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The Dermatophyte Masquerade: A Comprehensive Review of Atypical Clinical Presentations and the Diagnostic Pitfalls of Superficial Fungal Infections

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Abstract:

Background: Dermatophytes are the most prevalent pathogenic fungi globally, infecting an estimated 20-25% of the world's population. While classically recognized by annular, scaly plaques with central clearing, dermatophytes exhibit a remarkable and underappreciated clinical polymorphism. This chameleonic ability frequently leads to diagnostic confusion with a broad spectrum of inflammatory, autoimmune, and neoplastic skin diseases.

Objective: To systematically characterize the atypical morphological variants of superficial dermatophytosis that mimic other dermatoses and to provide a practical diagnostic framework to avoid misdiagnosis.

Methods: We present a descriptive case series of nine patients with culture-proven or KOH-confirmed dermatophyte infections, each initially misdiagnosed as a distinct dermatological entity. We review the underlying pathophysiological mechanisms—including host immune response, fungal strain variation, and the confounding effect of topical corticosteroid use (tinea incognito)—that contribute to these atypical presentations.

Results: Our series illustrates nine distinct mimetic patterns: (1) Alopecia Areata-Like Tinea Capitis, (2) Contact Dermatitis-Like Tinea Faciei, (3) Discoid Eczema-Like Tinea Corporis, (4) Tinea Imbricata-Like Concentric Scaling, (5) Folliculitis-Like Tinea Barbae/Corporis, (6) Lichen Planus Actinicus-Like Tinea, (7) Rosacea-Like Tinea Faciei, (8) Pustular Psoriasis-Like lesions on the groins and (9) Dermatitis Cosmetica like tinea on the face and neck. In all cases, the correct diagnosis was established only after mycological examination, leading to resolution with systemic or topical antifungal therapy.

Conclusion: Dermatophytosis has rightfully earned its place among the "Great Imitators" in dermatology. Failure to recognize these masqueraders results in prolonged morbidity, inappropriate immunosuppressive therapy, and increased healthcare costs. We propose a "Fungal-First" diagnostic algorithm and emphasize that any scaly, erythematous, or pustular eruption unresponsive to standard therapy warrants mycological investigation.

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Introduction

Superficial fungal infections caused by dermatophytes—organisms of the genera *Trichophyton*, *Microsporum*, and *Epidermophyton*—represent a global public health burden. They are the fourth most common cause of human disease worldwide, with prevalence rates exceeding 20% in some tropical and subtropical regions [1]. The traditional clinical gestalt for tinea corporis, known colloquially as "ringworm," involves an erythematous, pruritic, polycyclic plaque with an active, scaly, raised border and a relatively clear, hypopigmented center. However, this textbook description is merely the tip of the iceberg.

Clinical dermatology has long recognized the concept of the "Great Imitator," historically attributed to syphilis and later to lupus erythematosus. We propose that dermatophytes have earned a place alongside these conditions due to their unparalleled ability to mimic an extensive array of unrelated dermatoses [2]. This morphological chameleonism is not a biological anomaly but rather a dynamic interplay between the virulence factors of the infecting strain, the host's immunological response (both innate and adaptive), and, critically, iatrogenic modifications [3].

The widespread availability of over-the-counter and prescription topical corticosteroids has exacerbated this diagnostic dilemma. When applied to a fungal infection, corticosteroids suppress the inflammatory response, alleviating pruritus and erythema temporarily. However, they simultaneously inhibit local Langerhans cell function and neutrophil chemotaxis, allowing the fungus to proliferate unchecked. This condition, termed tinea incognito (or tinea atypica), results in bizarre, non-annular, crusted, or pustular lesions that defy all classical descriptions [4,5].

The clinical consequences of misdiagnosis are profound. Patients are frequently subjected to prolonged courses of high-potency topical steroids, systemic corticosteroids, or even immunosuppressive biologics (in cases misdiagnosed as psoriasis or eczema). These interventions not only aggravate the underlying fungal infection, leading to deeper tissue invasion and a more extensive disease burden and great liability of misdiagnosis but also expose the patient to significant iatrogenic side effects [6].

