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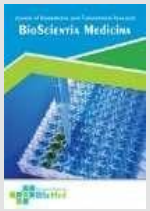

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Nummular Dermatitis Masquerading as Recalcitrant Tinea Corporis: A Diagnostic Pitfall Resolved by Dermoscopy and Histopathology

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A B S T R A C T

Background: The morphological mimicry between nummular dermatitis (ND) and tinea corporis represents a significant source of diagnostic error in clinical practice. This convergence on a similar clinical phenotype—the annular plaque—often leads to inappropriate antifungal therapy and a protracted, debilitating course for the patient, as powerfully illustrated in this case. **Case presentation:** We present the case of a 22-year-old female with a five-year history of chronic, recalcitrant, and intensely pruritic annular plaques on her lower legs, with a baseline pruritus score of 8/10 on a Visual Analog Scale (VAS). Previous treatments based on presumptive diagnoses, including potent topical corticosteroids and systemic antifungals, had failed. This report demonstrates a pivot from a morphology-based assessment to a multimodal diagnostic algorithm. The cornerstone of this shift was dermoscopy, which revealed patchy, irregularly distributed red dotted vessels—a classic signature of an eczematous process—and a notable absence of features suggestive of a dermatophyte infection. The diagnosis was confirmed by histopathology. A strong correlation between disease flares and psychosocial stressors was identified, and a holistic management plan targeting the cutaneous inflammation, the compromised skin barrier, and the neurogenic triggers led to a rapid and substantial improvement in her clinical status and quality of life, with the Dermatology Life Quality Index (DLQI) score decreasing from 9 to 3 and the pruritus VAS score dropping to 1/10. **Conclusion:** This case argues for a revised diagnostic paradigm for chronic annular lesions, one that de-emphasizes ambiguous morphology and prioritizes the integration of a detailed psychodermatological history with in-vivo microscopic analysis via dermoscopy. This approach not only prevents therapeutic futility but also underscores that successful, long-term management of ND is contingent upon a patient-centered strategy that addresses the interconnectedness of the skin barrier, the immune system, and the psycho-neuro-cutaneous axis.

1. Introduction

The diagnosis of inflammatory skin disease is a complex intellectual exercise, demanding a synthesis of pattern recognition, pathophysiological reasoning, and clinical acumen.[1] A formidable challenge arises when disparate pathological processes converge on a single, often misleading, morphological endpoint. The annular, or ring-shaped, plaque is one such endpoint, and its interpretation is a frequent source of clinical error, leading to therapeutic inertia and significant patient morbidity.[2] This manuscript explores this fundamental challenge through the lens of a compelling case that pits an endogenous inflammatory disease against an exogenous infectious one.[3] The core diagnostic tension lies in the distinction between nummular dermatitis (ND), an "inside-out" pathology driven by internal dysregulation of the skin barrier and immune system, and tinea corporis, a

quintessential "outside-in" pathology caused by an external pathogen invading the stratum corneum. ND, also known as discoid eczema, is an "inside-out" process fundamentally rooted in a primary defect of the epidermal barrier.[4] This dysfunction is a complex molecular phenomenon that extends far beyond simple xerosis cutis (dry skin). It involves quantifiable deficiencies in the key structural and functional components of the stratum corneum. The lipid matrix, composed of a precise ratio of ceramides, cholesterol, and free fatty acids, is often disrupted, with a notable reduction in ceramide levels leading to disordered lipid lamellae, increased transepidermal water loss (TEWL), and a lowered threshold for irritation.[5] Furthermore, the integrity of the cornified envelope can be compromised by an acquired deficiency in filaggrin, a protein essential for keratin filament aggregation and the production of natural moisturizing factors (NMFs). This deficiency, driven by the local inflammatory milieu itself, can occur even in non-atopic individuals and further degrades the skin's ability to retain moisture and resist environmental insults.[6]

This structurally weakened barrier becomes a permissive environment, allowing the penetration of irritants, allergens, and microbes, which in turn triggers a complex and often biphasic immune response. Acute lesions of ND are typically characterized by a Th2-dominant immune polarization. The release of cytokines such as Interleukin-4 (IL-4) and Interleukin-13 (IL-13) drives eosinophilic inflammation, promotes IgE production, and, critically, further downregulates the expression of key barrier proteins like loricrin and filaggrin, thus perpetuating the barrier defect.[7] As lesions become chronic and lichenified, the immune profile often shifts towards a Th1/Th17-dominant response. The increased presence of Interferon-gamma (IFN-γ) and IL-17 drives the keratinocyte hyperproliferation that manifests clinically as the thickened, scaly plaques characteristic of chronic eczema. This inflammatory state is potently exacerbated by colonization with *Staphylococcus aureus*. Bacterial components are recognized by innate immune sensors like Toll-like receptor 2 (TLR2) on keratinocytes and Langerhans cells, while secreted superantigens can induce a massive, polyclonal T-cell activation, dramatically amplifying the underlying inflammatory cascade.[8] Conversely, tinea corporis is a pure "outside-in" process initiated by the invasion and proliferation of dermatophyte fungi within the keratinized stratum corneum. The organism spreads centrifugally, eliciting a host cell-mediated immune response at the advancing edge, which results in the classic erythematous, scaly border of the "ringworm" lesion.

