Abstract

Tinea capitis is a superficial and dermatophytes infection primarily located in the hair follicles and surrounding stratum corneum. The infection is widely disseminated among prepubertal children, difficult to treat due to poor efficacy of available antifungal agents and mimic ailments related to the infection. This study reviewed the current and predominant attributes of tinea capitis including diagnostic measures and therapies against the infection. A Google search was conducted using the key term “Tinea capitis”. The search strategy includes literature reviews, clinical trials, meta-analyses, observational studies and randomized controlled trials. The result showed that Trichophyton tonsurans and Microsporum canis causes the higher number of tinea capitis cases with ectothrix and endothrix as a clinical manifestation. Terbinafine therapy had the highest cure rate in a short period of time against the infection. Non-commercial kits had the highest specificity and sensitivity than real time PCR commercial kits. The mimic ailments related to infection include trichotillomania, alopecia areata, seborrheic dermatitis, atopic dermatitis, psoriasis and bacterial scalp abscess. Allylamines and imidazole are commonly used antifungal agents against infection. Effective antifungal agents, new methods of diagnosis should be employed such as PCR targeting the 28S rDNA gene for the identification and characterization of etiological agents of tinea capitis, and practice good habits of personal and environmental hygiene should be encouraged through health education and promotion activities.

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Hair follicles;
Trichophyton tonsurans;
Microsporum canis;
Prepubertal children;
Terbinafine

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1.0 Introduction

Tinea capitis is a common superficial fungal infection of the scalp hair commonly affecting children and adults occasionally (Alkeswani et al., 2019). Currently, surveys was conducted among elementary school children in Alabama and Ohio, and found a prevalence rate of 11% of tinea capitis (Alkeswani et al., 2019). Tinea capitis is commonly caused by dermatophytes that can use keratin, the substantial component of the hair, and the causative agents of this superficial infection belong to two genera which include Microsporum and Trichophyton species (Alkeswani et al., 2019). In the United States, Microsporum audouinii was the common agent of tinea capitis in the earlier half of the 20th century (Gupta & Summerbell, 2000). Recently, Trichophyton tonsurans is examined to be responsible for almost 95% cases of tinea capitis in the United States (Foster et al., 2004). Favus, ectothrix, or endothrix are the major clinical manifestations of tinea capitis. The hyphae propagate down the hair follicle and invade the hair shaft in the endothrix form and then thrive completely within the hair shaft (Alkeswani et al., 2019). This fungal invasion is caused commonly by Trichophyton violaceum and Trichophyton tonsurans. The hyphae penetrate the hair shaft in the ectothrix form at mid follicle, then the hyphae thrive out of the hair follicle covering the hair surface (Alkeswani et al., 2019). This fungal invasion is predominantly caused by Trichophyton verrucosum, Microsporum canis, Microsporum ferrugineum and Microsporum audouinii. The dermatophytes degenerate and leaving long tunnels within the hair shaft when the hyphae thrive parallel to the hair shaft in favus pattern. The
favus form of tinea capitis is commonly caused by *Trichophyton schoenleinii* and usually characterized by yellow crust surrounding the hair shafts and form a permanent alopecia (Alkeswani *et al*., 2019). Systemic antifungal therapy is the significant treatment of tinea capitis due to the poor penetration ability of topical antifungal agents. Griseofulvin has been reported recently with high treatment failure against tinea capitis (Alkeswani *et al*., 2019). Effectiveness of griseofulvin and terbinafine are largely dependent on the etiological agents causing the infection. Tinea capitis caused by *Microsporum canis* responded better to griseofulvin with cure rate of 35.1% to 51.1% than terbinafine with the cure rate of 23% to 30.6% while infection caused by *Trichophyton tonsurans* responded better to terbinafine with the cure rate of 47.7% to 56.1% compared to griseofulvin with the cure rate of 34.4% to 36.5% (Jartarkar *et al*., 2022). The patient compliance in children is improves by given itraconazol pulse therapy at a dose of 5 mg/kg/day for a week and two weeks gab between the second and third pulse and that produced complete clinical cure rate of 100% twelve weeks after therapy (Jartarkar *et al*., 2022). Shampoos such as zinc pyrithrone, selenium and ciclopirox used twice weekly and with addition of systematic treatment, are reported as a preventive measure to treat asymptomatic carriers (Jartarkar *et al*., 2022).

2.0 Materials and Methods

This research work follow the steps proposed by Moher *et al*. (2009) and Cooper (2009) through selecting and evaluating the sources of information, integrating and analyzing the results of the studies. Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses recommended by PRISMA.

2.1 Sources of Information

A search for studies that comprise the superficial mycoses precisely tinea capitis from October 2021 to June 2022 was conducted in databases that include indexed journals, journals that publish fungal infections (superficial mycoses) and the studies cited in each of the articles selected for this study were also searched again. In the search for information, only English keywords were used. The databases evaluated were Science Direct, Scielo, Web of Science, Scopus and the publications from all years included in the databases were evaluated. In the article search, the tinea capitis as a keywords were commonly used.