Given this diagnostic quagmire, this review aims to: (1) characterize nine distinct mimetic phenotypes of dermatophytosis through illustrative case vignettes; (2) elucidate the pathophysiological mechanisms driving these atypical presentations; and (3) propose a practical, evidence-based diagnostic algorithm to aid clinicians in differentiating true dermatophytosis from its clinical mimics.

2. METHODS (Case Series Design)

We conducted a retrospective review of patients presenting to the Al-Dhahia private Dermatology**'Clinic between** 1st January 2022 and 1st December 2025. Inclusion criteria were: (1) clinical presentation consistent with an atypical or mimetic dermatophyte infection; (2) confirmation of dermatophytosis via direct microscopic examination using 20% potassium hydroxide (KOH) with dimethyl sulfoxide (DMSO) and/or positive fungal culture on Sabouraud dextrose agar; and (3) complete resolution of lesions following appropriate antifungal therapy. We excluded patients with confirmed bacterial, viral, or parasitic infections, as well as those with definitive non-infectious dermatoses confirmed by histopathology. Informed consent was obtained from all patients for the use of clinical images and data for publication.

3. CASE VIGNETTES (The Nine Masqueraders) (Table 1)

Case 1: The Alopecia Areata Mimic

Patient: A 7-year-old boy presented with a 3-month history of a two, well-circumscribed, non-scarring patches of alopecia on the right occipital scalp. There was no visible scaling, erythema, or inflammation. Strikingly, The is easy and painless hair removal at the periphery of the patch. Previous diagnosis: Alopecia areata. He had been treated with topical clobetasol propionate without improvement (Figure 1).

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Examination: Dermoscopy revealed multiple "black dots" (broken hairs) and subtle perifollicular scaling and other features of tinea capitis (Figur 21). KOH scraping from the affected area demonstrated endothrix hyphae. Culture grew *Trichophyton tonsurans*.

Outcome: Treated with oral griseofulvin and 2% ketoconazole shampoo for 8 weeks; complete hair regrowth was observed at 6 months.

Case 2: The Contact Dermatitis Mimic

Patient: A 34-year-old male presented with an acute, weeping, vesicular, and edematous plaques on both hands and right foot, associated with intense pruritus. The eruption and itching shwoed partial and temporary response to topical steroids.

Examination: Patch testing was negative. The lesion lacked the "string-of-pearls" vesiculation typical of acute eczema (Figure 3). A KOH preparation from the vesicle roof showed abundant septate hyphae. Culture was positive for *Trichophyton rubrum* (Figure 4).

Outcome: Treated with oral itraconazole 100 mg twice daily for 4 weeks and then once daily for another 4 weeks to reduce recurrence; the lesion resolved with mild post-inflammatory hyperpigmentation.

Case 3: The Discoid Eczema (Nummular Dermatitis) Mimic

Patient: A 38-year-old male with a 2-year history of multiple, sharply demarcated, coin-shaped, hyperkeratotic, and intensely pruritic plaques on the extensor surfaces of the forearms and lower legs.

Examination: No central clearing was visible (Figure 5). He had been treated with potent topical corticosteroids and emollients with only transient relief, followed by paradoxical worsening. KOH scraping from the scaly edge revealed branching hyphae. Culture grew *Epidermophyton floccosum* (Figure 6).

Outcome: Treated with oral itraconazole pulse therapy (200 mg BID for 1 week/month for 3 months) and topical naftifine; complete clearance achieved by month 3.

Case 4: The Tinea Imbricata Mimic (Concentric Rings)

Patient: A 32-year-old immunocompetent male returning from South of Iraq presented with widespread, concentric, overlapping rings of scaling on his trunk, resembling wood grain (Figure 7).

Examination: Classic tinea imbricata is usually caused by *T. concentricum*. However, KOH revealed hyphae, and culture unexpectedly identified *T. rubrum*—a rare presentation of a "rubrum imbricata" variant.

Outcome: Treated successfully with a 6-week course of oral terbinafine.

Case 5: The Folliculitis Mimic

Patient: A 45-year-old male farmer presented with multiple, tender, erythematous follicular papules and superficial pustules on the mandibular area (beard region) (Figure 8).

Examination: Initially treated with oral cephalexin for suspected bacterial folliculitis, with no improvement. Direct microscopy revealed ectothrix fungal elements around the hair shafts that proved on culture to be Trichophyton schoenleinii (Figure 9).