The diagnostic conundrum arises because these two distinct pathophysiological pathways can produce remarkably similar clinical pictures. This necessitates a broad and carefully considered differential diagnosis for any annular plaque. Plaque psoriasis, for instance, can present with annular plaques but is distinguished by its unique, silvery, micaceous scale and, upon removal, the pathognomonic Auspitz sign of pinpoint bleeding. Atopic dermatitis, for which ND may be a morphological variant, is typically suggested by a personal or family history of atopy and the classic involvement of flexural sites.[9] Allergic contact dermatitis requires a meticulous history to identify a causative exposure and confirmation by epicutaneous patch testing. Critically, for any chronic, pruritic, and treatment-refractory plaque, the astute clinician must consider early-stage mycosis fungoides, a cutaneous T-cell lymphoma whose deceptive inflammatory appearance often necessitates histopathological analysis with immunohistochemistry to rule out. The ambiguity inherent in clinical morphology often leads to a cascade of therapeutic trials based on unconfirmed assumptions, causing significant patient distress and a diminished quality of life. To break this cycle, a paradigm shift is required—moving away from simple visual inspection towards a more integrated diagnostic process. This involves leveraging adjunctive tools like dermoscopy, a non-invasive technique that bridges the gap between clinical and histopathological evaluation by revealing subsurface patterns invisible to the naked eye.[10] Therefore, this report aims to argue for a revised diagnostic algorithm for chronic annular

plaques. The novelty and primary aim of this manuscript is to demonstrate that by de-emphasizing ambiguous clinical morphology and prioritizing an integrated analysis of the patient's psychodermatological history and the in-vivo microscopic data from dermoscopy, clinicians can dismantle diagnostic pitfalls, prevent therapeutic futility, and deliver more effective, patient-centered care.

2. Case Presentation

A 22-year-old female was referred to our tertiary care center for the evaluation of a chronic, refractory skin condition that had profoundly impacted her life for five years. Her chief complaint was intensely pruritic, enlarging reddish patches on both lower legs. The timeline presented in Figure 1 provides a compelling visual narrative of a five-year diagnostic and therapeutic odyssey, illustrating a classic case of clinical mimicry and diagnostic inertia. The journey begins five years prior with an seemingly unremarkable initial onset of itchy papules that coalesced into a larger patch, for which no treatment was sought. This quiescent start belies the complexity that would follow. Approximately 4.5 years ago, the patient's condition prompted a medical consultation, leading to a presumptive diagnosis of unspecified dermatitis and the initiation of standard anti-inflammatory therapy. The recorded outcome of "no improvement" marks the first instance of therapeutic failure, a theme that characterizes the subsequent years. As the condition progressed and spread, it was re-labeled as refractory dermatitis, and the treatment was escalated to a more potent topical corticosteroid. The continued lack of improvement, with patches even enlarging, underscored the inefficacy of a purely anti-inflammatory approach and highlighted that the underlying pathology was not being adequately addressed. A pivotal and critical turning point occurred about one year ago. The annular, or ring-shaped, morphology of the lesions led clinicians down a different diagnostic path, resulting in a misdiagnosis of suspected tinea corporis, a fungal infection. This shift from an inflammatory to an infectious etiology prompted a complete change in therapy to antifungal agents. The outcome was not merely a lack of improvement but a definitive worsening of the condition, with intensified pruritus. This adverse result is a powerful indicator of a diagnostic pitfall, where treatment aimed at an incorrect pathology can be detrimental. The timeline culminates in the patient's referral to tertiary care, a necessary step after years of failed treatments. It is at this stage that crucial new information came to light: the patient's own identification of a link between her symptoms and psychosocial stress, as well as a noted improvement after a change in her environment.

Upon presentation, a thorough physical and dermatological examination was conducted. The patient was in good general health with stable vital signs. Figure 2 provides a comprehensive macroscopic summary of the patient's clinical presentation, integrating detailed photographic evidence with a systematic breakdown of the dermatological findings. The clinical photographs vividly display multiple erythematous plaques symmetrically distributed on the lower third of both legs, confirming the location and appearance of the pathology. The examination details a precise morphology of the lesions, characterizing them as multiple, well-demarcated, annular (ring-shaped), and discoid (coin-shaped) plaques, with a diameter ranging from 3 to 6 cm. A key feature noted is the evidence of central healing, which manifests as hypopigmentation surrounded by elevated, active borders composed of coalescing erythematous papules. The surface of these plaques is described as having a fine, whitish, and adherent scale, with an absence of vesicles or weeping, which suggests the condition is in a subacute to chronic inflammatory stage. Furthermore, the figure 2 documents significant secondary changes resulting from the disease's primary symptom of intense pruritus. The presence of scattered linear excoriations and mild lichenification (a thickening of the skin) serves as direct physical evidence of persistent scratching. Crucially, one of the key associated signs

noted is that the surrounding, uninvolved skin was markedly xerotic, or dry. This finding is highlighted as a vital clinical clue, pointing towards an underlying defect in the epidermal barrier function. A systematic panel of investigations was performed to deconstruct the initial diagnosis and build a new, evidence-based one.

Figure 1. Chronological summary of patient's medical history.

Figure 2. Clinical and dermatological examination.

Figure 3 masterfully encapsulates the multimodal diagnostic strategy that was essential to resolving this complex clinical case. It visually documents the systematic process of moving from a broad differential diagnosis to a definitive etiology by integrating patient-reported outcomes with a cascade of microscopic and cellular investigations. The figure illustrates not just the results of the tests, but the logical progression of a modern diagnostic workup. The investigation began by quantifying the patient's subjective experience, grounding the clinical findings in the reality of the disease's impact. A Dermatology Life Quality Index (DLQI) score of 9 and a severe Pruritus Visual Analog Scale (VAS) score of 8/10 established a baseline of a moderate impact on quality of life driven by intense itch, providing objective metrics against which therapeutic success could be measured. The process of deconstructing the presumptive diagnosis started with the most direct and crucial test: the Microscopic Fungal Exam (KOH). The negative result from this test provided the first piece of objective, contradictory evidence against the long-held suspicion of tinea corporis, effectively dismantling the previous diagnostic framework and necessitating a new

approach. The pivotal moment in this diagnostic journey is highlighted in the Dermoscopy Examination. This non-invasive, in-vivo microscopy was not merely an exclusionary test but an active diagnostic tool. The findings of patchy, irregularly distributed red dotted vessels and diffuse white scales were critical. As the figure notes, this pattern is the dermoscopic hallmark of an eczematous process and stands in stark contrast to the uniform vascular patterns of psoriasis or the peripheral scaling of tinea corporis. This single examination effectively resolved the primary diagnostic dilemma at the point of care, steering the diagnosis firmly toward dermatitis. Finally, the Histopathology Examination served as the gold-standard confirmation. The findings from the 4-mm punch biopsy revealed features of a non-specific chronic dermatitis, including mild acanthosis and focal parakeratosis. While these features are common to many inflammatory conditions, the most crucial finding was the negative PAS stain, which definitively excluded a fungal etiology at the cellular level. This result perfectly corroborated the dermoscopic impression and provided the final, conclusive piece of evidence.

