the MIC range of terbinafine against T. rubrum (0.125–8 µg/mL) was significantly higher than that observed for T. mentagrophytes (0.125–2 µg/mL). Overall, terbinafine MIC values in our study were higher than those reported in several previous studies.[23,25,27,35,36] However, studies by Bhatia and Sharma and Dabas et al. reported even higher MIC-90 values for terbinafine than those observed in the present study.[37,38]

In the present study, itraconazole showed the highest clinical efficacy at 4 weeks, with 54.55% of patients achieving complete cure and 45.45% showing marked improvement, which was significantly superior to other antifungal agents ($p < 0.0001$). These findings are in concordance with recent Indian studies that have reported clinical cure rates with itraconazole ranging from 50–70% within 4–6

weeks, particularly in chronic and recalcitrant dermatophytosis[39,40]. The superior performance of itraconazole is attributed to its high lipophilicity, strong keratin binding, and prolonged retention in the stratum corneum, resulting in sustained antifungal activity.

Terbinafine demonstrated comparatively lower efficacy in the present study, with 36.36% complete cure and 63.64% marked improvement. Recent literature has reported declining cure rates with terbinafine, varying between 30–50%, largely due to the emergence of terbinafine-resistant Trichophyton mentagrophytes genotype VIII.[39] Mutations in the squalene epoxidase gene have been identified as a major mechanism for this resistance, leading to reduced clinical response despite adequate dosing.[41]

In contrast, patients treated with griseofulvin, fluconazole, and ketoconazole showed poor clinical response in the current study. Similar low cure rates have been reported in recent studies, with fluconazole achieving clinical cure in only 20–30% of cases and griseofulvin in 25–40%, particularly in extensive or chronic infections.[40] Fluconazole's lower keratin affinity and griseofulvin's requirement for prolonged therapy may contribute to these inferior outcomes. Ketoconazole, though effective in vitro, is now rarely preferred due to safety concerns and lower clinical success rates compared to itraconazole.

Overall, recent evidence strongly supports itraconazole as the most effective systemic antifungal agent in the current epidemiological setting of dermatophytosis, especially in regions with high prevalence of terbinafine resistance, which is consistent with the findings of the present study.

Relapse rates differed significantly among treatment groups in the present study ($p = 0.004$). Itraconazole showed the most sustained response, with 45.45% of patients remaining relapse-free, followed by terbinafine (27.27% relapse-free). In contrast, 100% relapse was observed in patients treated with griseofulvin, ketoconazole, and fluconazole. Similar trends have been reported in recent studies, where itraconazole demonstrated lower relapse rates (40–60% relapse-free) owing to its high keratin affinity and prolonged tissue persistence, whereas terbinafine showed higher recurrence rates due to emerging resistance in Trichophyton mentagrophytes. [39,40,41] The universally high relapse rates with griseofulvin and fluconazole further underscore their limited role in achieving durable remission in the current epidemiological scenario.

Tinea corporis often requires prolonged treatment due to the persistence of dermatophytes within keratinized tissues and hair follicles, which act as reservoirs for infection and contribute to relapse if therapy is prematurely discontinued. The emergence of terbinafine-resistant Trichophyton mentagrophytes strains, widespread misuse of topical corticosteroid-antifungal combinations, and suboptimal drug penetration into the stratum corneum further necessitate longer treatment durations to achieve complete mycological clearance. Additionally, high relapse rates reported with shorter regimens highlight the need for extended therapy to ensure sustained remission in the current epidemiological setting.[39,40,41]

No adverse effects were observed in any treatment group at 2 weeks. However, by 4 weeks, a significant difference in tolerability was noted among the antifungal agents ($p = 0.0002$). Terbinafine, itraconazole, griseofulvin, and fluconazole were generally well tolerated, with more than 80–100% of patients remaining free of adverse effects. In contrast, ketoconazole demonstrated poor tolerability, with 80% of patients developing adverse effects, including gastrointestinal intolerance, hepatic dysfunction, and renal impairment. Similar safety profiles have been reported in recent studies, where itraconazole and terbinafine were associated with low rates of mild, reversible adverse events, while systemic ketoconazole showed a significantly higher risk of hepatotoxicity and systemic toxicity, leading to its restricted use in dermatophytosis.[40,41,42] The findings of the present study further reinforce the limited role of ketoconazole in the management of tinea corporis.

