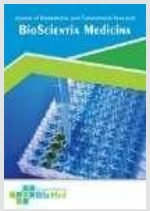
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Gray Patch Tinea Capitis by *Microsporum canis* in a Child: A Case Report Highlighting Environmental Risk Factors and Diagnostic Nuances

Mario Korwa[1*], Luh Made Mas Rusyati[1], Handelia Phinari[1], Andrew Wicaksono[1], Nandya Dwizella[1]

[1]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Udayana/Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Indonesia

ARTICLE INFO

Keywords:
Fomite transmission
Microsporum canis
Pediatric dermatology
Tinea capitis
Trichoscopy

***Corresponding author:**
Mario Korwa

E-mail address:
mariokorwa286@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v9i11.1429

A B S T R A C T

Background: Tinea capitis, a dermatophytosis of the scalp, is a leading cause of hair loss in children. Its successful management hinges on understanding its complex etiology, including host susceptibility and pathogen virulence. *Microsporum canis*, a zoophilic fungus, is a primary causative agent, yet its transmission pathways and diagnostic markers are not fully elucidated. **Case presentation:** A 3-year-old female presented with a two-week history of progressive, pruritic alopecia. Clinical history was notable for the absence of animal contact but revealed significant environmental exposure at a hair salon. Dermatological examination showed multiple, well-demarcated, alopecic patches with fine scaling, characteristic of gray patch tinea capitis. While Wood's lamp examination was negative, trichoscopy revealed comma hairs and Morse code-like hairs, suggesting fungal infection. Microscopic examination of hair shafts confirmed an ectothrix invasion pattern, and fungal culture definitively identified *Microsporum canis*. The patient achieved complete resolution following a six-week course of oral griseofulvin and adjuvant topical ketoconazole. **Conclusion:** This case demonstrates that indirect fomite transmission from environmental reservoirs like hair salons is a critical risk factor for zoophilic tinea capitis, independent of animal contact. It further establishes trichoscopy as an essential tool for accurate, rapid diagnosis when classic signs, such as Wood's lamp fluorescence, are absent, thereby optimizing patient management and public health outcomes.

1. Introduction

Tinea capitis, an infection of the hair shaft and follicle by keratinophilic fungi known as dermatophytes, stands as the most prevalent dermatophytosis of childhood globally.[1] Beyond its clinical manifestations of alopecia and scaling, tinea capitis can lead to significant psychosocial distress, including social stigmatization and school absenteeism, underscoring the need for prompt and effective management. The clinical spectrum of this disease is remarkably broad, ranging from subtle, non-inflammatory presentations that mimic seborrheic dermatitis to profound, suppurative inflammatory reactions (kerion) that can culminate in permanent, scarring alopecia.[2] The classic "gray patch" variant, characterized by discrete patches of hair loss with lusterless, broken hair shafts, represents a common non-inflammatory phenotype frequently associated with the zoophilic species *Microsporum canis*.[3]

The epidemiological landscape of tinea capitis is a dynamic tapestry, woven with threads of geography, socioeconomic factors, and evolving human-animal interactions.[4] While the anthropophilic Trichophyton tonsurans has become the dominant pathogen in many Western nations, *M. canis* remains a formidable

and frequent etiologic agent across Europe, Asia, and the Americas.[5] The primary reservoir for *M. canis* is domestic animals, particularly asymptomatic kittens, which can shed infectious arthroconidia into the environment.[6] Consequently, direct contact with an infected animal is the canonical route of transmission. However, an often-underappreciated vector is the contaminated fomite. The remarkable resilience of *M. canis* spores, which can remain viable on inanimate objects for many months, transforms items like combs, brushes, hats, and upholstery into persistent environmental reservoirs.[7] This mode of transmission is of paramount importance in high-contact settings such as schools, daycare centers, and, as highlighted in this report, hair salons.

The diagnostic paradigm for tinea capitis is evolving.[8] For decades, the triad of clinical examination, Wood's lamp fluorescence, and conventional mycology (potassium hydroxide [KOH] microscopy and fungal culture) formed the diagnostic bedrock. However, this classical approach is imperfect. In recent years, trichoscopy has emerged as a powerful, non-invasive diagnostic adjunct that allows for the real-time visualization of the microscopic consequences of fungal invasion. Yet, a comprehensive understanding of tinea capitis requires more than just clinical acumen and diagnostic tools; it demands an appreciation of the underlying biology. The predilection of this disease for children is not coincidental but is rooted in the unique biochemical and immunological microenvironment of the prepubertal scalp.[9] Likewise, the ability of *M. canis* to establish infection is a testament to its sophisticated arsenal of virulence factors, including specialized enzymes and adhesins.

The novelty of this case report lies in its detailed illustration of a classic gray patch tinea capitis caused by *M. canis* in a young child whose primary risk factor was indirect environmental exposure in a hair salon, rather than direct contact with an infected animal.[10] This report aims to dissect the diagnostic and pathophysiological nuances of a classic gray patch tinea capitis infection by correlating a specific environmental exposure with modern trichoscopic findings and exploring the underlying host-pathogen interactions that define this clinical entity. By presenting a case where the classic sign of Wood's lamp fluorescence was absent, we highlight the critical, decisive role of trichoscopy in navigating such diagnostic ambiguities. Ultimately, this report seeks to illustrate how a single, well-characterized clinical case can serve as a model for understanding the intricate interplay between fungal ecology, host biology, and diagnostic science.

2. Case Presentation

A 3-year-old Javanese female was referred to the Dermatology and Venereology outpatient clinic with a chief complaint of a progressively enlarging bald patch on her scalp over a two-week period. A comprehensive review of the anamnestic and clinical data, as systematically presented in Figure 1, provides a compelling narrative that guides the diagnostic process, pointing strongly towards an infectious etiology while simultaneously challenging conventional assumptions about its transmission vector. The history of the present illness details a subacute clinical course that began approximately two weeks prior to consultation. The initial manifestation was a small, seemingly innocuous patch of hair loss accompanied by erythema on the posterior scalp. However, the condition did not remain static; the lesion began to progressively enlarge, and critically, new, distinct patches of alopecia started to appear elsewhere on the scalp. This pattern of expansion and the development of satellite lesions suggested an active and spreading pathological process. The clinical picture was further characterized by the presence of pruritus, a significant symptom confirmed by the mother's observation of the child frequently scratching the affected areas. This constellation of signs—progressive alopecia, erythema, and itching—is a classic triad for an inflammatory or infectious process affecting the hair follicles and surrounding skin. A crucial step in refining the differential diagnosis involves a thorough review of the patient's medical and