3.0 Results

Table 1 is the clinical attributes of tinea capitis due to *Microsporum canis* and *Trichophyton tonsurans*. The result revealed that *Trichophyton tonsurans* causes the larger number of tinea capitis cases in Central America and United States while *Microsporum canis* is the predominant dermatophyte causes tinea capitis in Western Europe, Africa, South America and Australia.

The clinical manifestations of tinea capitis reported in Africa include ectothrix or endothrix while clinical manifestation of tinea capitis reported in the United States and Central America is endothrix. Table 2 is the result of numerous clinical trials comparing griseofulvin and terbinafine therapy against tinea capitis. The result shows that terbinafine therapy had 39% to 94% cure rate with minimum of 14 to 28 days while griseofulvin therapy had 39% to 92% cure rate with minimum of 28 to 56 days across reported regions. Table 3 is the synopsis of the studies comparing specificity and sensitivity between in-house PCR methods and real time PCR commercials kits. The result shows that commercial kits had specificity of 74% to 75% and sensitivity of 80% to 89% respectively. Non-commercial kits revealed that specificity is ranging from 90% to 100% while sensitivity is ranging from 97% to 99% respectively. Table 4 is the differential diagnosis of tinea capitis. The result revealed the most common mimic ailments related to tinea capitis such as trichotillomania, alopecia areata, seborrheic dermatitis, atopic dermatitis, psoriasis and bacterial scalp abscess from the common distinguishing attributes of tinea capitis which involves cervical and suboccipital lymphadenopathy, one or more patches of alopecia, pruritus, scale, tenderness, erythema and pustules.

Table 5 is the synopsis of topical antifungal agents used against tinea capitis. The result shows that antifungal agents from allylamines and imidazole class is commonly used as topical agent against tinea capitis infections either as cream, power or lotion for 14 to 42 days applying either once or twice daily.

4.0 Discussion

4.1 Etiology

Dermatophytes are categorised as either geophilic (soil), anthropophilic (human), or zoophilic (animal) (Leung *et al*., 2020). Anthropophilic fungal infections of the scalp (Tinea capitis) are the most commonly disseminated worldwide. Anthropophilic fungi include *Microsporum audouinii*, *T. schoenleinii*, *T. tonsurans*, *T. soundanense*, *T. violaceum*, *Epidemophyton floccosum* and *T. rubrum* (Leung *et al*., 2020). Zoophilic fungal infections of the scalp are commonly acquired through direct contact with infected animals such rabbits, cats, puppies, dogs and pet kittens (Leung *et al*., 2020). Zoophilic fungi include *T. mentagrophytes* var. interdigitale, *T. verrucosum*, *M. canis*, *T. mentagrophytes* var. interdigitale, *M. nanum*, *M. distortum*, *M. ferrugineum*, *M. nanum*, *T. mentagrophytes* var. interdigitale, *T. equinum*, and *T. verrucosum* (Leung *et al*., 2020). Geophilic fungus include *M. gypseum* which is uncommonly cause of tinea capitis (Veasey...
and Muzy, 2018). The fungal species vary according to the geographical locations and may change in certain period of time. In adults, Tinea capitis is commonly caused by anthropophilic dermatophyte species. The form of tinea capitis in adults differs from a little, broken-off hairs with small scaling, noticeable only on careful examination, itching is variable and occasionally with painful and severe inflammatory kerion covering most of the infected spot. In all forms of Tinea capitis, the characteristic features include inflammation and partial hair loss. There is a correlation between T-lymphocyte activation, inflammatory responses and recovery (Ndiaye et al., 2022). Adesiji et al. (2019) reported a case-control study to examine the etiological agents and predispose factors of tinea capitis from school pupils and observed that Tinea capitis is more common in children of age between 4 to 11 years. These results apparently concludes that dermatophytosis especially Tinea capitis, is commonly a prepubertal disease which is consistent with the current study (Table 1). Some studies that have been accorded to this conclusions outline that the fatty acids in the sebum produced at puberty may contain some fungistatic features to inhibit the growth of dermatophytes in older children (Emele and Oyeka, 2008).