In the present study, itraconazole showed the highest clinical efficacy in tinea corporis, with all treated patients responding to therapy. Antifungal susceptibility testing revealed that itraconazole had the lowest MIC range, MIC_{50}, and MIC_{90}, indicating superior in-vitro activity. A statistically significant difference in sensitivity was observed for itraconazole compared with terbinafine, griseofulvin, fluconazole, and ketoconazole, suggesting a strong association between lower MIC values and favorable clinical response. Terbinafine demonstrated a clinical response in 66.7% of patients, with isolates of Trichophyton mentagrophytes showing MIC values between 0.125–0.5 µg/mL. Griseofulvin was less effective, achieving clinical cure in only 20% of cases, and response was limited to isolates with lower MIC values, whereas non-responders exhibited higher MIC ranges. No clinical response was observed with fluconazole or ketoconazole, which correlated with their consistently higher MIC values. Overall, itraconazole showed the greatest sensitivity, followed by terbinafine and griseofulvin, against T. mentagrophytes and T. rubrum. These findings support a positive correlation between antifungal susceptibility and clinical outcome, underscoring the clinical relevance of MIC testing in the management of tinea corporis.

CONCLUSION

This study offers an in-depth evaluation of the epidemiological, clinicomycological, and antifungal susceptibility characteristics of tinea corporis in the setting of the current dermatophytosis surge in India. A clear shift in etiological pattern was identified, with Trichophyton mentagrophytes emerging as the predominant pathogen, replacing the traditionally dominant T. rubrum. This evolving species distribution likely reflects the cumulative impact of indiscriminate antifungal use, widespread application of topical corticosteroid combinations, host and environmental factors, and adaptive fungal mechanisms.

Among the antifungal agents evaluated, itraconazole consistently demonstrated superior in-vitro activity, evidenced by the lowest MIC ranges against both dermatophyte species, and translated into the highest clinical and mycological cure rates with minimal relapse and acceptable tolerability. Terbinafine showed moderate effectiveness, whereas fluconazole and ketoconazole exhibited markedly elevated MIC values, poor clinical response, and higher rates of adverse effects, thereby limiting their utility in the current therapeutic landscape. Importantly, a strong concordance between low MIC values and favorable clinical outcomes underscores the relevance of antifungal susceptibility testing in routine practice.

The findings highlight the growing importance of MIC-guided antifungal therapy to optimize treatment selection, improve therapeutic outcomes, and mitigate recurrence in dermatophytosis. Although constrained by a limited sample size and short follow-up duration, this study contributes valuable region-specific susceptibility data. Future multicentric studies incorporating molecular resistance profiling and extended follow-up are essential to inform evidence-based guidelines and address the escalating challenge of antifungal resistance in dermatophyte infections.

Limitations of the study

1. The study was conducted on a relatively small sample size (n=52), which may limit the generalizability of the findings.
2. The single-center design may not reflect regional variations in dermatophyte species distribution and antifungal resistance patterns.
3. Molecular characterization of dermatophyte species and resistance mechanisms was not performed, which could have provided deeper insights into emerging resistant strains.
4. The follow-up duration was limited to four weeks, restricting long-term assessment of sustained cure and delayed relapse.
5. Patient compliance and environmental reinfection factors could not be completely controlled, which may have influenced relapse rates.
6. Serum drug level monitoring was not undertaken to assess pharmacokinetic variability among patients.