family history. As outlined in Figure 1, this aspect of the history was entirely unremarkable. The patient had no personal history of atopic conditions, such as atopic dermatitis or allergic rhinitis, nor any known autoimmune disorders. This negative history is profoundly significant, as it diminishes the likelihood of common non-infectious causes of pediatric alopecia. For instance, alopecia areata, an autoimmune condition, is a primary differential, but the absence of a personal or family history of autoimmunity makes it statistically less probable. Similarly, the lack of an atopic diathesis makes a severe, isolated eczematous process less likely. The family history was also negative for any similar dermatological conditions, helping to exclude heritable hair shaft disorders or genetic predispositions to alopecia. The most revealing and scientifically informative data emerged from the exploration of the patient's social and environmental history, which presented a classic epidemiological puzzle. The section on key risk factors in Figure 1 highlights a pivotal negative finding: the complete absence of animal contact. Tinea capitis, particularly the gray-patch type, often caused by zoophilic (animal-derived) fungi like *Microsporum canis*, is traditionally linked to exposure to infected pets, especially kittens or puppies. This lack of a traditional zoonotic vector compelled a deeper investigation into alternative transmission routes. The investigation yielded a powerful positive correlation: the child's mother worked as a hairdresser, and the child was a frequent visitor to the high-contact environment of the salon. This finding immediately shifted the focus from a zoonotic to a fomite-based transmission model. Hair salons are well-recognized as potential reservoirs for dermatophytes, where infectious fungal spores can contaminate shared instruments. The history was further solidified by the specific report of the child having direct interaction with shared salon combs. This behavior provides a direct and highly plausible mechanism for the inoculation of infectious arthroconidia onto the child's susceptible scalp. Finally, the patient's hygiene and lifestyle were assessed and found to be standard, involving twice-daily bathing and regular hair washing. Figure 1, effectively rules out poor hygiene as a contributing factor and reinforces the conclusion that the infection was acquired from an external, contaminated source rather than resulting from an overgrowth of commensal organisms due to inadequate cleansing. The anamnestic profile presented in Figure 1 constructs a cohesive clinical narrative. We have a patient in the peak age demographic for tinea capitis—a prepubertal 3-year-old whose scalp provides a biochemically permissive environment for fungal growth. The clinical presentation of progressive, pruritic, and erythematous alopecia is classic for the condition. The medical and family histories help to systematically exclude the main non-infectious differential diagnoses. Most importantly, the environmental history uncovers a clear, potent risk factor for fomite transmission in the absence of a traditional animal vector. This complete dataset, drawn from the figure, allows for the formulation of a strong presumptive diagnosis of tinea capitis, likely caused by a dermatophyte transmitted within the salon environment.

Upon examination, the patient was in good general health with stable vital signs. The primary findings were confined to the scalp. The dermatological examination revealed classic signs of a non-inflammatory tinea capitis. The general physical assessment, systematically outlined in Figure 2, established that the patient was systemically well, a crucial initial finding. She was described as alert and cooperative, though in mild distress, likely secondary to the pruritus associated with her scalp condition. Her vital signs were stable and fell within the normal physiological limits for her age, confirming the absence of fever or other systemic responses that might indicate a more severe, disseminated infection. Furthermore, a thorough systemic examination was unremarkable; specifically, there was no palpable cervical, axillary, or inguinal lymphadenopathy.

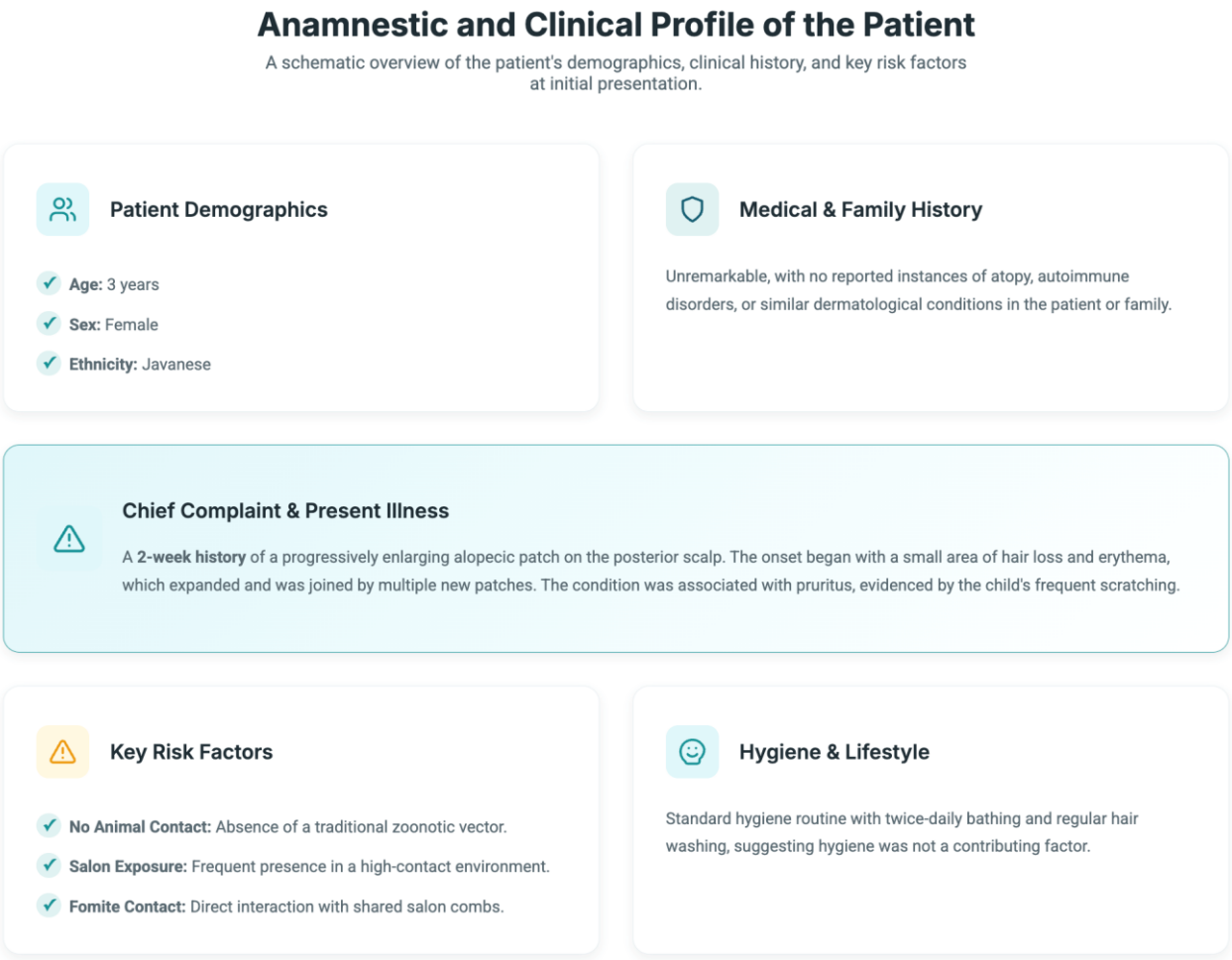


Figure 1. Anamnestic and clinical profile of the patient.