### 4.2 Epidemiology

Tinea capitis is a common superficial fungal infection of the scalp hair notably affecting prepubescent children and occasionally adults (Alkeswani et al., 2019). Tinea capitis is a common superficial fungal infections across the continents, have a higher incidence in tropical and subtropical regions of the world especially Africa due to the presence of environmental temperature and high humidity. Recently, studies have reported a rising trend in the prevalence and change in the spectrum of Tinea capitis infection along with the isolation of previously uncommon etiological agents (Jartarkar et al., 2022). The rising trend of Tinea capitis could be due to an epidemiological shift in the growth patterns of causative agents providing them with potential of better survival and persistence, enhancing their virulence and pathogenicity, an evolution in the genetic make-up of the fungi, rapid emergence of drug-resistant species due to the common use of inadequate doses of effective antifungal agents. Current studies have shown a recent shift in the clinical isolates from T. rubrum to T mentagrophytes complex and M audounii (Jartarkar et al., 2022). And increasing proportion of T. mentagrophytes complex, mainly T. mentagrophytes var mentagrophytes with increased minimum inhibitory concentrations to the commonly used antifungal agents (Jartarkar et al., 2022). Dogo et al. (2016) showed that the prevalence of Tinea capitis with regards to the age was lower for the age group from 5 to 10 years (42.6%) compared to the age group between 11 to 15 years (50%). Ndiaye et al. (2022) reported the prevalence of Tinea capitis in prepubertal males (35.3%) was lower than that of females (56.7%) while the prevalence of Tinea capitis among the pubertal age between 11 to 15 years was lower in females (20%) compared to males (48.4%). Microsporum canis is commonly responsible for tinea capitis cases in South America, Australia, Africa and Western Europe (Table 1) while Trichophyton violaceum causes the majority of this superficial infection in South Asia and Eastern Europe (Gupta and Summerbell, 2000). A study reported that 6 months to 12 years of age are commonly affected with Tinea capitis and clinical appearance of Tinea capitis is varies, depending on the level of host resistance, degree of inflammatory host response and the type of hair invasion (Hay, 2017). Nenoff et al. (2014) revealed that Trichophyton soudanense is widely disseminated throughout the African countries which is not agreed with the current study (Table 1). Tinea capitis is predominantly affects prepubertal children of African heritage from 3 – 9 years of age in the United States.

### 4.3 Predisposing Factors

Environmental factors which usually predispose children to higher chances of fungal infection include high humidity and temperature. The complex interplay between host, etiological agents, and the environment plays a significant role in the pathogenesis of fungal infections. Studies have shown that anthropophilic T. rubrum is the most predominate isolate, but recently it is being increasingly replaced by T. mentagrophytes and T. interdigitale complex in many regions of the world (Jartarkar et al., 2022). Tinea capitis is commonly spread among family members. Fungal virulence in numerous fungal species of dermatophytes are likely to play a significant role in the resistance or recurrence of infections. After fungal invasion into the stratum corneum, epidermal adhesion occurs within 60 minutes which is facilitated by adhesins present on the fungal cell wall, mediated by fungalysin, proteases, and serine subtilisin which act as a potential immunogenic stimulus and digests the keratin (Jartarkar et al., 2022). Numerous conclusions exist concerning the gender predominance of Tinea capitis which may likely attributed to hair and stratum corneum. Styling and dressing practices such as plaiting, tight hair braiding, use of hair ointments and shaving of the scalp may contribute to disease dissemination. Poor hygiene is another predisposing factor that may support the dissemination of Tinea capitis among prepubertal children. However, older children may not be affected with this factor because of their relatively good hygiene practices when they reach their pubertal ages (Ndiaye et al., 2022).
### Table 1. The clinical attributes of tinea capitis due to the most predominant dermatophytes

<table>
<thead>
<tr>
<th>Dermatophyte</th>
<th>Region of predominant species</th>
<th>Source of infection</th>
<th>Clinical presentation</th>
<th>Most frequent alopecia form</th>
<th>Infectious form</th>
<th>Wood’s lamp exam</th>
<th>Eventually resolves by puberty</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microsporum canis</td>
<td>Western Europe, Africa, South America and Australia</td>
<td>Zoophilic; frequently caused by dogs and cats</td>
<td>Ruptured, scaly, inflamed with hair loss from 2 to 3 mm above the scalp</td>
<td>Scanty and can reach larger diameter</td>
<td>Ectothrix or endothrix</td>
<td>Yellow-green fluorescence with low sensitivity and high specificity</td>
<td>Yes</td>
<td>(Veasey and Muzy, 2018; Alkeswani et al., 2019; Hay, 2017; Elewski, 2000)</td>
</tr>
<tr>
<td>Trichophyton tonsurans</td>
<td>Central America and United States</td>
<td>Anthropophilic</td>
<td>Characterized as black dot with less inflammation and hair loss at scalp level</td>
<td>Small and numerous</td>
<td>Endothrix</td>
<td>No fluorescence</td>
<td>No</td>
<td>(Veasey and Muzy, 2018; Alkeswani et al., 2019; Hay, 2017; Elewski, 2000)</td>
</tr>
</tbody>
</table>