REFERENCES

1. Kaaman T. The clinical significance of cutaneous reactions to trichophytin in dermatophytosis. Acta Derm Venereol. 1978;58:139–43.
2. Rajamohanan R, Raj R, Chellam J, Rengasamy M. Epidemiological Trends and Clinicomycological Profile of Chronic Dermatophytosis: A Descriptive Study From South India. Indian J Dermatol. 2021 Jul-Aug;66(4):445.
3. Saxena V, Shenoy MM, Devrari JC, Pai V, Agrawal V. A mycological study of tinea corporis: A changing epidemiological trend from Trichophyton rubrum to Trichophyton mentagrophytes in India. Indian J Dermatol Venereol Leprol 2020;86:607
4. Sacheli R, Hayette MP. Antifungal Resistance in Dermatophytes: Genetic Considerations, Clinical Presentations and Alternative Therapies. J Fungi (Basel). 2021 Nov 18;7(11):983
5. Dogra S, Shaw D, Rudramurthy SM. Antifungal drug susceptibility testing of dermatophytes: Laboratory findings to clinical implications. Indian Dermatol Online J 2019;10:225-33
6. Dalei SR, Nayak D, Bhue PK, Das NR, Behera B. Current status of dermatophytosis: A Hospi tal-Based Study in Northern Odisha, India. Cu reus. 2023 Nov 11;15(11):e48664.
7. Sudhaharan S, Shaw D, Naaz S, Kovida S, Pamidimukkala U. Clinical and Microbiological Spectrum of Dermatophytosis From a Tertiary Care Institute. Cureus. 2025 Jan
8. Tahiliani S, Saraswat A, Lahiri AK, Shah A, Hawelia D, Shah GK, et al. Etiological prevalence and antifungal sensitivity patterns of dermatophytosis in India – A multicentric study. Indian J Dermatol Venereol Leprol 2021;87:800-6
9. Vineetha M, Sheeja S, Celine MI, Sadeep MS, Palackal S, Shanimole PE, Das SS. Profile of dermatophytosis in a tertiary care center in Ker ala, India. Indian journal of dermatology. 2019 Jul 1;64(4):266-71.
10. Patro N, Panda M, Jena AK. The menace of su perficial dermatophytosis on the quality of life of patients attending referral hospital in Eastern India: A cross-sectional observational study. In dian Dermatology Online Journal. 20 19 May 1;10(3):262-266.
11. Kaur I, Chaudhary A, Singh H. Clinico-microbiological aspects of tinea corporis in North India: emergence of Trichophyton tonsurans. Int J Res Dermatol 2019;5:144-9.
12. Khurana A, Masih A, Chowdhary A, Sardana K, Borker S, Gupta A, Gautam RK, Sharma PK, Jain D. 2018. Correlation of in vitro susceptibility based on MICs and squalene epoxidase mutations with clinical response to terbinafine in patients with tinea
13. Mahajan S, Tilak R, Kaushal SK, Mishra RN, Pandey SS. Clinico-mycological study of dermatophytic infections and their sensitivity to antifungal drugs in a tertiary care center. Indian J Dermatol Venereol Leprol. 2017 Jul-Aug;83(4):436-440.
14. Rather PA, Tilwani MR. Clinico Epidemiological Study of Cutaneous Superficial Fungal Infections: Multicentre Descriptive Study. Natl J Med Res 2025;15(01):9-13.
15. Jegadeesan M, Kuruvila S, Nair S. Clinico-etiological Study of Tinea Corporis: Emergence of Trichophyton mentagrophytes. Int J Sci Stud 2017;5(1):161-165.
16. Bindu V, Pavithran K. Clinicomycological study of dermatophytosis in Calicut. Indian J DermatolVenereol Leprol 2002;68:259-61.
17. Vanapalli S, Turpati NR, Gopal KV, Krishnam Raju PV, Devi BG. A Clinico- mycological, antifungal drug sensitivity and therapeutic study of extensive dermatophytosis in coastal Andhra Pradesh. Indian Dermatol Online J 2022;13:747-53

