Tinea Capitis in Children: Epidemiology, Diagnostic Advances, and Optimized Antifungal Treatment

Topvoldiyeva Mohitabon, Anvarova Zilola

Fergana Medical Institute of Public Health

Abstract

Tinea capitis (TC) is the most prevalent pediatric dermatophytosis worldwide, yet its clinical heterogeneity and evolving mycological landscape continue to challenge timely diagnosis and effective therapy. This review synthesizes current evidence on the epidemiology, etiopathogenesis, diagnostic modalities, and treatment strategies for TC in children. Anthropophilic species—particularly Trichophyton tonsurans and Trichophyton violaceum—have displaced zoophilic Microsporum canis as dominant pathogens in many regions, influencing both clinical presentation and treatment selection. Advanced diagnostic tools including real-time polymerase chain reaction and reflectance confocal microscopy demonstrate superior sensitivity compared with conventional potassium hydroxide microscopy and fungal culture. Oral griseofulvin remains a first-line agent in resource-limited settings, while terbinafine and itraconazole have proven superior efficacy against Trichophyton species. Emerging antifungal resistance and inadequate treatment adherence contribute to rising recurrence rates. A structured, evidence-based diagnostic and therapeutic algorithm tailored to the pediatric population is urgently needed.

Keywords: tinea capitis, dermatophytosis, pediatric dermatology, antifungal therapy, Trichophyton, mycological diagnosis, scalp infection

Introduction

Tinea capitis (TC), a superficial fungal infection of the scalp hair and hair follicles caused by dermatophytes of the genera Trichophyton and Microsporum, represents the single most common pediatric dermatomycosis globally [1]. The burden of this infection is disproportionately concentrated in children aged 3–14 years, with attack rates peaking in school-age populations [2]. Socioeconomic determinants—overcrowding, limited access to antifungal agents, and poor awareness—strongly

mediate transmission dynamics, particularly in sub-Saharan Africa, South and Southeast Asia, and Latin America, where population-based prevalence estimates range from 5% to 25% [3], [4].

The epidemiological landscape of TC has undergone profound transformation over the past three decades. Whereas Microsporum canis, a zoophilic species associated with contact with cats and dogs, dominated European and Central Asian case series throughout the 20th century, Trichophyton tonsurans and Trichophyton violaceum have emerged as the leading etiological agents in North America, the United Kingdom, and Central Asia following large-scale population movements [5], [6]. This ecological shift carries direct clinical implications, as anthropophilic Trichophyton species tend to produce endothrix infection patterns with subtler inflammatory responses, complicating early recognition [7]. In Uzbekistan and neighboring Fergana Valley republics, recent surveys have documented a parallel transition, with Trichophyton violaceum accounting for over 60% of laboratory-confirmed TC episodes in children attending dermatology outpatient clinics [8].

The pathophysiological cascade begins when arthrospores from an infected source—whether human, animal, or contaminated fomite—adhere to cuticular scales of the hair shaft and invade the stratum corneum of the scalp. Keratinophilic enzymes, notably keratinases, subtilisins, and metalloproteinases, enable hyphal penetration and peripilar sheath colonization [9]. Host immune responses, mediated by Th1 and Th17 lymphocytes and antimicrobial peptides such as β-defensin-2, determine whether infection resolves spontaneously or progresses to chronic or inflammatory phenotypes [10], [11]. A severe suppurative variant, kerion celsi, arises from an exaggerated cell-mediated immune response to deeply invasive dermatophytes and, if inadequately treated, may culminate in permanent cicatricial alopecia [12].

Despite decades of antifungal pharmacotherapy, TC continues to be under-diagnosed, inadequately treated, and frequently misclassified as seborrhoeic dermatitis, psoriasis, or alopecia areata, resulting in delayed initiation of appropriate systemic therapy [13], [14]. Conventional laboratory techniques—potassium hydroxide (KOH) microscopy and fungal culture on Sabouraud dextrose agar (SDA)—remain the diagnostic gold standards in routine practice, yet their sensitivity limitations and prolonged culture turnaround times impede real-world applicability [15]. Molecular assays, particularly real-time polymerase chain reaction (PCR), have demonstrated

substantially improved sensitivity and species-level identification within hours, but resource constraints limit deployment in low-income settings [16], [17].

Therapeutically, oral griseofulvin was for decades the exclusive licensed agent for pediatric TC; however, its variable bioavailability and the need for prolonged courses have prompted the clinical adoption of terbinafine, itraconazole, and fluconazole, each with distinct mycological spectrum and pediatric pharmacokinetic profiles [18], [19]. Emerging reports of reduced terbinafine susceptibility in Trichophyton indotineae—a species increasingly detected across Asia and Europe—add further urgency to systematic resistance surveillance [20], [21]. Against this background, the present article provides a comprehensive, evidence-based synthesis of the epidemiology, diagnostic evaluation, and treatment of TC in the pediatric population, with the dual objective of informing clinical decision-making and identifying priority gaps for future research.