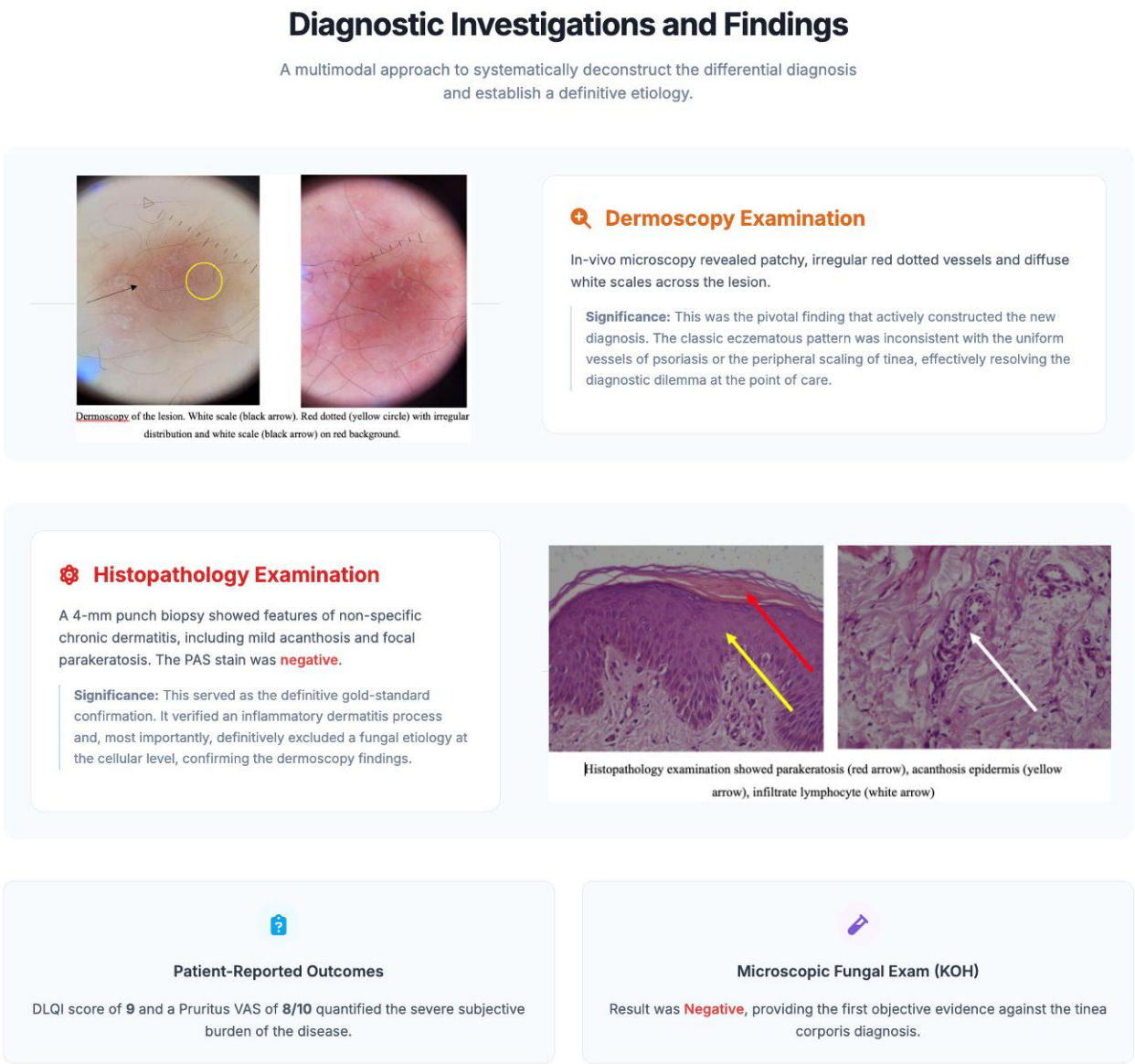

Figure 3. Diagnostic investigation and findings.

The final diagnosis of nummular dermatitis was established by synthesizing the compelling historical narrative, the clinical signs, and the unequivocal results of the diagnostic workup. A multi-pronged therapeutic strategy was designed, shifting from a simple lesion-directed approach to a patient-centered, pathophysiology-based plan. The foundation of the successful intervention was the Final Diagnosis of Nummular Dermatitis, which was definitively established following a thorough clinical and investigational workup. This precise diagnosis allowed for a targeted therapeutic plan, moving away from the previous ineffective and morphology-driven treatments. The core of the intervention is presented as a Multi-Pronged Management Strategy, a sophisticated approach that addresses the different facets of the disease's pathophysiology rather than just the visible lesions. This strategy was built upon three key pharmacological pillars: An Anti-inflammatory Pillar, utilizing Mometasone Furoate 0.1% Cream to directly suppress the underlying cutaneous immune response, thereby reducing the erythema and inflammation. A Barrier Repair Pillar, employing Urea 10% Cream to address the fundamental "inside-out" defect characteristic of eczematous conditions. This component aimed to restore the integrity of the epidermal barrier, improve hydration, and combat the background xerosis. A Symptom Control Pillar, consisting of a Cetirizine 10 mg Tablet, which was included to manage the patient's most debilitating symptom—severe pruritus—and to help break the deleterious itch-scratch cycle. Crucially, Figure 4 also highlights that this pharmacological strategy was built upon a foundation of non-pharmacological patient education, which focused on the avoidance of known triggers such as environmental irritants and psychosocial stress. The efficacy of this holistic approach is powerfully demonstrated in the Two-Week Follow-Up Outcome section. The success is quantified through two key metrics: a dramatic reduction in the Dermatology Life Quality Index (DLQI) score from 9 to 3, signifying a shift from a moderate to a minimal impact on the patient's quality of life, and a significant decrease in the Pruritus Visual Analog Scale (VAS) score from 8 to 1, indicating a change from severe to mild itch. This quantitative data is supported by qualitative and photographic evidence of clinical improvement. The plaques showed significant resolution, evolving into faint post-inflammatory macules with a marked reduction in erythema and pruritus. The high satisfaction reported by the patient serves as the ultimate confirmation of the treatment's success. In essence, Figure 4 provides a clear and compelling illustration of how a precise, evidence-based diagnosis coupled with a comprehensive management plan that targets the multiple drivers of a disease can lead to rapid and meaningful improvements in both objective clinical signs and the patient's overall well-being.