This initial assessment allowed for the confident conclusion that the pathology was localized to the integumentary system. The core of the diagnosis rested upon the detailed dermatological findings, which painted a near-pathognomonic picture of the condition. As visually documented in the clinical photograph and detailed in the schematic (Figure 2), the examination of the scalp revealed multiple alopecic patches. The presence of more than one lesion indicated a multifocal process. These patches were well-demarcated, with distinct borders that clearly separated the affected areas from the healthy, hair-bearing scalp. Their configuration was described as circular to geographic, consistent with the centrifugal growth pattern of dermatophyte infections. The lesions were of a moderate size, ranging from 2x2 cm to 2x3 cm, large enough to be easily noticeable and concerning for the parent. A closer inspection of the surface of these patches provided further critical clues. The skin was characterized by fine, whitish, and adherent scales. This scaling is a direct result of the fungal activity in the epidermis, which induces an accelerated rate of keratinocyte turnover and a mild inflammatory response, leading to the shedding of corneocytes. Crucially, the examination noted a complete absence of significant inflammation, pustules, or crusting. This finding is paramount, as it definitively places the condition within the non-inflammatory spectrum of tinea capitis, distinguishing it from the more destructive inflammatory variants.

Perhaps the most diagnostically significant findings were related to the hair shafts within the alopecic patches. The remaining hairs were not merely absent; they were pathologically altered. They appeared dull, gray, and were characteristically broken just a few millimeters above the scalp surface (Figure 2). This "gray patch" appearance is a hallmark sign. The gray color is not a true change in hair pigmentation but an optical illusion created by the sheath of fungal spores (arthroconidia) that coats the exterior of the hair shaft in an ectothrix-type invasion. This fungal sheath alters the way light reflects off the hair, giving it a lifeless, gray appearance. The breakage of the hair shaft is a direct consequence of the fungus's virulence; dermatophytes produce powerful keratinase enzymes that digest the keratin protein, fundamentally weakening the structural integrity of the hair and causing it to become brittle and snap easily. To complement these observations, a hair pull test was performed, yielding a negative result (Figure 2). This simple bedside maneuver is invaluable in differentiating the cause of alopecia. A positive test, where several hairs are easily extracted from the root, suggests a pathology at the follicular level, such as telogen effluvium or active alopecia areata. In this case, the negative result strongly indicated that the hair loss was not due to shedding from the follicle but rather from the breakage of the structurally compromised hair shafts themselves. This finding corroborates the visual evidence of broken hairs and further solidifies the diagnosis of tinea capitis. In synthesizing these findings, the clinical photograph in Figure 2 serves as the definitive visual record. It captures the well-demarcated patch of alopecia, the fine, dandruff-like scaling on the skin surface, and the presence of the short, dull hair stubs characteristic of this condition. The collective evidence from the general and dermatological examination, from the macroscopic appearance of the patches to the microscopic characteristics of the hair shafts and the result of the hair pull test, converges on a single, confident clinical diagnosis: a classic, non-inflammatory, gray patch tinea capitis.

To establish a definitive diagnosis and differentiate from other causes of pediatric alopecia, a suite of diagnostic tests was performed. While the Wood's lamp examination was negative, both microscopic and cultural investigations strongly supported a diagnosis of tinea capitis caused by *M. canis*. To establish a definitive diagnosis and differentiate from other causes of pediatric alopecia, a suite of diagnostic tests was performed. While the Wood's lamp examination was negative, both microscopic and cultural investigations strongly supported a diagnosis of tinea capitis caused by *M. canis*. The results of these ancillary tests, as presented in Figure 3, formed a cohesive and compelling chain of evidence, moving from rapid initial confirmation to specific, irrefutable identification of the pathogen. The first step in the laboratory investigation was the direct microscopic examination of a plucked hair using a 20% potassium hydroxide (KOH) preparation. This fundamental mycological technique is designed to rapidly visualize fungal elements by dissolving the host keratin and cellular debris, making the fungal structures more apparent. As shown in the micrograph in Figure 3, the examination was conclusively positive. It revealed numerous small fungal spores meticulously arranged in a dense sheath on the exterior of the hair shaft. This specific arrangement is known as an ectothrix pattern of invasion. This microscopic finding directly correlates with the clinical presentation of a "gray patch," as the external mass of spores weakens the hair shaft and alters its light-reflecting properties, giving it a dull, gray appearance. Complementing this traditional microscopic technique, a non-invasive dermoscopic evaluation of the hair and scalp, known as trichoscopy, was performed. This modern tool allows for in-vivo magnification of the affected area, revealing characteristic structural changes to the hair shafts that are often pathognomonic for tinea capitis. The trichoscopic evaluation, as summarized and depicted in Figure 3, identified several key signs highly suggestive of the diagnosis.

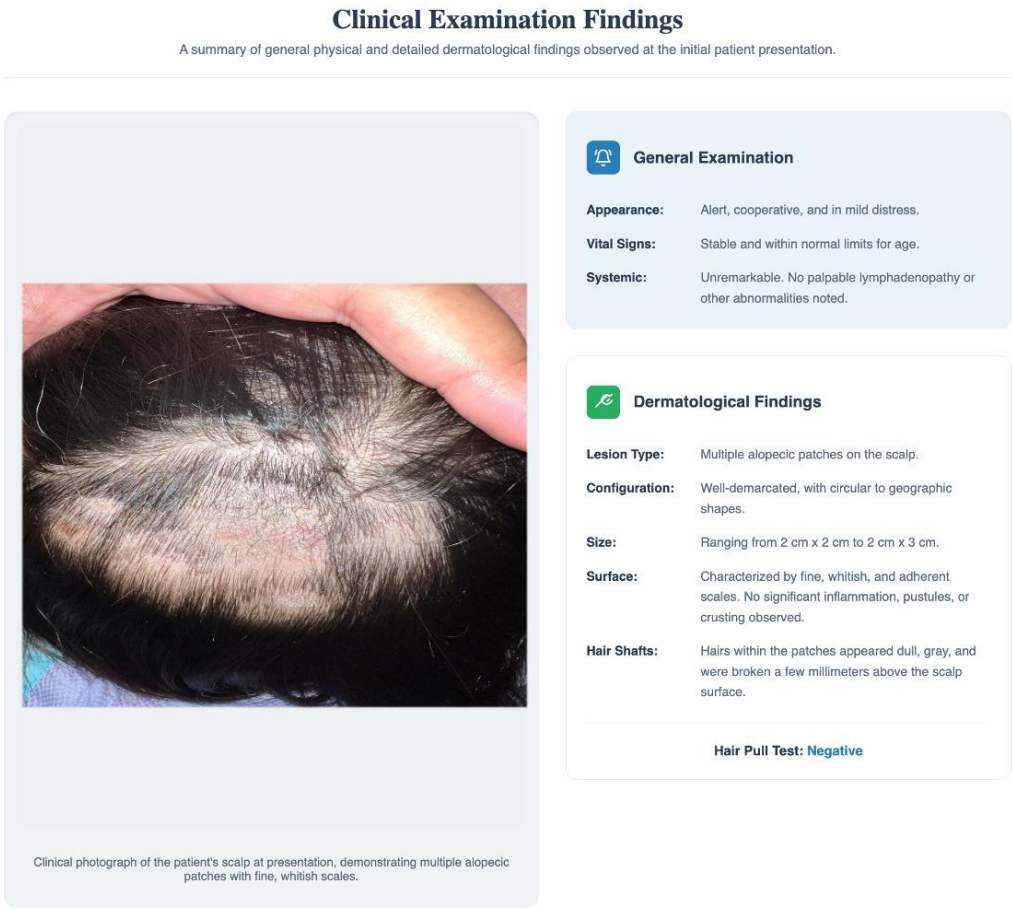


Figure 2. Clinical examination findings.