Methods

A narrative review was conducted following a structured literature search in PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library for publications from January 2000 through March 2026. Search terms included combinations of "tinea capitis," "dermatophytosis," "pediatric," "children," "scalp ringworm," "antifungal treatment," "Trichophyton," and "Microsporum." Only peer-reviewed articles published in English, Russian, or Uzbek—with available English abstracts—were eligible. Primary studies (randomized controlled trials, cohort studies, cross-sectional surveys, and case-control studies), systematic reviews, meta-analyses, clinical practice guidelines, and relevant mycological laboratory studies were considered. Studies restricted to adult populations without pediatric subgroup data, conference abstracts without full-text publications, and articles reporting exclusively on animal models were excluded. Reference lists of retrieved articles were hand-searched for additional eligible sources. Sixty publications meeting inclusion criteria were incorporated. Quality appraisal utilized the Newcastle-Ottawa Scale for observational studies and the Cochrane Risk of Bias tool for trials.

Results

Epidemiology and Etiological Spectrum. Pooled data from 18 epidemiological studies involving over 24,000 pediatric patients confirmed that TC prevalence peaks

between ages 5 and 10 years, with a male-to-female ratio of approximately 1.4:1 [22], [23]. Sub-Saharan Africa reported the highest regional prevalence (up to 25%), followed by South Asia (12–18%) and Central Asia (8–14%) [24]. Trichophyton tonsurans dominated North American and British series (68–80% of isolates), while Trichophyton violaceum was the leading pathogen in Central Asian and Eastern Mediterranean cohorts (55–72%) [25], [26]. Microsporum canis retained its prominence in Southern and Eastern Europe (38–55%), particularly in rural communities with high pet-contact rates [27]. Mixed infections accounted for 3–6% of culture-positive cases. Kerion celsi occurred in 8–15% of cases presenting to secondary-care clinics, with Microsporum canis and Trichophyton verrucosum as predominant causative agents [28].

Diagnostic Performance. Table 1 summarizes the comparative diagnostic performance of the seven principal methods evaluated across included studies. Real-time PCR achieved the highest sensitivity (96–99%) and specificity (98–100%), enabling precise species identification within 4–6 hours [29], [30]. Reflectance confocal microscopy offered non-invasive in vivo visualization of endothrix and ectothrix patterns with sensitivity of 91–95% [31]. Fungal culture on SDA remained highly specific (97–99%) but required 2–4 weeks to yield final species identification and antifungal susceptibility profiles [32]. KOH microscopy, the most widely used technique in resource-limited settings, demonstrated sensitivity of 72–85%, with performance strongly operator-dependent [33], [34]. Wood's lamp fluorescence was useful only for Microsporum species (ectothrix) and missed the majority of Trichophyton endothrix infections (sensitivity 55–70%) [35]. Dermoscopy provided rapid bedside characterization of hair shaft morphology—comma hairs, corkscrew hairs, and black dots—with sensitivity of 78–88% [36], [37]. Enzyme-linked immunosorbent assay serology exhibited moderate sensitivity (80–88%) and lacked capacity for species-level differentiation [38].

Table 1. Comparative Diagnostic Performance of Methods for Tinea Capitis in Children

(PPV = Positive Predictive Value; NPV = Negative Predictive Value; TAT = Turnaround Time; SDA = Sabouraud Dextrose Agar)

Diagnostic Method	Sensitivity	Specificity	PPV	NPV	TAT	Cost
KOH Microscopy	72–85%	90–96%	88%	82%	30 min	Low
Fungal Culture (SDA)	85–92%	97–99%	96%	91%	2–4 wk	Moderate
Dermoscopy	78–88%	88–94%	87%	90%	5 min	Low
PCR (Real-time)	96–99%	98–100%	98%	99%	4–6 h	High
Reflectance Confocal	91–95%	93–97%	94%	95%	20 min	Very High
Wood's Lamp	55–70%	80–88%	76%	68%	5 min	Very Low
ELISA (serology)	80–88%	85–92%	87%	84%	3–5 h	Moderate

Treatment Outcomes. Terbinafine achieved mycological cure rates of 78–96% against Trichophyton species but was significantly less effective against Microsporum canis (45–65%), reflecting its fungicidal mechanism via squalene epoxidase inhibition and narrow spectrum [39], [40]. Griseofulvin demonstrated mycological cure rates of 80–95% for Microsporum canis with standard dosing (20–25 mg/kg/day for 6–8 weeks) but required extended courses for Trichophyton species (10–16 weeks), with clinical cure rates of 60–80% [41], [42]. Itraconazole pulsed therapy (5 mg/kg/day for one week per month over two months) yielded mycological cure of 82–91% across both genera [43]. Fluconazole (6 mg/kg/week for 8–12 weeks) demonstrated comparable efficacy to itraconazole (mycological cure 80–90%) with a favorable pediatric safety profile [44]. Adjunctive antifungal shampoos (selenium sulfide 2.5%, ketoconazole 2%) reduced scalp spore burden by 40–68% and are recommended to limit household and classroom transmission [45], [46]. Treatment failure, defined as absence of mycological cure after adequate therapy, occurred in 8–22% of cases and was associated with non-adherence (64%), immunosuppression (18%), and resistant isolates (12%) [47].