### Table 2. The result of numerous clinical trials comparing griseofulvin and terbinafine therapy against tinea capitis

<table>
<thead>
<tr>
<th>Region</th>
<th>Year</th>
<th>Reported number of patients</th>
<th>Griseofulvin dose (day, mg or kg)</th>
<th>Cure Rate</th>
<th>Days of Treatment of Griseofulvin</th>
<th>Terbinafine dose (day, mg or kg)</th>
<th>Cure rate</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turkey</td>
<td>1999</td>
<td>78</td>
<td>6 – 13 for 56 days</td>
<td>44%</td>
<td>84 days</td>
<td>3 – 6 for 28 days</td>
<td>39%</td>
<td>(Memisoglu et al., 1999)</td>
</tr>
<tr>
<td>China</td>
<td>2011</td>
<td>88</td>
<td>20 for 28 days</td>
<td>84%</td>
<td>56 days</td>
<td>3 – 6 for 28 days</td>
<td>78%</td>
<td>(Deng et al., 2011)</td>
</tr>
<tr>
<td>Pakistan</td>
<td>1995</td>
<td>105</td>
<td>6 – 13 for 56 days</td>
<td>80%</td>
<td>84 days</td>
<td>3 – 6 for 28 day</td>
<td>93%</td>
<td>(Haroon et al., 1995)</td>
</tr>
<tr>
<td>International</td>
<td>2008</td>
<td>1,549</td>
<td>10 – 20 for 42 days</td>
<td>39%</td>
<td>70 days</td>
<td>5 – 8 for 28 days</td>
<td>45%</td>
<td>(Elewski et al., 2008)</td>
</tr>
<tr>
<td>Peru</td>
<td>2000</td>
<td>50</td>
<td>6 – 12 for 56 days</td>
<td>44%</td>
<td>84 days</td>
<td>3 – 6 for 28 days</td>
<td>76%</td>
<td>(Caceres-Rios et al., 2000)</td>
</tr>
<tr>
<td>Canada and South Africa</td>
<td>2001</td>
<td>100</td>
<td>20 for 42 days</td>
<td>92%</td>
<td>84 days</td>
<td>3 – 6 for 14 – 21 days</td>
<td>94%</td>
<td>(Gupta et al., 2001)</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>2001</td>
<td>147</td>
<td>10 for 56 days</td>
<td>57%</td>
<td>84 days</td>
<td>3 – 6 for 28 days</td>
<td>57%</td>
<td>(Fuller et al., 2001)</td>
</tr>
</tbody>
</table>
Table 3. Synopsis of the studies comparing specificity and sensitivity between PCR commercials and non-commercial kits methods

<table>
<thead>
<tr>
<th>Type of Kits</th>
<th>Country</th>
<th>Year</th>
<th>Type of Specimens</th>
<th>Number of Specimens</th>
<th>Specificity</th>
<th>Sensitivity</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Commercials</td>
<td>Senegal</td>
<td>2022</td>
<td>Hair</td>
<td>129</td>
<td>75%</td>
<td>89%</td>
<td>(Ndiaye et al., 2022)</td>
</tr>
<tr>
<td></td>
<td>Belgium</td>
<td>2019</td>
<td>Nail</td>
<td>138</td>
<td>74%</td>
<td>80%</td>
<td>(Hayette et al., 2019)</td>
</tr>
<tr>
<td>Non-commercial</td>
<td>Switzerland</td>
<td>2019</td>
<td>Hair, Nail, Skin</td>
<td>3,052</td>
<td>90%</td>
<td>97%</td>
<td>(Hayette et al., 2019)</td>
</tr>
<tr>
<td></td>
<td>Sweden</td>
<td>2013</td>
<td>Hair, Nail, Skin</td>
<td>202</td>
<td>92%</td>
<td>99%</td>
<td>(Bergman et al., 2013)</td>
</tr>
<tr>
<td></td>
<td>Netherlands</td>
<td>2011</td>
<td>Hair, Nail, Skin</td>
<td>1,437</td>
<td>100%</td>
<td>97%</td>
<td>(Wisselink et al., 2011)</td>
</tr>
</tbody>
</table>

Table 4. Differential Diagnosis of Tinea Capitis

<table>
<thead>
<tr>
<th>Differential Diagnosis</th>
<th>Distinguishing Attributes</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trichotillomania</td>
<td>Hairs of different length, no skin lesions and commonly involves eyebrows and eyelashes</td>
<td>(Kelly, 2012; Durosaro et al., 2009)</td>
</tr>
<tr>
<td>Alopecia areata</td>
<td>Fine miniature hair growth, exclamation point hairs or total loss of hair, no skin lesions but with discrete patches of hair loss, no inflammation and crusting but with possible nail pitting</td>
<td>(Ely et al., 2014; Durosaro et al., 2009)</td>
</tr>
<tr>
<td>Seborrheic dermatitis</td>
<td>Greasy scale with uncommon lymphadenopathy and alopecia, evenly spread across chest, nasolabial folds, postauricular folds, hairline and eyebrows</td>
<td>(Kelly, 2012; Ely et al., 2014)</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>Alopecia, annular and lymphadenopathy are less common, family or personal history of atopy</td>
<td>(Kelly, 2012; Ely et al., 2014)</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>More than 68% of affected individuals have family history of psoriasis with nail pitting, silver or gray scale</td>
<td>(Durosaro et al., 2009; Ely et al., 2014)</td>
</tr>
<tr>
<td>Bacterial scalp abscess</td>
<td>Hair pluck is sore and uncommon alopecia</td>
<td>(Kelly, 2012; Ely et al., 2014)</td>
</tr>
</tbody>
</table>
### Table 5. Synopsis of topical antifungal agents used against Tinea capitis