3. Discussion

The case presented offers a profound and instructive journey into the complexities of diagnosing and managing chronic inflammatory skin disease. The successful resolution of this patient's five-year ordeal was not merely the result of prescribing the correct medication; it was the result of a fundamental shift in diagnostic philosophy—from a reliance on ambiguous morphology to an integrated analysis of the patient's story and the microscopic evidence hidden just beneath the skin's surface.[11] The starting point of this patient's diagnostic challenge was the macroscopic appearance of her lesions: the annular plaque. This morphology is notoriously non-specific, representing a shared final pathway for numerous pathologies. In this case, it created a powerful clinical picture of tinea corporis, a diagnosis that was further entrenched by the failure of topical corticosteroids. This therapeutic failure was likely misinterpreted as evidence of tinea incognito. The pathophysiology of tinea incognito is a critical concept for clinicians; the application of corticosteroids suppresses the host's normal Th1-mediated cellular immune response required to contain and clear the dermatophyte infection.[12]

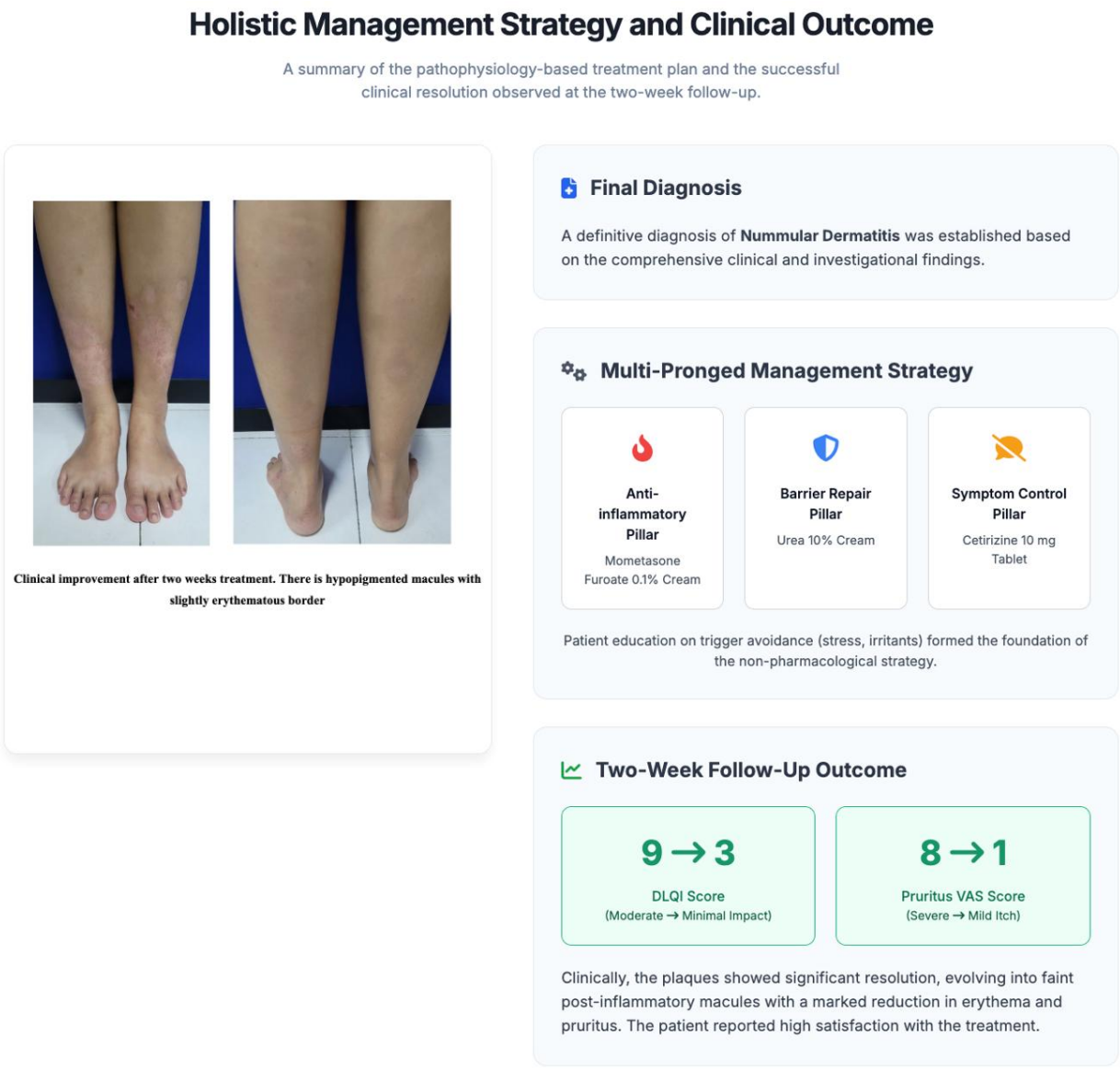

Figure 4. Holistic management strategy and clinical outcome.