These included comma hairs, which are short, broken hair stubs that bend due to the structural weakening caused by the fungus, and Morse code-like hairs, which appear as hair shafts with multiple transverse, whitish bands representing internal fractures or air gaps from the fungal invasion. The presence of these specific hair shaft abnormalities provided a strong, independent line of evidence for tinea capitis. The interpretation noted in the figure is critical: these findings are not only suggestive of the disease but are also instrumental in differentiating it from other common causes of pediatric alopecia. For instance, alopecia areata, a primary clinical mimic, would typically present with different trichoscopic signs, such as exclamation mark hairs and yellow dots. Thus, trichoscopy served as a powerful bedside tool that bridged the gap between clinical suspicion and mycological confirmation. While the KOH and trichoscopy provided strong evidence, the gold standard for diagnosis, as noted in Figure 3, is the fungal culture. This method is essential for definitively identifying the specific causative species, a crucial step for guiding targeted therapy and understanding the local epidemiology. Hair and scale samples were inoculated onto a culture medium. The macroscopic examination of the resulting colony revealed several characteristic features: it was rapidly growing and produced a white-to-yellowish colony with a powdery surface. Critically, the reverse side of the culture plate showed a deep yellow-orange pigment, a classic feature associated with *Microsporum canis*. The final and most definitive piece of evidence came from the microscopic examination of the cultured fungus. As shown in the micrograph in Figure 3, the fungus

produced numerous large, distinctive macroconidia. These structures were spindle-shaped, thick-walled, and crucially, possessed more than six internal septa or cross-walls. This unique morphology of the macroconidia is the pathognomonic microscopic feature of *Microsporum canis*, allowing for its unequivocal identification. The combination of the macroscopic colony characteristics and the unique microscopic morphology provided the final, irrefutable identification of the etiologic agent. In synthesis, the diagnostic journey presented in Figure 3 was a model of clinical science. The KOH preparation offered a rapid confirmation of an ectothrix fungal infection. Trichoscopy provided immediate, non-invasive corroboration and helped exclude other diagnoses. Finally, the fungal culture delivered the specific and definitive identification of *Microsporum canis*, providing a solid foundation for the subsequent therapeutic plan.

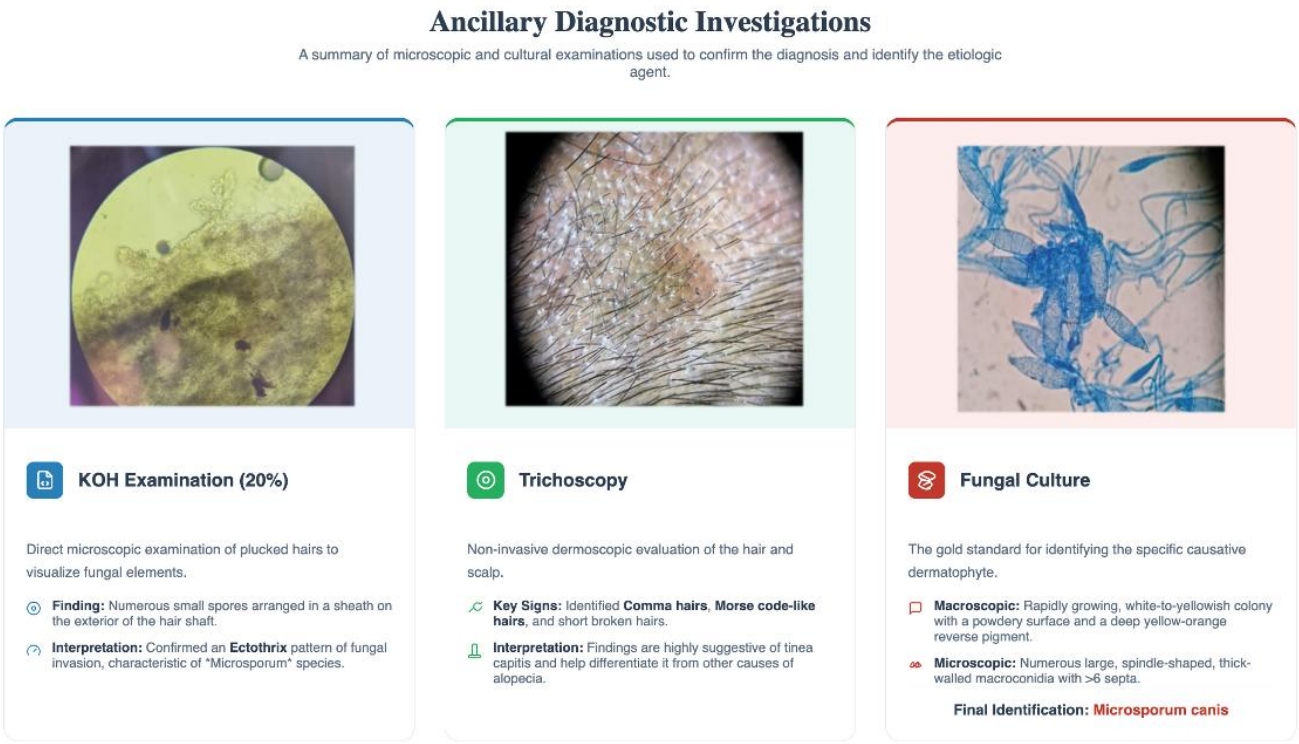


Figure 3. Ancillary diagnostic investigation.

Based on the definitive diagnosis, a targeted therapeutic plan was initiated. The patient's response to treatment was monitored over several weeks, showing significant clinical improvement and eventual resolution. Based on the definitive diagnosis, a targeted therapeutic plan was initiated. The patient's response to treatment was monitored over several weeks, showing significant clinical improvement and eventual resolution. The therapeutic plan was designed to attack the infection from two angles: a systemic oral therapy to eradicate the fungus from within the hair follicle and an adjuvant topical therapy to control the spread of infectious spores on the scalp surface (Figure 4). The cornerstone of the treatment was systemic therapy, for which oral griseofulvin (microsize) was prescribed at a dose of 250 mg once daily for a total duration of six weeks (Figure 4). The choice of griseofulvin is a deliberate and evidence-based decision, particularly for tinea capitis caused by *Microsporum canis*. Griseofulvin is a fungistatic agent,

meaning it inhibits fungal replication rather than killing the fungus outright. Its mechanism of action is to disrupt the fungal mitotic spindle by binding to tubulin, thereby halting cell division. After oral administration, the drug is absorbed and preferentially deposits in keratin precursor cells of the skin, hair, and nails. As these cells mature and form new keratin, the griseofulvin becomes an integral part of the hair shaft. This creates a new, drug-impregnated hair that is resistant to fungal invasion. As this healthy hair grows, it gradually pushes out and replaces the old, infected hair shaft, which is eventually shed. This process necessitates a prolonged treatment course, with the six-week duration selected to align with the hair growth cycle and ensure the complete replacement of all infected hairs. Complementing the systemic treatment was an adjuvant topical therapy consisting of 2% ketoconazole shampoo, used twice weekly (Figure 4). The role of the topical agent is not to cure the infection—as it cannot penetrate the hair follicle to the required depth—but to serve as a crucial public health and hygiene measure. Ketoconazole is an azole antifungal that works by inhibiting the synthesis of ergosterol, a vital component of the fungal cell membrane. By disrupting the membrane, it effectively reduces the fungal load on the scalp's surface and, most importantly, decreases the viability and shedding of infectious arthroconidia into the environment. This minimizes the patient's contagiousness, reducing the risk of transmission to family members, playmates, or through contaminated fomites.