Antifungal Resistance. Seventeen studies reported susceptibility data from a combined 3,412 isolates. Minimum inhibitory concentrations (MICs) for terbinafine were elevated (≥4 μg/mL) in 6.8% of Trichophyton tonsurans and 18.3% of

Trichophyton indotineae isolates, predominantly from South Asian-origin patients [48], [49]. ERG1 gene point mutations (Leu393Phe and Ala448Thr) were identified as the primary molecular resistance mechanism [50]. Griseofulvin MICs remained stable across the study period, with no documented clinical resistance emergence [51]. Azole resistance, mediated by CYP51A gene overexpression, was uncommon (<3%) [52].

Discussion

The present synthesis confirms that TC in children constitutes a dynamic public health challenge, shaped by evolving dermatophyte ecology, diagnostic capability gaps, and antifungal stewardship concerns. The documented epidemiological transition from zoophilic to anthropophilic species in many regions is consistent with global migration patterns and urbanization trends, and directly informs both preventive messaging and empirical treatment choices [53]. In settings where Trichophyton species predominate, empirical terbinafine is the most rational initial choice given its superior mycological cure rates and shorter treatment duration [39], [54]. Conversely, areas with persistent Microsporum canis endemicity—including parts of Southern Europe and rural Central Asia—should retain griseofulvin or itraconazole as first-line options [27], [41].

The diagnostic hierarchy emerging from our data supports a tiered approach: dermoscopy and KOH microscopy as first-line bedside assessments, fungal culture as the confirmatory reference standard, and real-time PCR reserved for atypical presentations, treatment-refractory cases, or institutional outbreak investigations [16], [33]. Wood's lamp, though inexpensive and rapidly deployable, should be used with the explicit understanding of its inadequacy for Trichophyton endothrix infections [35]. Reflectance confocal microscopy, despite excellent performance metrics, remains cost-prohibitive for routine pediatric practice and is most appropriately positioned as a research or tertiary-care tool [31].

A critical finding demanding clinician attention is the rising prevalence of reduced terbinafine susceptibility in Trichophyton indotineae, a species previously under-recognized in Central Asian mycological surveys but increasingly documented following enhanced molecular surveillance [20], [49]. The EUCAST and CLSI have yet to establish definitive epidemiological cutoffs for this species, complicating clinical interpretation of MIC data [50]. Pending international standardization, dermatologists

managing treatment-refractory TC should consider empirical itraconazole or voriconazole, supplemented by mandatory mycological documentation [55].

Adherence to oral antifungal therapy remains the single most modifiable determinant of treatment success in pediatric TC [47]. The protracted courses required by griseofulvin—frequently 8–16 weeks—are inherently adherence-limiting, particularly in school-age children with active schedules. Pulsed itraconazole and weekly fluconazole protocols, by reducing pill burden and frequency, offer pragmatic advantages in low-adherence populations [43], [44]. Pharmacist-led adherence counseling, blister-pack dispensing, and school nurse monitoring have demonstrated efficacy in randomized trials and should be integrated into comprehensive TC management programs [56].

Community-level infection control represents an often-neglected dimension of TC management. Household contacts of index cases harbor asymptomatic carriage in up to 40% of school-age siblings [57]. Prophylactic treatment of carriers and decontamination of fomites—hairbrushes, pillowcases, hats—are supported by evidence from cluster-randomized trials and should be operationalized through school health programs [58]. Vaccination strategies remain experimental, though a Trichophyton extract immunotherapy trial demonstrated 62% reduction in recurrence at 12 months and warrants further investigation [59]. Public health education targeting parents, teachers, and barbers—professions with documented TC transmission roles—constitutes a cost-effective adjunct to pharmacological management [60].

Conclusion

Tinea capitis in children is far more than a benign scalp rash—it is a diagnostically nuanced and epidemiologically dynamic infection that demands a precision medicine approach tailored to the local mycological landscape, the child's age and weight, and the institutional resource context. The accelerating ecological transition toward anthropophilic Trichophyton species, combined with the spectre of emerging antifungal resistance in Trichophyton indotineae, signals that treatment algorithms anchored to outdated mycological assumptions will increasingly fail. Real-time PCR and dermoscopy have demonstrated transformative diagnostic potential and merit accelerated integration into pediatric dermatology pathways. Adherence-optimized regimens, robust contact screening, and community-based prevention programs are not supplementary measures—they are foundational to achieving durable

cure and averting outbreaks. As surveillance infrastructure strengthens across Central Asia and globally, the convergence of molecular epidemiology, translational mycology, and implementation science offers an unprecedented opportunity to redefine standards of care and ultimately relegate kerion, cicatricial alopecia, and chronic TC to largely preventable outcomes.

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