This immunosuppression masks the classic inflammatory signs, leading to lesions with less scale, more diffuse erythema, and an uninhibited centrifugal spread of the fungus, deepening the diagnostic trap and solidifying the incorrect diagnosis.[13] The failure of anti-inflammatory therapy was paradoxically seen as confirmation of an infectious etiology, a classic cognitive error in clinical reasoning. The escape from this trap required a deliberate shift in observational scale from the macroscopic to the microscopic.[14] This began with the simple, yet crucial, KOH preparation, which provided the first piece of negative evidence against the prevailing hypothesis. However, it was the in-vivo microscopy of dermoscopy that provided the positive evidence needed to construct a new diagnosis. Dermoscopy allows visualization of the skin's micro-architecture, particularly the vascular patterns that are often unique to different inflammatory conditions.[15]

The dermoscopic profile of Nummular Dermatitis, as illustrated in Figure 5, is fundamentally a portrait of disorganization, perfectly mirroring the chaotic nature of an active eczematous reaction. The key to its diagnosis lies not in finding a single pathognomonic

feature, but in recognizing a characteristic constellation of irregular and unpredictable patterns.[16] The vascular pattern is the most revealing aspect. The figure describes this as "patchy and irregular dotted vessels". This seemingly simple description holds deep histopathological significance. The "dots" represent the tips of dilated capillary loops within the dermal papillae, which have become more visible due to overlying epidermal changes. In dermatitis, the underlying inflammatory infiltrate of lymphocytes and eosinophils is often patchy and haphazard, and the resultant edema (spongiosis) is non-uniform. This anatomical chaos is directly reflected in the dermoscopic view: the red dots vary in size, shape, and spatial distribution across the lesion, lacking any discernible order. This stands in stark contrast to more organized inflammatory processes and serves as a primary diagnostic clue. The scale characteristics provide further evidence. The figure notes the presence of "diffuse white scales" over the lesion surface. This corresponds to the subacute or chronic nature of the condition, reflecting areas of parakeratosis (retained nuclei in the stratum corneum) and hyperkeratosis. A highly specific clue for acute phases of nummular dermatitis, also mentioned in the figure, is the presence of yellowish serocrusts, sometimes referred to as the "yellow clod sign". These crusts are the macroscopic and dermoscopic correlate of intense, acute spongiosis, representing the dried serous exudate that has oozed from within the epidermis. Their presence is a powerful confirmation of an acute eczematous flare. Ultimately, the specific clues section of Figure 5 synthesizes this information into a cohesive diagnostic principle: the key to identifying nummular dermatitis is its "patchy, disorganized" nature. The diagnosis is achieved by recognizing this characteristic lack of a predictable pattern. The chaotic arrangement of the vessels and the diffuse nature of the scale are the strongest and most reliable indicators of an underlying eczematous process.[17] In direct and stark contrast to the disorganization of dermatitis, the dermoscopic profile of Plaque Psoriasis is a paradigm of order and uniformity. Its features are predictable, regular, and reflect the highly organized and proliferative nature of the disease. The vascular pattern is the most pathognomonic feature. Figure 5 describes this as "uniform and regularly distributed dotted vessels". This remarkable regularity is a direct window into the underlying histopathology. Psoriasis is characterized by a very regular pattern of epidermal hyperplasia (psoriasiform acanthosis), with uniform elongation and thickening of the rete ridges.[18] Within the dermal papillae that sit between these ridges, the capillary loops become uniformly dilated and tortuous. It is the tips of these evenly spaced, monomorphic vessels that appear as perfectly round, evenly distributed red dots on dermoscopy. This anatomical precision is what creates the striking visual of uniformity that is the hallmark of psoriasis. The scale characteristics in psoriasis are also distinct. The figure describes them as "silvery-white, cohesive scales" that are often thick. This corresponds histopathologically to confluent and extensive parakeratosis, often containing collections of neutrophils (Munro's microabscesses). A key clinical point noted in the figure is that these thick, micaceous scales can often obscure the underlying vascular pattern. To properly evaluate the vessels, it may be necessary to gently remove the surface scale, often with the application of immersion fluid or oil, to unmask the highly informative vascular signature beneath. The specific clues section for psoriasis distills the diagnosis into a single, powerful concept: "uniformity". The highly organized, predictable, and almost geometric arrangement of the monomorphic red dots is the single most reliable feature that differentiates the structured inflammation of psoriasis from the chaotic inflammation of dermatitis.[19] The dermoscopic evaluation of tinea corporis requires a shift in focus. While the previous two conditions are primarily defined by their vascular architecture, the diagnosis of this superficial fungal infection relies more heavily on identifying characteristic surface features, particularly at the lesion's edge. The vascular pattern in tinea is described as "often sparse and non-

specific". While an inflammatory response to the fungus can certainly produce some dotted vessels, they typically lack the specific patterns of either dermatitis or psoriasis. They are not a primary or reliable diagnostic feature. The figure does note that "comma-shaped vessels" can sometimes be seen, which can serve as a subtle clue, but these are not universally present. The diagnostic key lies in the scale characteristics. As Figure 5 emphatically states, the most characteristic feature is a "peripheral or 'leading edge' distribution of fine white scales". This dermoscopic finding is a direct visualization of the infection's pathophysiology. The dermatophyte fungi are most metabolically active and are actively invading the stratum corneum at the advancing, centrifugal border of the lesion. It is here that the host's inflammatory response and the disruption of keratin are most prominent, resulting in the characteristic scaling at the periphery. The center of the lesion, where the infection may be waning or has already passed, often shows less scale, leading to the classic "ringworm" appearance. Therefore, the specific clues section provides a clear directive for the clinician: "look for the 'scales on the edge'". The anatomical location of the scale is far more diagnostically significant than the morphology of the underlying vascular pattern when attempting to differentiate tinea from its inflammatory mimics.[20]