Therapy Initiation This initial visit marked the baseline where the diagnosis was confirmed and the comprehensive treatment plan was implemented (Figure 4). A critical component of this visit was the extensive counseling provided to the parents regarding the nature of the fungal infection, the importance of strict medication adherence over the full six weeks, and strategies for preventing transmission, such as avoiding the sharing of combs, hats, and pillowcases. Early Response At the two-week follow-up, the first signs of a positive therapeutic response were evident. The parents reported that no new lesions had appeared and the existing patches were stable, indicating that the systemic griseofulvin was successfully halting the progression of the infection (Figure 4). Furthermore, the patient's symptoms had significantly improved, with a marked reduction in both pruritus and visible scalp scaling. This symptomatic relief is a direct result of the treatment controlling the fungal activity and the associated inflammatory response. The note that trichoscopy showed a decrease in pathological hair forms provided an objective, microscopic confirmation of this early improvement. Clear Improvement The four-week follow-up represented a major milestone in the patient's recovery. The pruritus, a distressing symptom for the child, had completely resolved (Figure 4). The most significant finding at this stage was the visible regrowth of fine vellus hair within the alopecic patches. This is the definitive sign that the fungal assault on the hair follicles has been successfully neutralized, allowing the natural hair growth cycle to resume. This observation confirmed a robust therapeutic response and demonstrated that the scalp was actively healing. The medication was also noted to be well-tolerated, with no reported adverse effects. Clinical Resolution At the conclusion of the six-week treatment course, the patient had achieved complete clinical resolution (Figure 4). A final examination confirmed the disappearance of all signs of active infection, including scaling and erythema. The initial vellus hairs had progressed to more robust regrowth, indicating a full restoration of follicular function. This successful outcome was the culmination of the evidence-based therapeutic plan and diligent adherence. The visit concluded with final counseling for the parents on preventative measures to minimize the risk of future recurrence.

3. Discussion

This case of gray patch tinea capitis provides a unique opportunity to dissect the multifaceted nature of a common pediatric disease, moving beyond a

simple clinical description to explore the intricate interplay of environmental epidemiology, host biology, pathogen virulence, and diagnostic science. The central epidemiological lesson of this case is the powerful demonstration of fomite-mediated transmission in the absence of a direct zoonotic link.[11]

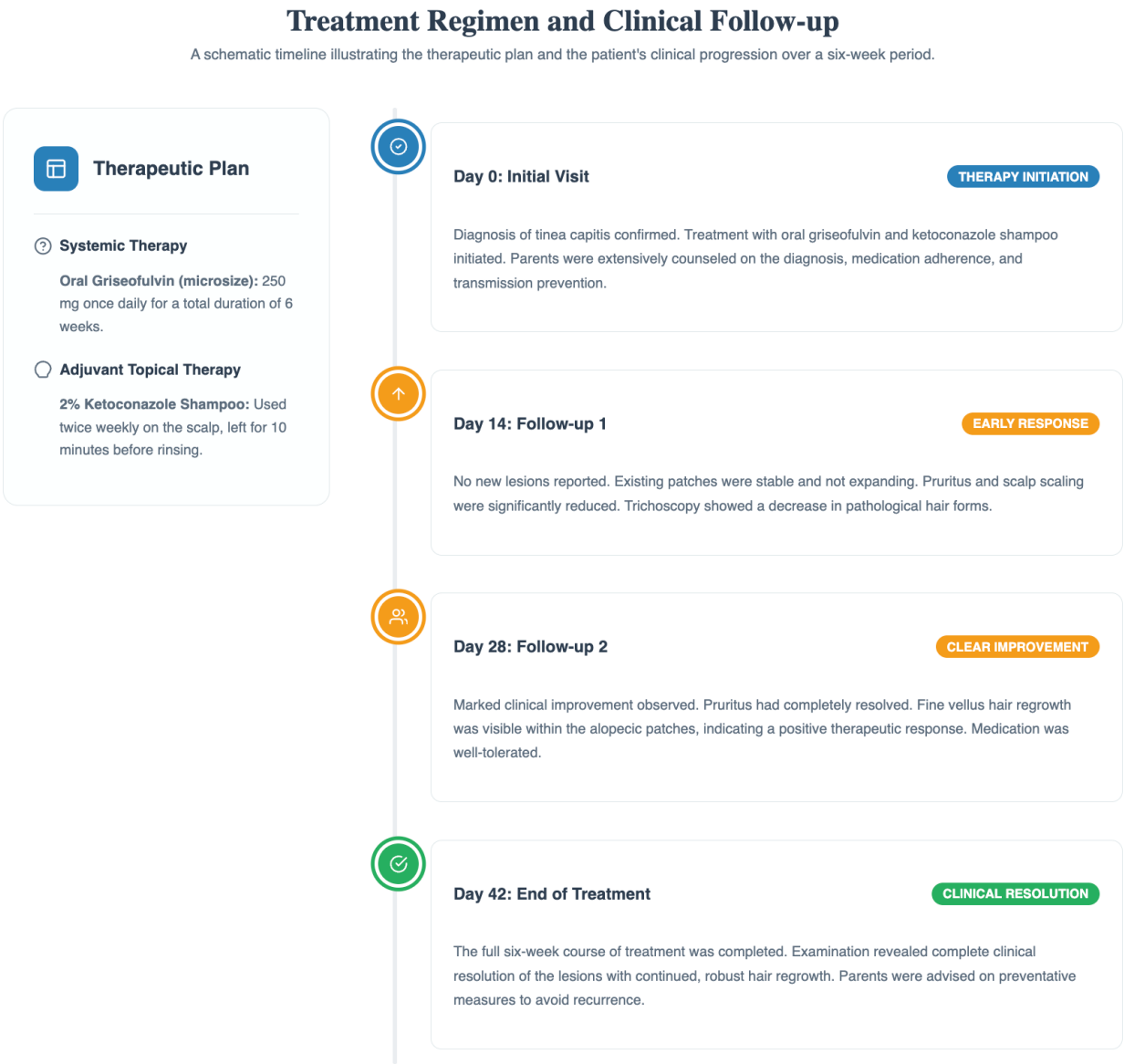


Figure 4. Treatment regimen and clinical follow-up.