Outcome: Diagnosed as Tinea barbae. Treated with oral terbinafine and warm compresses; resolved in 6 weeks.

Case 6: The Lichen Planus Actinicus Mimic

Patient: A 28-year-old female presented with violaceous, Annular plaques with slight central hypopigmentation with a subtle adherent scale distributed over the forehead and eminence of right cheek, sparing the nasolabial folds (Figure 10).

Examination: Oral mucosa and nails were normal. A biopsy was initially considered; however, KOH from a scaly papule was positive for hyphae. Culture identified *Microsporum gypseum*.

Outcome: Treated with topical sertaconazole and systemic itraconazole 100 mg daily for 6 weeks; lesions flattened and repigmented.

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Case 7: The Rosacea Mimic

Patient: A 39-year-old woman with persistent centrofacial erythema, telangiectasia (mild), and numerous inflammatory papules and pustules.

Examination: She had failed multiple courses of metronidazole gel and oral doxycycline. The lack of true telangiectasia, the presence of fine scaling, and the unilateral predominance prompted a fungal scrape (Figure 11). KOH was positive. Culture grew *T. rubrum*.

Outcome: Treated with oral itraconazole 100 mg twice daily for 8 weeks; the lesions disappeard completely.

Case 8: The Pustular Psoriasis Mimic

Patient: A 32-year-old male presented with sterile-appearing, yellow-brown pustules superimposed on erythematous, scaly plaques bilateral and symmetrical on the groins and extending down to the inner thighs (Figure 12).

Examination: Pustular psoriasis was the initial diagnosis. He was prescribed methotrexate, but his skin condition worsened. A biopsy revealed spongiotic dermatitis with fungal hyphae, and culture grew *T. rubrum*.

Outcome: Methotrexate was stopped. He was treated with oral terbinafine for 6 weeks, resulting in dramatic clearing.

Case 9: Dermatitis Cosmetica Mimic

Patient: A 35-year-old female presented with diffuse erythema and scale of the face of three months' duration. She was treated with topical steroids many times and showed only partial response. Removal of the scalp and face cover showed extension of the lesion to the neck with a well-defined advancing border (figure 13). This suggested the fungal infection diagnosis, which was proved by positive KOH examination.

Outcome: Treated with oral itraconazole 100 mg twice daily for 8 weeks; the lesions disappeared completely.

4.DISCUSSION

4.1. The Pathophysiological Basis for Clinical Polymorphism

The clinical morphology of a dermatophyte infection is not a fixed property of the organism but is largely dictated by the host's immune response.

Cell-Mediated Immunity (CMI): The classic inflammatory, annular lesion is driven by a robust delayed-type hypersensitivity (DTH) reaction to fungal antigens. When the host's CMI is intact, the inflammatory response confines the fungus to the stratum corneum, creating the "ring" as the fungus spreads centrifugally and the center clears [7].

Tinea Incognito: Topical corticosteroids disrupt CMI by inhibiting macrophage migration, reducing IL-1 and TNF-alpha production, and decreasing the expression of adhesion molecules on endothelial cells. This turns a classic annular plaque into a diffuse, erythematous, crusted, or papulopustular lesion (Cases 2, 3) [8].

Strain Virulence Factors: T. rubrum is particularly notorious for its ability to induce minimal inflammation, leading to chronic, subtle, and scaly presentations (e.g., eczema-like). Conversely, *T. mentagrophytes* and *M. canis* often provoke a brisk inflammatory response, presenting as kerion or severe folliculitis [9]. There is an increasing incidence of superficial fungal infection which may be the most important cause of appearnce of atypical presentation in addition to emergence of new resistant species and the misuse of topical or even systemic steroids by medical, paramedical or even non medical persons (10,11)

4.2. The Diagnostic Imperative: Beyond Clinical Gestalt

Our case series demonstrates that relying solely on clinical morphology in dermatophyte infections is fraught with risk. To avoid diagnostic errors, we strongly advocate for the "Three Pillars of Mycological Diagnosis":

Direct Microscopy (KOH Preparation): The rapid, bedside gold standard. A 20% KOH with DMSO and calcofluor white (for fluorescence) can detect hyphae in <5 minutes with high sensitivity [10]. We emphasize that every scaly lesion that fails to

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respond to conventional treatment should undergo KOH examination (12).