18. Naglot A, Shrimali DD, Nath BK, Gogoi HK, Veer V, Chander J, et al. Recent trends of dermatophytosis in Northeast India (Assam) and interpretation with published studies. Int J Curr Microbiol Appl Sci 2015;4:111-20.
19. Kucheria M, Gupta SK, Chhina DK, Gupta V, Hans D, Singh K. Clinicomycological profile of dermatophytic infections at a tertiary care hospital in North India. Int J Commun Health Med Res 2016;2:17-22.
20. Ramaraj V, Vijayaraman RS, Rangarajan S, Kindo AJ. Incidence and prevalence of dermatophytosis in and around Chennai, Tamilnadu, India. Int J Res Med Sci 2016;4:695-700
21. Verma SB, Panda S, Nenoff P, Singal A, Rudramuruthy SM, Uhrlass S, et al. The unprecedented epidemic-like scenario of dermatophytosis in India: I. Epidemiology, risk factors and clinical features. Indian J Dermatol Venereol Leprol. 2021;87:154–75.
22. Kumar P, Ramachandran S, Das S, Bhattacharya SN, Taneja B. Insights into Changing Dermatophyte Spectrum in India Through Analysis of Cumulative 161,245 Cases Between 1939 and 2021. Mycopathologia. 2023 Jun;188(3):183-202
23. Poojary S, Miskeen A, Bagadia J, Jaiswal S, Uppuluri P. A Study of In vitro Antifungal Susceptibility Patterns of Dermatophytic Fungi at a Tertiary Care Center in Western India. Indian J Dermatol. 2019 Jul-Aug;64(4):277-284
24. Pathania S, Rudramurthy SM, Narang T, Saikia UN, Dogra S. A prospective study of the epidemiological and clinical patterns of recurrent dermatophytosis at a tertiary care hospital in India. Indian J Dermatol Venereol Leprol. 2018;84:678–84
25. da Silva Barros ME, de Assis Santos D, Hamdan JS. Evaluation of susceptibility of Trichophyton mentagrophytes and Trichophyton rubrum clinical isolates to antifungal drugs using a modified CLSI microdilution method (M38-A) J Med Microbiol. 2007;56:514–8. doi: 10.1099/jmm.0.46542-0. [DOI] [PubMed] [Google Scholar]
26. Santosh DA, Barros MES, Hamdan JS. Establishing a method of inoculum preparation for susceptibility testing of Trichophyton mentagrophytes and Trichophyton rubrum. Mycoses. 2005;48(5):351–356.
27. Santos DA, Hamdan JS. Evaluation of broth microdilution antifungal susceptibility testing conditions for Trichophyton rubrum. J Clin Microbiol. 2005;43:1917–20
28. Das, Sudeshna; Rawat, Deepti; Kaur, Ravinder; Mendiratta, Vibhu1; Singh, Pradeep K.2. Comparative Analysis of In vitro Susceptibility Profile of Dermatophytes Against 8 Antifungal Agents: A Cross-Sectional Study. Indian Journal of Dermatology 70(3):p 117-124, May–Jun 2025.
29. Maurya VK, Kachhwaha D, Bora A, Khatri PK, Rathore L. Determination of antifungal minimum inhibitory concentration and its clinical correlation among treatment failure cases of dermatophytosis. J Family Med Prim Care. 2019 Aug 28;8(8):2577-2581.
30. Kumar MB, Gulati N, Chander J, Singla N, Bhalla M, Nayyar C, et al. Species Distribution and Antifungal Susceptibility Profile of Dermatophytes from a Tertiary Care Centre in North India. J Lab Physicians 2022;14:449–55.
31. Budhiraja RK, Sharma S, Sharma S, Kaur J, Bassi R. Antifungal susceptibility pattern of dermatomycosis in a tertiary care hospital of North India. Int J Res Dermatol 2018;4:240.
32. Badali H, Mohammadi R, Mashedi O, de Hoog GS, Meis JF. In vitro susceptibility patterns of clinically important trichophyton and epidermophyton species against nine antifungal drugs. Mycoses 2015;58:303–7.
33. Deng S, Zhang C, Seyedmousavi S, Zhu S, Tan X, Wen Y, et al. Comparison of the in vitro activities of newer triazoles and established antifungal agents against Trichophyton rubrum. Antimicrob Agents Chemother 2015;59:4312–4.
34. Ebert A, Monod M, Salamin K, Burmester A, Uhrlaß S, Wiegand C, et al. Alarming India-wide phenomenon of antifungal resistance in dermatophytes:A multicentre study. Mycoses 2020;63:717–28.
35. Indira G. In vitro antifungal susceptibility testing of 5 antifungal agents against dermatophytic species by CLSI micro dilution method. Clin Microbial. 2014;3:145.
36. Sowmya N, Appalaraju B, Srinivas CR, Surendran P. Antifungal susceptibility testing for dermatophytes isolated from clinical samples by broth dilution method in a tertiary care hospital. JMR. 2015;1:64–7.
37. Bhatia VK, Sharma PC. Determination of minimum inhibitory concentrations of itraconazole, terbinafine and ketoconazole against dermatophyte species by broth microdilution method Indian J Med Microbiol. 2015;33:533–7
38. Dabas Y, Xess I, Singh G, Pandey M, Meena S. Molecular identification and antifungal susceptibility patterns of clinical dermatophytes following CLSI and EUCAST guidelines? J Fungi. 2017;3:E17
39. Rudramurthy SM, Shankarnarayan SA, Dogra S, Shaw D, Mushtaq K, Paul RA, et al. Mutation in the squalene epoxidase gene of Trichophyton mentagrophytes associated with terbinafine resistance. **Antimicrob Agents Chemother.** 2018;62(5):e02522-17.
40. Nenoff P, Verma SB, Ebert A, Süß A, Fischer E, Auerswald E, et al. Spread of terbinafine-resistant Trichophyton mentagrophytes type VIII—A new challenge in dermatophytosis**. Mycoses.** 2020;63(10):1021-7.
41. Khurana A, Sardana K, Chowdhary A. Antifungal resistance in dermatophytosis: Recent trends and therapeutic implications**. Fungal Genet Biol.** 2019;132:103255.
42. Verma S, Madhu R. The changing face of dermatophytosis in India: A current perspective**..** 2017;62(3):227-36.