The traditional paradigm for *M. canis* infection implicates direct contact with an infected animal reservoir. However, the patient's history was unequivocally negative for such contact. Instead, a meticulous history revealed a potent environmental risk: the mother's workplace, a hair salon. This setting represents a near-perfect ecological niche for the transmission of dermatophytes.[12] It is a high-traffic environment with a rapid turnover of potential hosts, and it is replete with instruments—combs, brushes, clippers, towels—that are ideal vectors for transferring infectious arthroconidia from one individual to another. The arthroconidia of *M. canis* are hardy, environmentally resistant structures that can be shed

from infected hairs and scales and remain viable for many months. Studies have confirmed the presence of pathogenic dermatophytes on salon instruments, underscoring their role as mechanical vectors.[13] In this case, the child's habit of playing with shared combs provided a direct and plausible mechanism for inoculation. This observation elevates the clinical importance of the environmental history from a routine inquiry to a critical forensic investigation. It compels clinicians to think beyond the household and consider other high-risk community settings. This case argues that in urban and suburban environments, where direct contact with stray or unknown animals may be limited, these "professional" environments may emerge as increasingly significant nexuses of transmission, not just for anthropophilic but also for zoophilic fungi.[14]

The ability of *M. canis* to infect a human host is not a passive process but an active invasion orchestrated by a sophisticated array of virulence factors.[15] The infection cascade begins with the adherence of arthroconidia to the keratinocytes of the stratum corneum. This is a specific, receptor-mediated event, likely involving fungal adhesins that recognize host cell surface glycoproteins. Once attached, the fungus must breach the formidable barrier of keratin, the protein that comprises over 90% of the hair shaft. To do this, dermatophytes secrete a cocktail of extracellular proteases, chief among them being the keratinases. These are not generic proteases; they are highly specialized enzymes adapted to degrade the mechanically tough and chemically resilient alpha-keratin. These enzymes belong to several families, including the subtilisin-like proteases (SUBs) and the fungalysin-like metalloproteases (MEPs). These enzymes work synergistically to dismantle the keratin structure, breaking down the disulfide bonds and peptide backbone. This process of keratinolysis serves two purposes: it provides the fungus with a rich source of nitrogen and carbon for its own growth, and it physically clears a path for the hyphae to invade deeper into the hair follicle.[16] The fine scaling seen on the patient's scalp is a direct result of this enzymatic activity and the subsequent inflammatory response and accelerated epidermal turnover. As the hyphae penetrate the hair follicle, they grow downwards towards the hair bulb. In an ectothrix infection, as seen in this case, the fungus colonizes the exterior of the hair shaft. It forms a dense sheath of hyphae and arthroconidia around the cuticle, a structure known as Adamson's fringe. This external colonization is what structurally compromises the hair shaft, leading to the characteristic breakage a few millimeters above the scalp surface and causing the clinical picture of alopecia.[17]

The striking predilection of tinea capitis for prepubertal children is a cornerstone of its epidemiology and is deeply rooted in the unique biology of the pediatric scalp.[18] The "prepubertal scalp hypothesis" posits that the biochemical composition of sebum, the oily substance secreted by sebaceous glands, changes dramatically at puberty. Prepubertal sebum is composed primarily of triglycerides and wax esters. At puberty, under the influence of androgens, sebaceous gland activity increases, and the composition of sebum shifts. There is a marked increase in the secretion of free fatty acids, squalene, and cholesterol. Crucially, many of the free fatty acids present in post-pubertal sebum, such as sapienic acid, possess potent fungistatic properties. They can disrupt fungal cell membranes and inhibit key metabolic enzymes, creating a hostile microenvironment that is highly resistant to dermatophyte colonization. The prepubertal scalp, lacking these protective lipids, is therefore a biochemically "permissive" environment. It provides a warm, moist, nutrient-rich setting without the chemical defenses that are present after puberty.[19] This case, in a 3-year-old child, perfectly exemplifies this window of vulnerability. The infection is not merely a matter of exposure; it is a matter of exposure to a susceptible host whose innate cutaneous defenses have not yet matured.

The clinical appearance of tinea capitis—whether it is inflammatory or non-inflammatory—is a direct reflection of the immunological dialogue between the

host and the fungus. The host's innate immune system is the first line of defense. Keratinocytes and Langerhans cells in the epidermis are equipped with Pattern Recognition Receptors (PRRs), such as Toll-like receptors (TLR2, TLR4) and C-type lectin receptors (Dectin-1, Dectin-2).[18] These receptors recognize conserved molecular patterns on the fungal cell wall, such as chitin and β-glucans. This recognition triggers a signaling cascade that leads to the production of pro-inflammatory cytokines and chemokines, recruiting neutrophils, macrophages, and lymphocytes to the site of infection. This innate response then shapes the subsequent adaptive immune response, which is primarily cell-mediated. A robust Th1-type response, characterized by the production of interferon-gamma, is highly effective at clearing the fungal infection but often results in a vigorous inflammatory reaction, leading to the formation of a kerion.[19] Conversely, the non-inflammatory gray patch presentation seen in this case suggests a state of relative immunological tolerance. Zoophilic fungi like *M. canis* are not as well-adapted to the human host as anthropophilic species. This lack of adaptation can, paradoxically, lead to a less aggressive immune response. The fungus may fail to trigger the strong pro-inflammatory signals required for a full-blown Th1 response. Instead, the immune system may default to a less effective Th2-type response or a state of anergy. This allows the fungus to persist and establish a chronic, low-grade infection without being eliminated by the host's defenses. The lack of significant inflammation in this patient is therefore not a sign of a mild infection, but rather a sign of a successful fungal strategy of immune evasion.

This case beautifully illustrates how modern diagnostic tools can visualize the direct pathophysiological consequences of fungal infection. The negative Wood's lamp finding, while initially confounding, is a key diagnostic nuance. The characteristic green fluorescence associated with *M. canis* is caused by pteridine, a heterocyclic compound that is a byproduct of the fungus's tryptophan metabolism.[20] However, not all strains of *M. canis* produce pteridine in sufficient quantities to be detectable. The expression of the metabolic pathway responsible for pteridine synthesis can be variable and may be influenced by genetic factors within the fungus or by the specific nutrient environment of the host scalp. Therefore, the absence of fluorescence is not a definitive ruling-out criterion but a diagnostic clue that must be integrated with other findings. It is in this context that trichoscopy proves its immense value. The trichoscopic signs observed are not random artifacts; they are direct images of the biomechanical failure of the hair shaft under fungal assault. Comma Hairs, These are short, broken hair stubs that are bent into a comma shape. This occurs because the fungal sheath on the outside of the hair weakens the cortex asymmetrically. As the hair breaks, the residual internal tension causes the weakened stub to curl. Morse Code-like Hairs, These hairs show multiple, discontinuous whitish bands along the shaft. This is thought to be caused by air-filled spaces or pockets of fungal elements that create transverse micro-fractures within the hair shaft, disrupting its optical properties. Zigzag Hairs, These are hairs that are bent at sharp angles, representing another form of structural failure under the stress of fungal invasion.