Fungal Culture: Essential for identifying the specific dermatophyte species and guiding systemic therapy (e.g., *M. canis* often requires griseofulvin, while *T. rubrum* responds well to terbinafine). Culture takes 2-4 weeks but remains the definitive diagnostic test [11].

Skin Biopsy (with PAS staining): In cases where KOH and culture are negative (e.g., post-treatment or tinea incognito), a skin biopsy with Periodic acid-Schiff (PAS) stain is highly sensitive and can reveal fungal elements within the stratum corneum that are invisible on standard H&E staining [12].

4.3. Proposed Diagnostic Algorithm

We propose the following stepwise algorithm for practitioners evaluating a suspicious scaly rash:

Step 1: Assess for classic "ringworm" morphology. If present, perform KOH and treat empirically if positive.

Step 2: If classic morphology is absent (atypical or mimic), but the patient has risk factors (immunosuppression, prior steroid use, poor response to standard treatments), perform KOH immediately. Do not wait.

Step 3: If KOH is negative but clinical suspicion remains high, obtain a fungal culture and consider a 4-mm punch biopsy for PAS staining.

Step 4: Initiate systemic antifungal therapy based on culture sensitivity and lesion location (e.g., terbinafine for nail/skin, griseofulvin/terbinafine for scalp).

4.4. Clinical Pearls to Distinguish Mimics

(See Table 1 for details on distinguishing features)

5. CONCLUSION

The dermatophytes have successfully joined the ranks of the "Great Imitators" in medicine. Our series highlights nine distinct clinical masks worn by these common fungi, ranging from alopecia to psoriasis. The key takeaway for the practicing clinician is simple yet critical: do not be deceived by morphology. In an era of increasing topical corticosteroid misuse and rising immunocompromised populations, the incidence of atypical tinea is likely to rise. We recommend a low threshold for performing KOH preparations and fungal cultures on any scaly, inflammatory, or pustular eruption that is chronic, recurrent, or unresponsive to first-line therapies. A "Fungal-First" diagnostic approach will not only spare patients from unnecessary immunosuppression but will also provide a rapid, curative pathway through targeted antifungal therapy.

Declarations

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Approval: This study was approved by the Institutional Review Board of the Faculty of Medicine, University of Kufa, Iraq. All procedures were performed in accordance with the ethical standards of the Helsinki Declaration (2013).

Patient Consent: Written informed consent was obtained from all patients (or their guardians) for participation and for the publication of de-identified clinical photographs.

Conflict of Interest: The authors declare that they have no competing financial or non-financial interests that could have influenced the design, conduct, or reporting of this study.

Data Availability: The datasets generated and/or analyzed during this study are not publicly available to maintain patient confidentiality but are available from the corresponding author upon reasonable request and with appropriate institutional approvals.

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Tables

Table 1: The Nine Masqueraders of Dermatophytosis

Mimicked Dermatosis	Typical Site	Key Clinical Clue to Fungal Etiology	Common Causative Organism
Alopecia Areata	Scalp	Black dots, perifollicular scale, adenopathy	*T. tonsurans*
Contact Dermatitis	Hands, Feet	No allergen history; lacks uniform vesicles	*T. rubrum*
Discoid Eczema	Extremities	Poor steroid response; visible border	*E. floccosum*
Tinea Imbricata	Trunk	Concentric, overlapping rings	*T. concentricum* / *T. rubrum*
Folliculitis	Beard, Extremities	Involvement of hair shafts; negative bacterial culture	*T. mentagrophytes*
Lichen Planus Actinicus	Face (Malar)	Violaceous color, lack of Wickham striae	*M. gypseum*
Rosacea	Centrofacial	Prominent scale, asymmetry, steroid	*T. rubrum*

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Mimicked Dermatosis	Typical Site	Key Clinical Clue to Fungal Etiology	Common Causative Organism
		use history	
Pustular Psoriasis	Groins, Upper thigh	Unilateral, negative nail pitting, KOH positive	*T. rubrum*
Dermatitis Cosmetica	Face	Diffuse erythema, scales on the face with advancing well defined border extending to the neck	*T. rubrum*