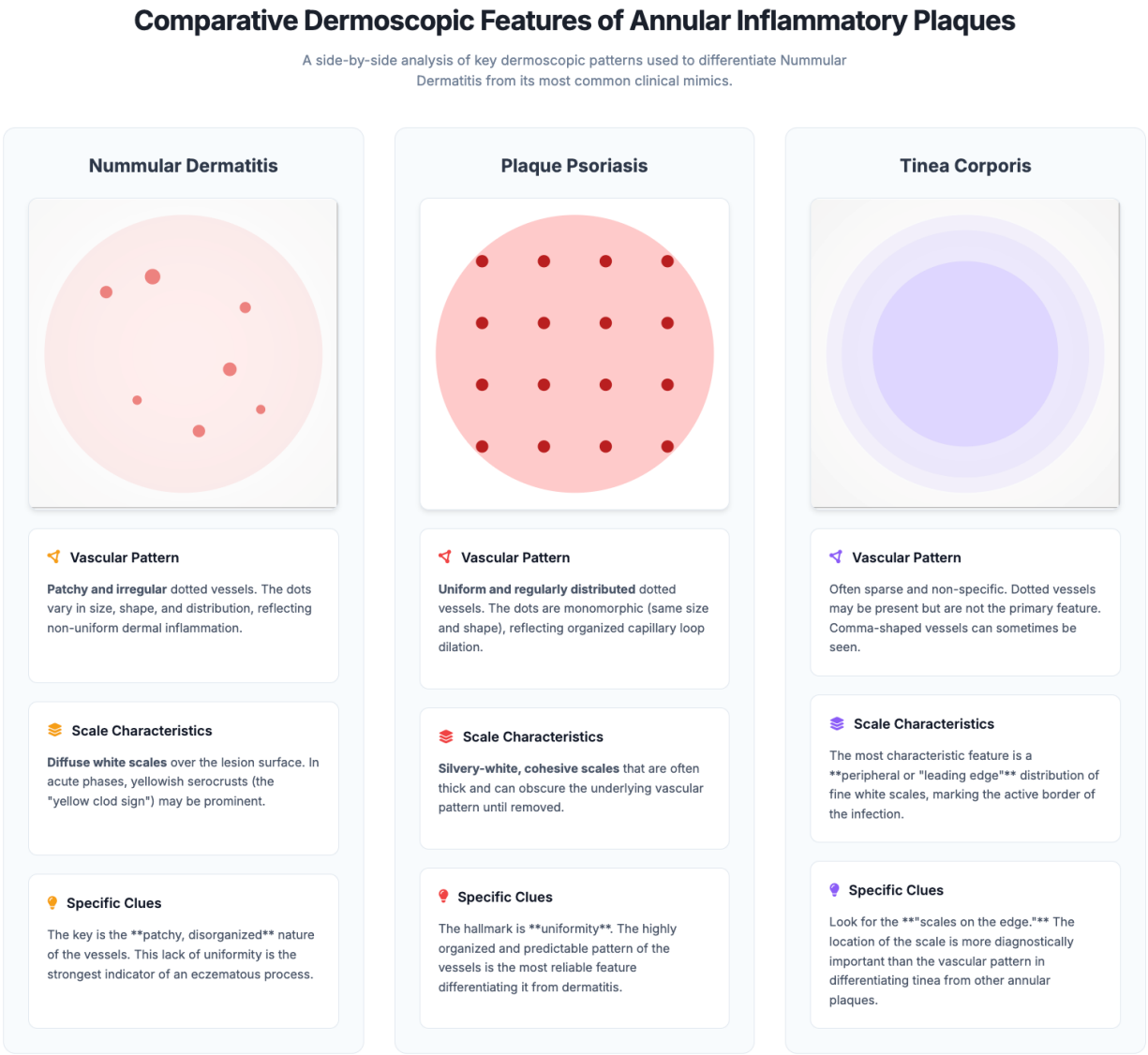

Figure 5. Comparative dermoscopic features of annular inflammatory plaques.

This systematic comparison, made possible at the bedside with dermoscopy, definitively moved the diagnosis away from tinea and psoriasis and strongly toward an eczematous process. The final step in this multiscale analysis was the histopathology. The report of "non-specific chronic dermatitis" must be interpreted within the context of the patient's history and the disease's natural evolution. Eczema is a dynamic process. An acute lesion is defined by marked spongiosis (intercellular edema) leading to the formation of intraepidermal vesicles, often accompanied by a prominent eosinophilic infiltrate and superficial dermal edema. A subacute lesion, as was likely biopsied here, shows the classic triad of acanthosis (epidermal thickening), parakeratosis (retained nuclei in the stratum corneum), and persistent, though often less dramatic, spongiosis. A chronic, heavily scratched lesion transitions to a state of lichen simplex chronicus, with prominent, irregular epidermal hyperplasia, hypergranulosis, compact hyperkeratosis, and vertical streaking of collagen bundles in the papillary dermis, reflecting the skin's response to chronic rubbing. The patient's biopsy, showing mild acanthosis, parakeratosis, and "uncertain" spongiosis, fits perfectly into the subacute/chronic spectrum. The uncertainty of the spongiosis is an expected "treatment effect" from her prior use of potent corticosteroids, which would have suppressed the edema. Despite this modification, the biopsy was invaluable: it confirmed an inflammatory process consistent with dermatitis, its features were inconsistent with psoriasis (lacking confluent parakeratosis or Munro's microabscesses), and the negative PAS stain definitively excluded a fungal etiology.