<table>
<thead>
<tr>
<th>Antifungal class</th>
<th>Antifungal agent</th>
<th>Percentage</th>
<th>Preparations</th>
<th>Uses</th>
<th>Frequency of Application</th>
<th>Duration of Use</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allylamines</td>
<td>Naftifine</td>
<td>1%</td>
<td>Cream</td>
<td>Tinea capitis</td>
<td>Once or twice daily</td>
<td>14 days</td>
<td>(Abdul-Rahman <em>et al.</em>, 2005; Lamisil. Package insert, 2012)</td>
</tr>
<tr>
<td></td>
<td>Terbinafine</td>
<td></td>
<td>Powder or cream</td>
<td>Tinea capitis</td>
<td>Twice daily</td>
<td>14 days</td>
<td>(Jartarkar <em>et al.</em>, 2022; Abdul-Rahman <em>et al.</em>, 2005)</td>
</tr>
<tr>
<td>Imidazoles</td>
<td>Eberconazole</td>
<td>1%</td>
<td>Cream</td>
<td>Tinea capitis</td>
<td>Once daily</td>
<td>14 – 28 days</td>
<td>(Gupta <em>et al.</em>, 1997; Gupta and Cooper, 2008)</td>
</tr>
<tr>
<td></td>
<td>Clotrimazole</td>
<td>1%</td>
<td>Lotion or cream</td>
<td>Tinea capitis</td>
<td>Twice daily</td>
<td>28 – 42 days</td>
<td>(Abdul-Rahman <em>et al.</em>, 2005)</td>
</tr>
<tr>
<td></td>
<td>Luliconazole</td>
<td>1%</td>
<td>Lotion or cream</td>
<td>Tinea capitis</td>
<td>Once daily</td>
<td>14 days</td>
<td>(Jartarkar <em>et al.</em>, 2022)</td>
</tr>
<tr>
<td></td>
<td>Econazole</td>
<td>1%</td>
<td>Cream</td>
<td>Tinea capitis</td>
<td>Once or twice daily</td>
<td>28 – 42 days</td>
<td>(Gupta <em>et al.</em>, 1997)</td>
</tr>
<tr>
<td></td>
<td>Sertaconazole</td>
<td>2%</td>
<td>Cream</td>
<td>Tinea capitis</td>
<td>Twice daily</td>
<td>28 days</td>
<td>(Gupta and Cooper, 2008)</td>
</tr>
<tr>
<td></td>
<td>Miconazole</td>
<td>1%</td>
<td>Lotion or cream</td>
<td>Tinea capitis</td>
<td>Twice daily</td>
<td>28 – 42 days</td>
<td>(Jartarkar <em>et al.</em>, 2022)</td>
</tr>
<tr>
<td></td>
<td>Oxiconazole</td>
<td>2%</td>
<td>Lotion or cream</td>
<td>Tinea capitis</td>
<td>Once or twice daily</td>
<td>28 days</td>
<td>(Abdul-Rahman <em>et al.</em>, 2005; Lamisil. Package insert, 2012)</td>
</tr>
</tbody>
</table>
4.4 Pathogenesis
A human may become infected through direct contact with infected persons, contaminated fomites such as pillow, brush, comb, or cap, asymptomatic carriers, soil, or animals especially house pets (Leung et al., 2020). Direct contact among family members is the most predominant route, children usually become infected by fungal spores shed by individual contact (Michaels and DelRosso, 2012). Mannan glycoproteins present in the fungal cell wall facilitate adherence of the fungal spores to the keratin-containing stratum corneum of the scalp (Leung et al., 2020). The fungus usually invade the scalp due to the enzymes produced such as proteases and keratinase which digest keratin and mediate penetration of keratinized tissue (Leung et al., 2020). Increased epidermal turnover and inflammation is resulted through scaling. From the spot of fungal invasion in the scalp, the fungus thrives along the plane of the skin. The fungus can also migrate from the skin to the hair follicle and to the hair. Depending on the type of hair invasion and spot of formation of the arthroconidia, Tinea capitis can be categorized into three forms which include endothrix, favus, and ectothrix infection (John et al., 2018). The hyphae are converted into arthroconidia within the hair shaft in endothrix fungal infection. Endothrix infection is characterized by formation of arthroconidia within the hair shaft without destroying the cuticle which is usually appeared like a bag of marble in endothrix fungal infection (Leung et al., 2020). It usually results to hair breakage easily close the follicular ostia which is giving rise to the black dot formation (Schechtman et al., 2015). Endothrix infections result from anthropophilic fungi which include *T. tonsurans*, *T. violaceum* and *T. soudanense* (Leung et al., 2020). The etiological agents of Tinea capitis include *M. naum*, *M. canis*, *M. distortum*, *T. verrucosum*, *M. audouinii*, *M. gypseum*, and *M. ferrugineum*. Favus (also known as Tinea favosa) is most commonly caused by *T. schoenleinii* (Farooqi et al., 2014). Fungal hyphae are arranged parallel to the hair shaft and arthroconidia, located within the hair shafts in favus and the air spaces within the hair shafts can be vividly seen (Leung et al., 2020). Infected hairs usually become brittle and can be easily broken off. Zoophilic dermatophytes mediate more severe inflammation when compared to anthropophilic dermatophytes. Kerion is an inflammatory variant of Tinea capitis usually caused by a dramatic immune response to a dermatophytes, most predominantly fungal species include *M. canis* and, less commonly caused by *Aspergillus protuberus*, *T. tonsurans*, *M. gypseum*, *T. violaceum*, *T. mentagrophytes*, and *T. verrucosum* (Leung et al., 2020). Histopathologic characteristics of non-inflammatory Tinea capitis include hyphae and arthroconidia surrounding or within hair shafts, perifollicular mononuclear infiltrate in the dermis and fungi sparsely distributed in the skin (John et al., 2018). If the hair follicles are disrupted, multinucleated giant cells may be present in the stratum corneum along with the degenerated hair follicles (Leung et al., 2020). The features of histopathologic of kerion include a perifollicular inflammatory infiltrate with spongiosis and lymphocytes, infiltrates of neutrophils, and fibrotic scars in later stages and plasma cells in the early stages (Leung et al., 2020). The development of presumed T cell mediated immunity and delayed-type hypersensitivity to causative agents’ antigen correlates with recovery from the Tinea capitis infection (Rasmussen and Ahmed, 1978).