Figure Legends

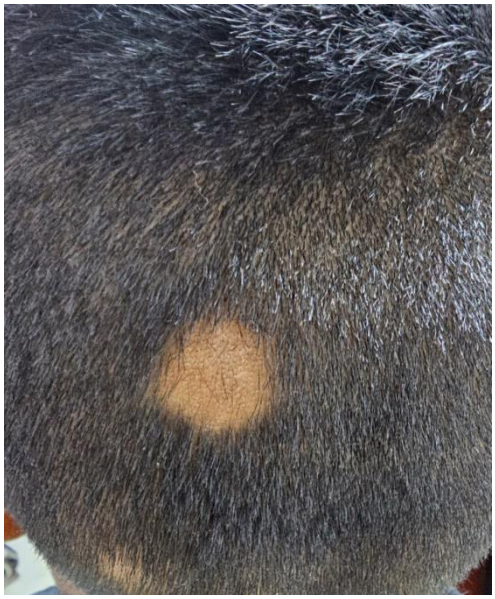

Figure 1: Alopecia Areata–Like: A non-inflammatory two patches of alopecia on the occipital scalp.

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Figure 2: Dermoscopy of one of the lesions in Figure 1 showed Zigzag hair (Green arrows), Black dots (Yellow arrows), Comma hair (Blue arrows), Broken hair (Red arrow), and dirty background with perifollicular scales.

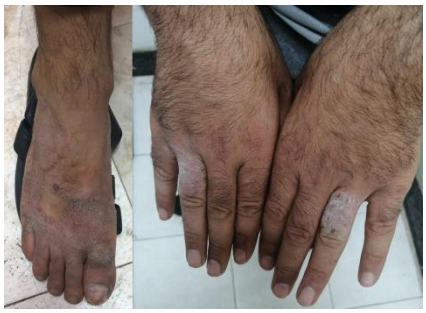

Figure 3: Contact Dermatitis–Like: Ill defined scaly and vesicular plaques on both hands and right foot.

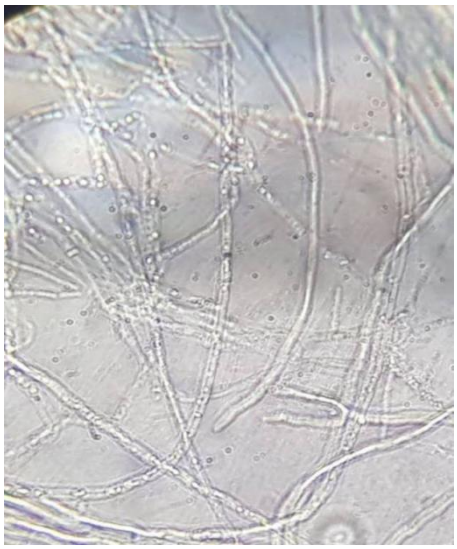

Figure 4: Culture lesion in Figure 3, was positive for *Trichophyton rubrum*.

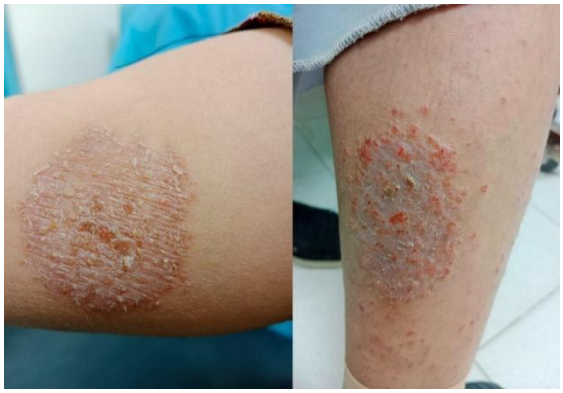

Figure 5: Discoid Eczema–Like: Coin-shaped hyperkeratotic plaques on the forearm and leg.