The resolution of the diagnostic puzzle is only half the story. The more profound lesson from this case lies in understanding why this patient's dermatitis was so chronic and recalcitrant. The answer is found in her own narrative—the direct link between her emotional state and her skin's inflammation. This is a clinical manifestation of the psycho-neuro-cutaneous axis, a complex, bidirectional communication system that functionally integrates the nervous system, the immune system, and the skin. The patient's stressful work environment served as the primary trigger, initiating a cascade of events beginning in the brain. Perception of stress activates two major pathways: the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system. While the systemic release of cortisol and catecholamines plays a role, the more immediate and potent effects in the skin are driven by neurogenic inflammation. The skin is densely innervated by sensory nerve fibers that act as conduits for stress signals. In response to stress, these nerves release a powerful cocktail of neuropeptides, including Substance P (SP), Calcitonin Gene-Related Peptide (CGRP), Vasoactive Intestinal Peptide (VIP), and Neuropeptide Y (NPY). These neuropeptides are not passive messengers; they are potent biological effectors. SP, for instance, binds to its neurokinin-1 receptor on dermal blood vessels, causing vasodilation and increased vascular permeability—the direct cause of the erythema seen in her plaques. It is also a powerful degranulator of cutaneous mast cells, triggering the release of histamine, tryptase, and other pro-inflammatory molecules that both drive inflammation and generate the intense sensation of pruritus. This establishes the first link in the vicious cycle: Stress causes itch. The sensation of pruritus is transmitted via C-fibers to the spinal cord and then to the brain, where it is processed in areas associated not just with sensation but also with emotion and motor planning. The nearly irresistible urge to scratch is the result. The act of scratching provides momentary relief by activating pain-inhibitory circuits, but at a devastating cost to the skin. The physical trauma from scratching decimates the already-compromised epidermal barrier, allowing further ingress of irritants and microbes. This damage also sends a distress signal from the keratinocytes themselves. Damaged keratinocytes release pro-inflammatory cytokines and, critically, nerve growth factor (NGF). NGF is a key neurotrophin that promotes the survival, growth, and sprouting of local sensory nerve endings, creating a state of peripheral sensitization.[19] The skin becomes

neurologically hyper-reactive, amplifying itch signals and lowering the threshold for their activation (a phenomenon known as alloknesis, where non-itchy stimuli are perceived as itchy). This completes the cycle: Itch leads to scratching, and scratching leads to more inflammation and more itchiness. This entire neuro-inflammatory cascade is directly linked back to the fundamental barrier defect. Neuropeptides like SP and stress hormones like cortisol have been shown to directly inhibit the synthesis of key barrier lipids, such as ceramides, and structural proteins, such as loricrin and involucrin, by keratinocytes. This creates a devastating feedback loop: stress impairs the barrier, a faulty barrier allows irritant entry, which causes more inflammation and stress, which in turn further degrades the barrier. The patient's experience is a perfect clinical model of this cycle. Her work-related stress continuously fueled the neurogenic inflammation and pruritus. Her scratching perpetuated the skin barrier damage and chronic inflammation. The visible and uncomfortable nature of her skin disease then became a new source of stress, completing the feedback loop. The cycle was only broken when the primary trigger—the external psychosocial stressor—was removed by her resignation. This act of self-care led to a down-regulation of the neuro-inflammatory cascade, allowing the skin to begin healing even before our targeted therapy was initiated. This demonstrates with absolute clarity that for this patient, the dermatitis was not just a "skin problem"; it was the cutaneous expression of systemic distress.

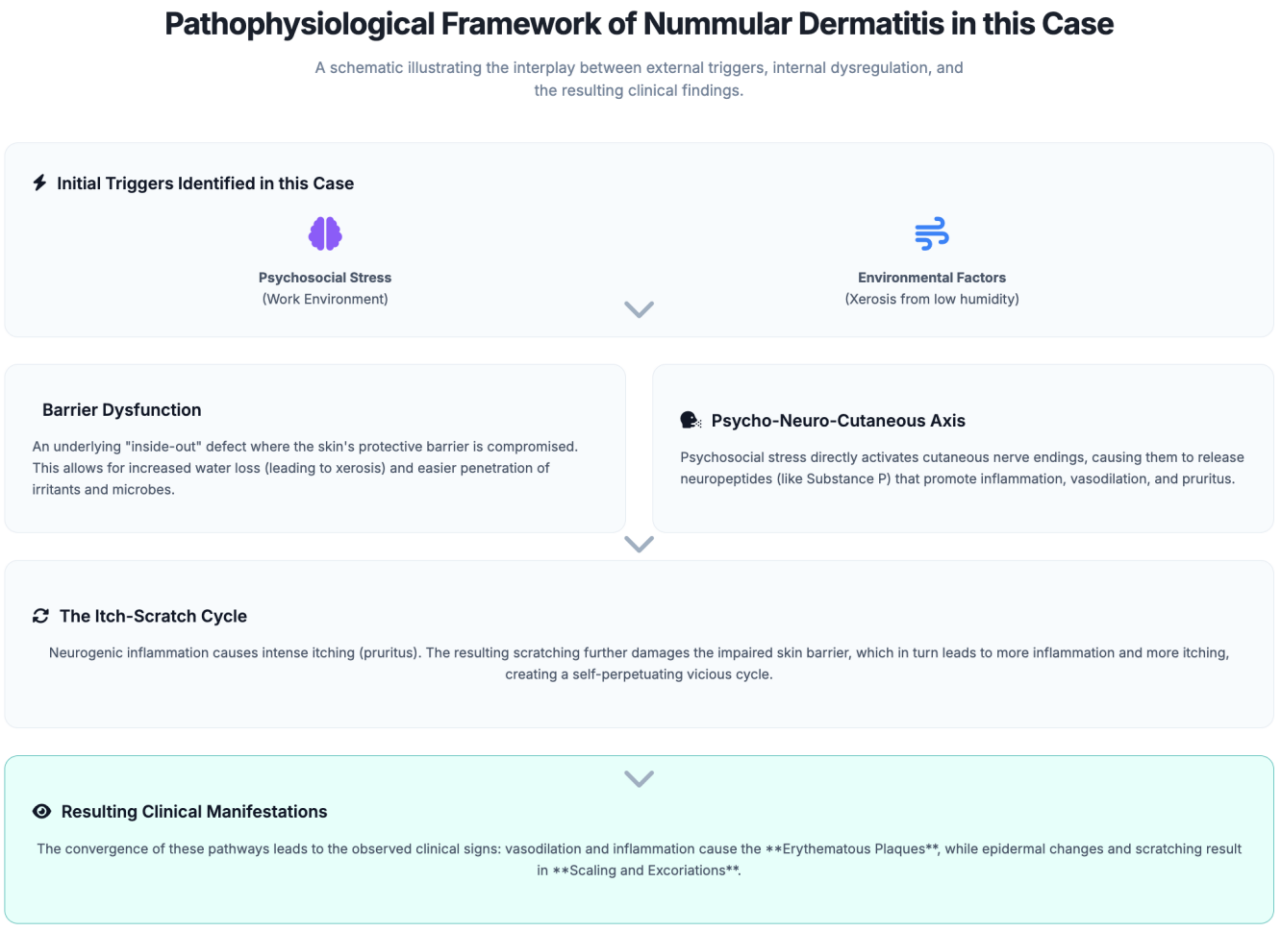

Figure 6. Pathophysiological framework of nummular dermatitis.