4.5 Management
Griseofulvin has been approved and used as a first-line treatment agent against Tinea capitis since the 1960s in the United State (Alkeswani et al., 2019). Currently, it was reported that substantial therapy failure, long duration and high cost of treatment are major challenges of Tinea capitis infection among children and adults. Griseofulvin is a fungistatic agent produced by numerous mold species such as *Penicillium*. This antifungal agent distorts the contraction of the mitotic spindle by bind microtubules (Alkeswani et al., 2019). Tinea capitis is a major challenge in dermatological consultation because of its common dissemination. In South Africa and Canada, reported the prevalence of Tinea capitis and treated with griseofulvin for 42 days and had a cure rate of 92% (Table 2). Shah et al., (1974) revealed that the griseofulvin reaches the stratum corneum via sweat and its hydrophobic elements permit it to concentrate in hair follicle. After the absorption, griseofulvin concentration is unrecognized in the stratum corneum from 48 to 72 hours, may be as a result of poor penetration ability to keratin and reversible protein binding and allowing for once a day dosing due to the terminal half-life of 10 to 21 hours approximately (Alkeswani et al., 2019). China, reported cases of Tinea capitis from 88 patients in 2011 with 84% cure rate treated with griseofulvin (Table 2). Griseofulvin is an inducer of estrogen and coumarin-type drugs which has negligible drug interactions (Albengres et al., 1998). The liver assimilates almost all of the drug elements via glucuronidation and demethylation reaction. Griseofulvin is included in the list of World Health Organization (WHO) as an essential medicine. Gupta et al. (2017) reported that griseofulvin is currently unavailable in some European countries and Canada and that disagreed with this study (Table 2). Other effective antifungal agents is being researched as an alternative therapy against Tinea capitis. Bennassar and Grimalt, (2010) reported that the new recommended dose is 10 to 15 mg/kg/day for ultramicrosized and 20 to 25 mg/kg/day for microsized
preparations for 42 to 84 days. Treatment should be continued for 14 days even after the clinical cure (Caceres-Rios et al., 2000). Gupta et al. (2018) reported that griseofulvin upholds a 72% complete cure rate of Tinea capitis and this is not consistent with the current study (Table 2). Gupta et al. (2018) reported that terbinafine was among antifungal agents to possessed high complete cure rate of 92% against Tinea capitis infection which is not consistent with the current study. Alkeswani et al. (2019) revealed that griseofulvin is considered as the best for treating Tinea capitis infection which caused by Microsporum species and this present study disagreed with the conclusion on griseofulvin (Table 2). Alkeswani et al. (2019) reported some common adverse effect of griseofulvin which include photosensitivity, gastrointestinal disorders, erythema multiforme, headaches, systemic lupus erythematosus exacerbation, embryotoxic effects in pregnant women, serum sickness-like reaction and men are warned against fathering a child for 6 to 8 months after receiving griseofulvin therapy. Terbinafine (Lamisil®) is an allylamine derivative contain fungicidal features, a notable enzyme in the synthesis of ergosterol, noncompetitive inhibitor of squalene epoxidase and significant agent of fungal cell membranes (Alkeswani et al., 2019). After an oral dose, more than 70% of the drug is quickly absorbed and reaches a highest plasma concentration within 120 minutes and that was among the reasons why terbinafine had the highest cure rate within few days compared to griseofulvin (Table 2 and Table 5). Food intake does not affects its absorption (Gianni, 2010). Most of the drug moves deep to plasma proteins which relates with chylomicrons that allow for a sizable lymphatic dissemination (Krishnan-Natesan, 2009). The features of lipophilic provide the capacity to reach a maximum concentration in adipose tissue, sebum-rich stratum corneum, hair follicles and nail plate. After two weeks of treatment, the concentration of terbinafine in stratum corneum is greater than its plasma concentration and has a terminal half-life of more than 200 hours, allowing once a day dosing (Alkeswani et al., 2019). It is reduced slowly from the stratum corneum and has showed the activity of antifungal agents for 60 days after its depletion from plasma (Elewski, 2000). These unique features of pharmacokinetic provide a distinct merit to terbinafine, allowing shorter courses of treatment (Table 2 and Table 5). Primarily, N-demethylation enzymes is metabolized by the liver, more than 15 metabolites have been reported and none of them have showed antifungal activity (Shafer-Korting et al., 2008). Tey et al. (2011) reported in their studies that four weeks of once a day dose for treating tinea capitis is more effective which is agreed with the present study (Table 2 and Table 5). The duration of terbinafine therapy against tinea capitis is significantly shorter compared to other antifungal agents such as griseofulvin (Table 2). Terbinafine have showed the superiority and effectiveness against Trichophyton violaceum and Trichophyton tonsurans while griseofulvin was more effective against Microsporum canis (Chen et al., 2016). Terbinafine do not improve its effectiveness against Microsporum species even for a longer courses of treatment. Therefore, terbinafine should not be a first line for treating tinea capitis infections caused by Microsporum species (Lipozencic et al., 2002). In pregnancy, terbinafine is the antifungal agent of choice and only systemic antifungal agent used against Tinea capitis infections. Topical agents do not penetrate the hair shaft, therefore systemic antifungal agents must be used in the treatment of tinea capitis. Permanent hair loss and scarring occur when failed to treat kerion promptly (Kelly, 2012).