These signs provide a real-time, in-vivo confirmation of a pathological process that is actively destroying the hair shafts. They allow the clinician to make a highly confident presumptive diagnosis and to differentiate tinea capitis from non-infectious causes of alopecia, like alopecia areata, which has its own distinct trichoscopic signature (exclamation mark hairs, yellow dots). The choice of treatment in this case was guided by a direct understanding of the pathogen's biology and the drug's mechanism of action. Systemic therapy is non-negotiable for tinea capitis, as topical agents cannot achieve therapeutic concentrations within the hair follicle. Griseofulvin was chosen as the first-line agent, and this choice is strongly supported by evidence. Griseofulvin is particularly effective against *Microsporum* species. Its mechanism of action is elegant: after oral administration, it is absorbed and incorporated into

the keratin precursor cells in the hair matrix.[20] As new keratin is synthesized, the drug becomes an integral part of the hair shaft. Griseofulvin is fungistatic; it binds to fungal tubulin, disrupting the formation of the mitotic spindle and thereby arresting fungal cell division. The fungus can no longer replicate, and as the new, drug-impregnated, resistant hair grows out, the old, infected portion of the hair is shed, eventually clearing the infection. This mechanism explains why a long course of treatment (6-8 weeks) is necessary—it must match the time it takes for the hair to grow out. The use of adjuvant 2% ketoconazole shampoo represents a targeted intervention aimed at both the patient and the community. Ketoconazole is an azole antifungal that inhibits the enzyme lanosterol 14-α-demethylase, a key step in the synthesis of ergosterol. Ergosterol is the primary sterol in the fungal cell membrane, analogous to cholesterol in human cells. Without ergosterol, the fungal membrane loses its integrity and cannot function properly. While the shampoo cannot cure the infection, it effectively kills the fungus on the scalp surface and reduces the shedding of viable arthroconidia into the environment. This is a critical public health measure that breaks the cycle of transmission by rendering the patient less contagious to family members and peers.

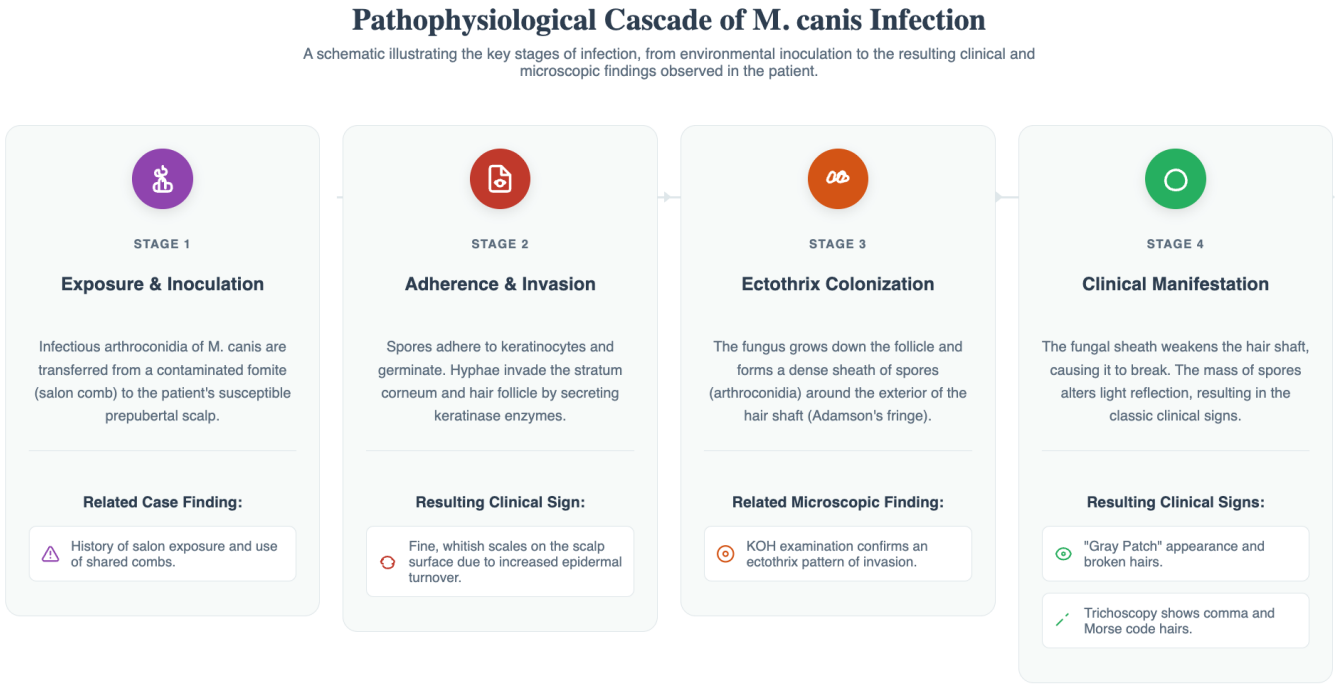


Figure 5. Pathophysiological cascade of *M. canis* infection.

Figure 5 showed a comprehensive and elegant schematic that detailed the four-stage pathophysiological cascade of the *Microsporum canis* infection observed in the patient. The entire infectious process, as illustrated in the first panel of Figure 5, began with the critical inciting event: Exposure and Inoculation. This stage represents the crucial link between the external environment and the susceptible host. The figure specifies that infectious arthroconidia of *M. canis* were transferred from a contaminated fomite, identified as a salon comb, directly onto the patient's scalp. Arthroconidia are the hardy, infectious spores of the fungus, designed for environmental survival and transmission. A fomite is any inanimate

object that can carry and transmit infectious agents, and in this context, the shared comb acted as a perfect mechanical vector. This process is explicitly linked to the patient's clinical history, which notes a clear history of salon exposure and the use of shared combs. The figure also highlights that the inoculation occurred on a "susceptible prepubertal scalp." This detail is of immense biological significance; the scalp of a young child lacks the fungistatic fatty acids found in adult sebum, creating a biochemically permissive environment that is uniquely vulnerable to dermatophyte colonization. Adherence & Invasion, This stage details the critical moments where the fungus establishes a foothold and begins its assault. The figure describes a two-step process. First, the spores adhere to the host's keratinocytes, the primary cells of the epidermis. This is an active process mediated by fungal adhesin proteins that recognize specific molecules on the skin cell surface. Following adherence, the spores germinate, sprouting hyphae, which are the long, branching filaments that constitute the body of the fungus. These hyphae then begin to actively invade the surrounding tissue, penetrating both the stratum corneum (the outermost layer of the skin) and the hair follicle. According to the figure, this invasion is facilitated by the secretion of powerful keratinase enzymes. These enzymes are the primary virulence factor for dermatophytes, allowing them to digest the tough, fibrous keratin protein that makes up skin and hair, using it as a source of nutrients. This enzymatic digestion and the resulting mild inflammatory response lead to the first visible clinical sign of the infection: the appearance of fine, whitish scales on the scalp surface, which is a direct result of the increased epidermal turnover caused by the fungal activity. Ectothrix Colonization, in this phase, the hyphae grow down the hair follicle and begin to colonize the hair shaft itself. As described in Figure 5, the fungus forms a dense sheath of spores (arthroconidia) around the exterior of the hair shaft. This characteristic external sheath is known as "Adamson's fringe." The term "ectothrix" specifically refers to this pattern of invasion, where the fungus remains on the outside of the hair shaft rather than penetrating its core (an endotrix pattern). This stage is a microscopic process, invisible to the naked eye, but it is the central pathological event that leads to the subsequent clinical signs. The figure directly links this stage to the patient's diagnostic findings, noting that the KOH examination, which allows for direct visualization of the fungus on the hair, confirmed this exact ectothrix pattern of invasion. The final stage of the cascade is the Clinical Manifestation, where the cumulative effect of the underlying microscopic pathology becomes visible as clinical disease. As Figure 5 explains, the dense ectothrix sheath of the fungus physically and chemically weakens the hair shaft through the continuous action of keratinase enzymes, causing it to become brittle and break easily just a few millimeters from the scalp surface. Simultaneously, the massive number of spores coating the hair stubs alters the way light reflects off them. These two processes directly result in the classic clinical signs observed in the patient. The breakage of the hair leads to patches of alopecia and the presence of short, broken hairs. The altered light reflection from the spore sheath is what causes the characteristic dull, "Gray Patch" appearance. Furthermore, the figure connects this stage to the more advanced diagnostic findings from trichoscopy. The structural weakening and breakage of the hair shafts are what cause them to curl into the "comma hairs" or show internal fractures as "Morse code hairs" when viewed under the dermatoscope, providing a direct visual link between the pathophysiology and the modern diagnostic signs.