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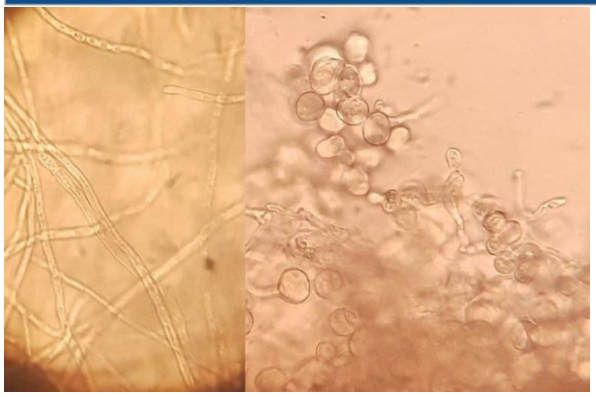

Figure 6: KOH examination was positive and culture grew *Epidermophyton floccosum*.

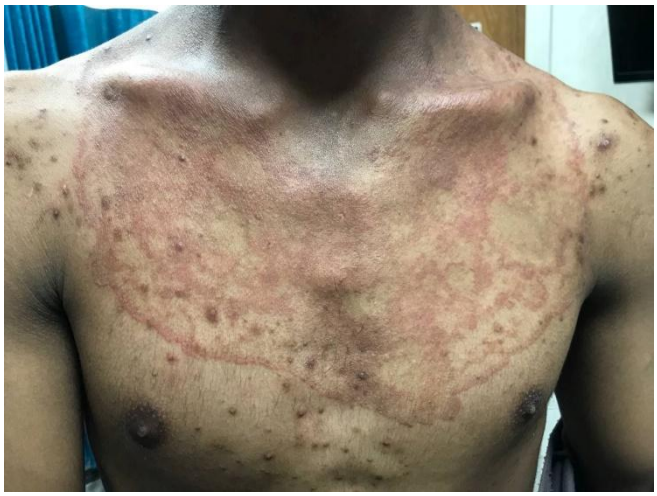

Figure 7: Tinea Imbricata–Like: Concentric rings of scaling on the anterior upper trunk.

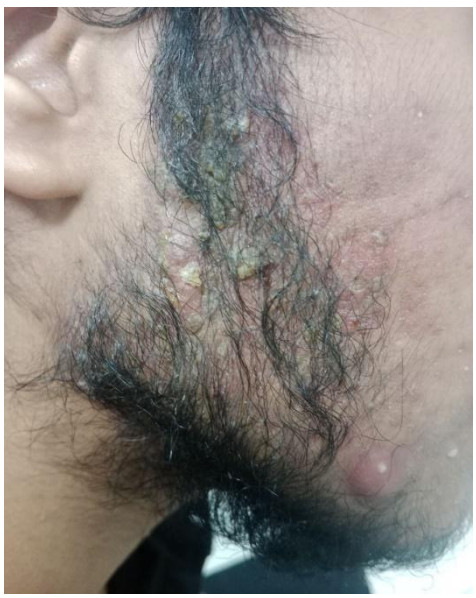

Figure 8: Folliculitis–Like: Pustular follicular papules on the mandible.

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Figure 9: Culture of the lesion in figure 8, grew *Trichophyton schoenleinii*.

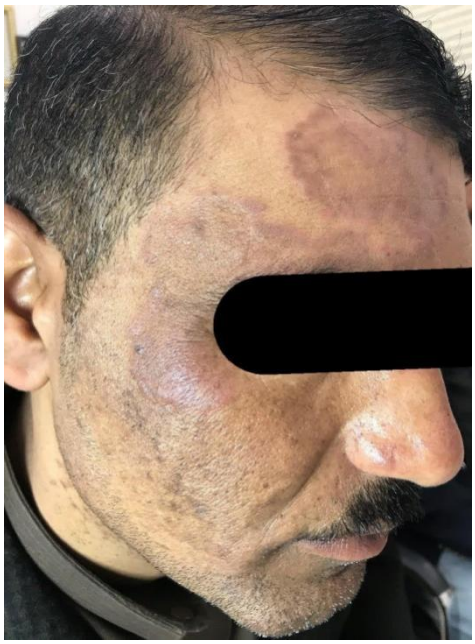

Figure 10: Lichen Planus Actinicus–Like: Violaceous plaques on the forehead and cheek.