4.6 Diagnosis

Real-time PCR is a potentially attractive diagnostic method, the detection and amplification of the pathogen DNA is examined in one step. Ndiaye et al. (2022) reported in their studies that the DG PCR assay revealed to be highly specific and sensitive for the identification and detection of dermatophytes directly from clinical specimen. Ndiaye et al. (2022) also reported that culture and microscopy were evaluated as the gold standard and PCR assay shows 89.3% and 75.3% of sensitivity and specificity respectively which is agreed with this study (Table 3). Uhrlab et al., (2019) reported a recent developed microarray test (EuroArray Dermatomycosis, Euroimmun Lubeck, Germany) (EuroArray) and DG PCR were assessed concerning their diagnostic specificity to identify the DNA of causative agents of tinea capitis. The EuroArray Dermatomycosis is a PCR based procedure for identification of 56 fungal species commonly caused hair, nails and skin infections. From the 56 fungal pathogens, 3 mould species, 23 dermatophytes and 3 yeasts can be examined. Recently, Uhrlab et al., (2019) in their studies, compared EuroArray tests and DG PCR concerning their diagnostic specificity to identify the DNA of dermatophytes. The result of the comparison revealed that the microarray dermatomycosis is highly specific when conducted in PCR with subsequent hybridization. Examination at the scanner is rapidly and easy to be conducted. The EuroArray Dermatomycosis detect notably more dermatophyte species than DG PCR which apparently not involve in general dermatophyte detection. The real-time PCR showed a 97% sensitivity, indicating a potential increase in the identification rate for dermatophytes in clinical specimen compared with the culture. The development of a single tube dermatophytes specific qPCR assay based on ITS1 sequences that permits the quick identification and detection of 11 clinically relevant fungal species.
within the 3 dermatophyte genera *Epidermophyton*, *Trichophyton* and *Microsporum* in hair, nail and skin specimen within a few hours (Bergman et al., 2013). The sequencing of the translation elongation factor 1-α (TEF1-α) gene for a differentiation between *Trichophyton violaceum* and *Trichophyton soudanense* were reported by Nenoff et al., (2014) and that indicates a clear distinction is apparently possible. However, the TEF1-α gene may be difficult to differentiate between *Trichophyton rubrum* and *Trichophyton soudanense*. DG PCR can be used as quick test when a clinician demands a precise and rapid diagnosis. It can be used instead of the ITS sequencing which may requires 4 to 5 days with culture before obtaining the results. Real-time PCR is an easy to use molecular technique in the laboratory because the detection of ampiclons is directly detected by a programmed software system related to the thermocycler without need of post PCR steps. The outcomes are rapid and easy to interpret for health professionals trained in molecular biology. Presently, only one other commercial real-time PCR assay has been authenticated for the identification of dermatophytes in nails that can also be used for the detection of dermatophytes in skin and hair samples (Petinaud et al., 2016).

Ndiaye et al. (2022) reported that DG PCR test shows two anthropophilic dermatophytes species, *Microsporum audouinii* and *Trichophyton soudanense*, that were the main causative agents identified from the infection caused by Tinea capitis followed by *Microsporum canis*. Coulibaly et al. (2014) and Ndiaye et al. (2015) reported in their studies that *Trichophyton soudanense* was the most common causative agent of Tinea capitis in Mali, Dakar 2008 and 2013. Related to polyparasitism, there were less coinfections noticed by culture method compared to real-time PCR assays. KOH preparation can be carried out when observing for fungal spores by scraping the black dots or broken hairs. The most common cause of tinea capitis is *Trichophyton tonsurans* and using wood lamp for identification of scalp lesions is usually not producing the desire results or does not fluoresce. Wood lamp examination is helpful to identify *Microsporum canis* in white children through exhibits a green fluorescence (Table 1).