4. Conclusion

This case of pediatric gray patch tinea capitis compels a paradigm shift in both diagnosis and epidemiological investigation. It firmly establishes that the search for a transmission source must extend beyond animal vectors to critical environmental reservoirs, such as salons, demanding a more forensic approach to the clinical history. Moreover, it champions the integration of trichoscopy not merely

as a supplementary tool, but as a primary diagnostic modality that reveals the direct pathophysiological consequences of fungal invasion, proving indispensable when traditional clues are absent. This integrated bio-clinical approach, which connects environmental ecology to molecular pathophysiology and targeted therapeutics, represents the new gold standard for managing dermatophytoses in the 21st century.

5. References

1. Xiao YY, Zhou YB, Chao JJ, Ma L. Successful treatment of tinea capitis caused by *Microsporum canis* in a 23-day-old newborn with itraconazole pulse therapy and a review of the literature. Dermatol Ther. 2021; 34(5): e15078.
2. Lekkas D, Ioannides D, Apalla Z, Lallas A, Lazaridou E, Sotiriou E. Dermoscopy for discriminating between trichophyton and microsporum infections in tinea capitis. J Eur Acad Dermatol Venereol. 2018; 32(6): e234–5.
3. Na N, Choi Y, Park J. Wood's lamp examination findings of tinea capitis by *Microsporum canis*. Taehan Uijinkyun Hakhoe Chi. 2019; 108–9.
4. Komatsu-Fujii T, Nonoyama S, Ogawa M, Fukumoto T, Ookura T, Anzawa K, et al. Usefulness of topical efinaconazole for infantile tinea capitis due to *Microsporum canis* diagnosed with Wood's light. J Dermatol. 2020; 47(11): e401–3.
5. Park TH, Kim WI, Cho MK, Whang KU, Kim S. Tinea capitis and tinea corporis caused by *Microsporum canis* in an 82-year-old woman: efficacy of sequence analysis. Taehan Uijinkyun Hakhoe Chi. 2020; 17–21.
6. Yotsu RR, Kouadio K, Yao A, Vagamon B, Takenaka M, Murota H, et al. Tinea capitis caused by *Microsporum audouinii*: a report of two cases from Côte D'Ivoire, West Africa. Trop Med Infect Dis. 2021; 6(1): 9.
7. Aspiroz C, Melcon B, Cerro PA, Gilaberte Y. Tinea capitis caused by *Microsporum canis* treated with methyl-aminolevulinate daylight photodynamic therapy and ketoconazole shampooing. Photodermatol Photoimmunol Photomed. 2021; 37(6): 567–8.
8. Wang T, Chen J-Q. Tinea capitis due to *Microsporum canis*. N Engl J Med. 2021; 385(22): 2077.
9. El Imene Ouni N, Aounallah A, Ghariani N, Mokni S, Belajouza C, Denguezli M. Tinea capitis by *Microsporum canis* in a 3-year-old girl with palmoplantar pustular dermatophytid reaction. Clin Exp Dermatol. 2022; 47(3): 596–7.
10. Zou X, Chen W, Li R. Neonatal-onset inflammatory Tinea capitis by *Microsporum canis* due to a woolen hat. Mycopathologia. 2023; 188(5): 585–7.
11. Wang R, Liu W, Liu X, Wan Z, Sybren de Hoog G, Li R, et al. Comparative analysis of whole genomes and transcriptomes of *Microsporum canis* from invasive dermatophytosis and tinea capitis. Emerg Microbes Infect. 2023; 12(1): 2219346.
12. Santino MFF, de Melo CS, Melo AS de A, Lima SL, Paixão M do N, Akiti T, et al. Microsporum audouinii: Emergence of an etiological agent of tinea capitis in Rio de Janeiro, Brazil (2012-2019). Med Mycol. 2024; 62(10).
13. Ebrahimibarogh R, Ghazanfari M, Shokohi T, Yousefiabdolmaleki E, Seyedmousavi A, Hedayati MT. Tinea capitis due to *Microsporum ferrugineum*: a case of unusual laboratory finding on direct microscopic examination of infected hairs and skin lesions. Med Mycol Case Rep. 2024 Mar; 43(100629): 100629.
14. Martínez-Ortega JI, Franco González S, Martinez-Jaramillo B, Ramirez Cibrian AG. *Microsporum canis*-induced Tinea capitis: a rapid screening algorithm. Cureus. 2024; 16(3): e55919.

15. Shi L, Li J, Liu W, Shi D. Coexistence of Malassezia species and *Microsporum canis* in the lesions of adults with tinea capitis. Infect Drug Resist. 2024; 17: 2431–8.
16. Capoor MR, Sharma S, Goenka S, Das S, Rudramurthy SM, Khunger N, et al. Tinea capitis caused by *Microsporum canis*: a case study of three family members in India, a non-endemic region. Indian J Med Microbiol. 2024; 50(100621): 100621.
17. Bennani K, Sekal M, Adadi S, Tlamçani Z. Tinea capitis due to *Microsporum canis* in the university hospital Hassan II of fez: Epidemiological and mycological profile. Int J Life Sci Res Arch. 2024; 7(1): 004–12.
18. Errichetti E, Pietkiewicz P, Salwowska N, Szlązak P, Żychowska M, Bhat YJ. Dermoscopy in tinea capitis/barbae and Tinea of glabrous skin: a comparative analysis between polarized and ultraviolet-induced fluorescence examination to differentiate Microsporum from trichophyton infections. Photodermatol Photoimmunol Photomed. 2024; 40(5): e12999.
19. Sun D, Lu J, Liu T, Wang J. Wood's lamp for early detection of *Microsporum canis* tinea capitis in children. Photodiagnosis Photodyn Ther. 2025; 51(104428): 104428.
20. Tchernev G, Shine A, Linkwinstar D, Broshtilova V, Nenoff P. *Microsporum canis* infection from a cat : a source of tinea capitis and dermatophytosis in an entire family. Dermatologie (Heidelb). 2025.