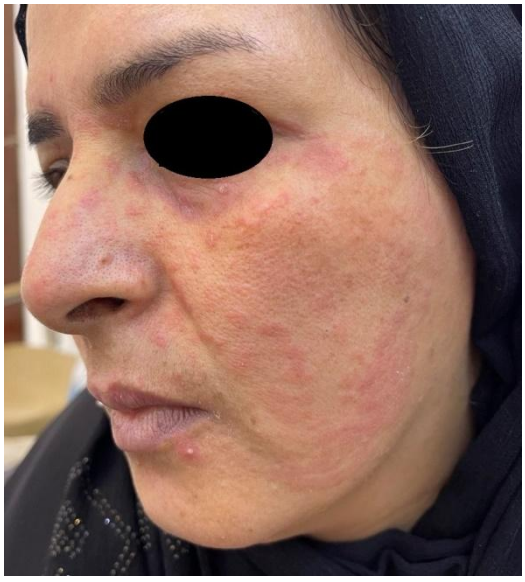

Figure 11: Rosacea–Like: Centrofacial erythema. papules and few pustules with scaling.

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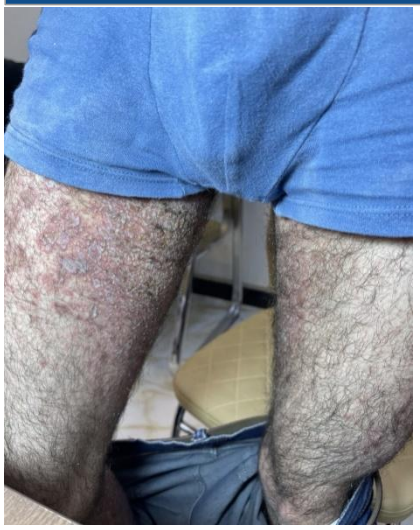

Figure 12: Pustular Psoriasis–Like: Pustules on an erythematous scaly base on the groins and upper thighs.

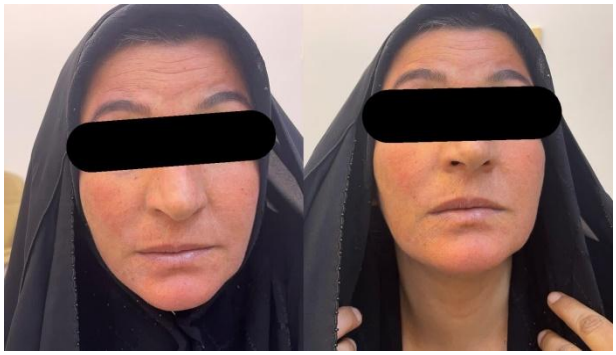

Figure 13: Dermatitis cosmetica-like: diffuse erythema and scales on the face with advancing well defined border on the upper neck.

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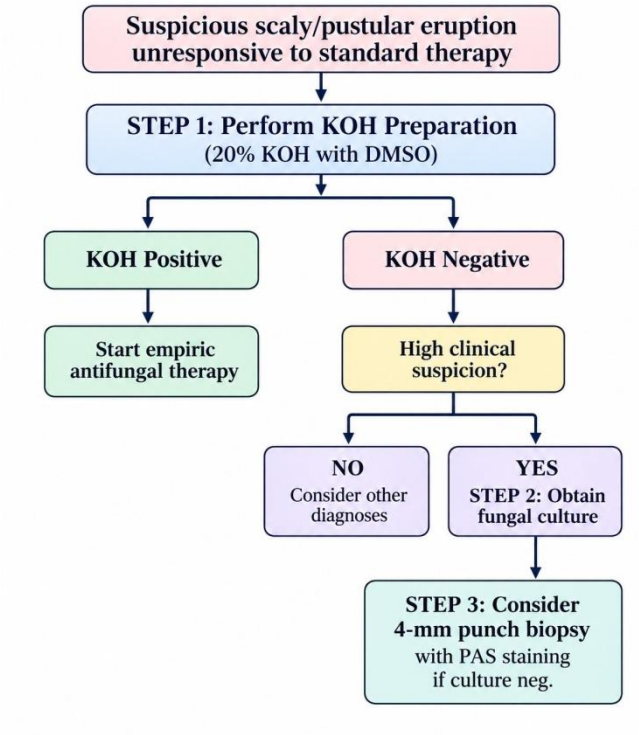

Figure 14: Proposed diagnostic algorithm for suspected atypical dermatophytosis.