### 4.7 Complications

Kelly, (2012) reported in his studies that the most common mimic infection include alopecia areata, seborrheic dermatitis, bacterial scalp abscess, trichotillomania, psoriasis and atopic dermatitis (Table 4). Total hair loss or patchy is unsightly and socially embarrassing and may have a significant influence on the quality of life, child’s self-esteem, and usually children experienced mild-to-severe psychosocial challenge (Leung et al., 2020). Inappropriately or untreated kerion and favus may result to permanent scarring alopecia and the secondary bacterial infection is also a potential complication of Tinea capitis infection (Leung et al., 2020). Id reaction, also known as dermatophytid reaction, autoeczematization, or disseminated eczema, may occur along with fungal infections especially after starting systemic antifungal therapy. Affected children usually develop widespread, papulovesicles, intensely pruritic, pustules, erythematous, maculopapules, or scaly papules, and the lesions are devoid of fungi (Bennassar and Grimalt, 2010). Occasionally, annular centrifugum and erythema nodosum may occur especially in association with kerion and disseminated systemic disease has been reported in immunocompromised patients (Leung et al., 2020). The spores of *Trichophyton tonsurans* will be noticed commonly within the hair shaft, but the spores of *Microsporum canis* will be observed less commonly coat the outside of the hair shaft. Infected cats and dogs may disseminate *Microsporum* infections and produce high inflammation than *Trichophyton* infections (Moriarty et al., 2012). Affected children with tinea capitis should return for clinical examination at the completion of treatment if notified, however, if there is clinical improvement, follow up cultures are often not necessary. Affected children may return back to school once the tinea capitis therapy has commenced, but within 14 days should not participate in sports that involve head to head contact such as wrestling or share pillowcases, combs, hats, caps, helmets or brushes (Ely et al., 2014). Household members should be clinically examined but not compulsory to be tested for tinea capitis infection (Ely et al., 2014).

### 4.8 Prevention

All family members should be carefully examined and treated simultaneously if Tinea capitis is observed. Playmates and parents of school or house mates should be informed immediately so that their children may be examined and treated effectively. Participation in contact sports, schools, sharing of fomites should be discouraged. Proper and frequent cleaning of fomites such as caps, combs, pillows, hats, brushes, clippers, and hats should be noted. It is advisable for family members to use effective antifungal shampoos during therapy of the infected patients in order to prevent ping pong dissemination between household members (Leung et al., 2020). There is a need to develop an effective technique to educate the population regarding the adverse effects of over the counter antifungal drugs, predisposing factors, need to expert suggestion, benefits of adhering to expert’s advice and effective preventive measures to control drug resistance by use of antifungal drugs, preferring use and in proper doses of combination treatment with different mechanism of action (Jartarkar et al., 2022).
5.0 Conclusion
The most predominant causative agents of tinea capitis are *Trichophyton tonsurans* and *Microsporum canis* which are commonly identified using traditional conventional methods. However, most of these causative agents are wrongly identified and time consuming. There is urgent need for accurate and rapid characterization and identification of the causative agents of tinea capitis and appropriate prescription therapy. Terbinafine might be used as first line treatment against tinea capitis infection because the antifungal agent has showed the efficacy, cost and shorter duration of therapy, tolerability and superiority to griseofulvin with similar adverse events rate. In terms of tinea capitis caused by *Microsporum canis*, terbinafine should not be used as first line agent against the infection. A large randomized controlled study of terbinafine versus griseofulvin is urgently needed to evaluate the best antifungal agent in the treatment of tinea capitis due to *Trichophyton tonsurans* and *Microsporum canis*. Topical antifungal agents alone are not considered to be more effective due to poor penetration ability into the deep root of the hair follicles. However, combination treatment of oral and topical antifungal agents may facilitate the cure rate rapidly. DG PCR have demonstrated the potential performance characteristics for the identification of causative agents of tinea capitis infection and shows faster outcomes compared to culture techniques which make it very promising for routine diagnostics of superficial fungal infections. The DG PCR technique is cheap, rapid and simple compared to other molecular methods for the identification of the causative agents of tinea capitis. Health education and promotion activities via media and posters are importance tools to raise the awareness to the public about tinea capitis infections, mode of dissemination and clinical presentation. Encourage the community, health centers and schools to practice good habits of personal and environmental hygiene. Tinea capitis is the responsibility of the government and community, not the responsibility of the patients alone. Currently, gold standard in the characterization and identification of the causative agents of tinea capitis is PCR targeting the 28S rDNA region. The use of amplified fragments of the 28S-rDNA gene embedded with regions of distinction amongst dermatophytes. The use of specific PCR targeting the 28S rRNA gene provided a potential ability in a confirmatory technique for accurate identification and characterization of dermatophytes causing tinea capitis infection.

**Declarations**

**Ethics approval and consent to participate**
Not Applicable

**Availability of data and material**
Not Applicable.

**Competing interests**
Author declare no competing interests.

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