Figure 6 presents a sophisticated and highly informative pathophysiological framework that is specifically tailored to the nuances of the case in question. It moves beyond a generic description of nummular dermatitis to construct a clear, linear narrative that illustrates the intricate interplay between external triggers, internal dysregulation, and the eventual clinical findings observed in the patient. The schematic's power lies in its ability to visually connect the patient's lived experience—her environmental exposures and psychological state—to the complex cellular and neuro-immunological mechanisms that drive the disease process. It serves as an elegant model for understanding how a multifactorial condition develops and perpetuates itself. The cascade begins with the Initial Triggers, which the figure identifies as a dual insult unique to this patient's circumstances. The first trigger is Psychosocial Stress, explicitly linked to her work environment. This is a critical point, framing stress not merely as an abstract complicating factor but as a primary, potent biological trigger. The perception of chronic stress is the inciting event that activates a cascade of neuroendocrine responses, setting the stage for the profound influence of the psycho-neuro-cutaneous axis. Working in synergy with this internal trigger is the second insult: Environmental Factors. In this case, the chronic exposure to a low-humidity, air-conditioned environment led to xerosis (dry skin). This is not a superficial issue of comfort but a direct assault on the biophysical integrity of the stratum corneum, the skin's outermost protective barrier. Figure 6 posits that the simultaneous action of these two triggers created the perfect storm for the initiation of the disease. Following the initial triggers, the framework diverges into two core pathological pathways that were activated concurrently. The first is Barrier Dysfunction. This is described as an underlying "inside-out" defect where the skin's protective function is fundamentally compromised. Worsened by the environmental xerosis, a dysfunctional barrier has a reduced capacity to retain water, leading to further dryness, and, more importantly, it exhibits increased permeability. This "leaky" barrier allows external irritants, allergens, and microbes to penetrate deeper into the epidermis, where they can be recognized by the skin's immune cells, thereby lowering the threshold for an inflammatory response. The second core pathway is the Psycho-Neuro-Cutaneous Axis. The figure elegantly illustrates how the psychosocial stress trigger directly translates into a cutaneous inflammatory event. Stress signals originating in the central nervous system travel down peripheral sensory nerves that terminate in the skin. These nerve endings then act as local neuro-inflammatory command centers, releasing a powerful cocktail of neuropeptides, such as Substance P, directly into the dermal microenvironment. As the figure explains, these neuropeptides have profound and immediate effects: they are potent vasodilators, causing the capillaries to widen and leading to the clinical sign of erythema (redness); they increase vascular permeability, allowing inflammatory cells and fluid to leak into the tissue; and, crucially, they directly trigger mast cells to degranulate and release histamine and other potent pruritogens, which generate the intense and debilitating sensation of itch. The convergence of these two pathways creates the engine of chronicity depicted in the next stage of the figure: The Itch-Scratch Cycle. The intense, neurally-driven pruritus (itching) generated by the psycho-neuro-cutaneous axis leads to a near-uncontrollable urge to scratch. The act of scratching provides a fleeting moment of relief, but at a tremendous biological cost. The mechanical trauma of scratching physically abrades and further damages the already weakened epidermal barrier. This new injury incites an even greater inflammatory response as the body attempts to repair the damage. This additional inflammation, in turn, triggers the release of more itch mediators from both immune cells and keratinocytes, thus perpetuating and amplifying the vicious cycle. Figure 6 correctly identifies this cycle as the mechanism that drives the disease from an acute, transient flare into a chronic, self-sustaining condition. Finally, the entire cascade culminates in the Resulting Clinical Manifestations.

The figure logically concludes by showing how the sum of these underlying processes produces the visible signs observed on the patient's skin. The vasodilation and cellular inflammation driven by the neuro-immune response manifest as the characteristic erythematous plaques. Concurrently, the chronic scratching, coupled with the abnormal keratinocyte turnover of an inflamed epidermis, results in the visible scaling and excoriations.

The successful treatment of this patient was predicated on the diagnostic and pathophysiological insights discussed above. The therapeutic plan was not simply a list of medications but a multi-pronged strategy that targeted each identified component of her disease process. This represents a critical shift from a lesion-directed to a patient-centered paradigm. Mometasone furoate 0.1% cream was used to directly suppress the cutaneous inflammatory cascade. It acts to down-regulate the production of pro-inflammatory cytokines and inhibit the function of inflammatory cells like lymphocytes and mast cells, thereby reducing erythema and pruritus. The prescription of 10% urea cream was just as critical as the steroid. Urea is a powerful humectant that binds water within the stratum corneum, directly combating the patient's background xerosis.[20] It also has mild keratolytic properties, helping to reduce scale and improve the penetration of the active steroid. This component of the therapy was aimed at restoring the fundamental structural integrity of the epidermis. The oral antihistamine, cetirizine, was included for symptomatic control of the pruritus, particularly to aid sleep, which is often disrupted by nocturnal itching. This was arguably the most important part of the management. The extensive patient education and counseling about the role of stress and the validation of her own experience empowered her to understand the nature of her condition. Her decision to leave her stressful job was the most potent therapeutic intervention of all, as it shut down the primary trigger of the neurogenic inflammation that was driving the entire disease process. The rapid and significant clinical improvement, along with the dramatic drop in her DLQI score from 9 to 3, was not the result of any single cream but the synergistic effect of a holistic strategy that treated the patient, not just the lesion. This case argues that for chronic inflammatory conditions, the therapeutic goal should be to identify and address as many nodes in the complex pathogenic network as possible.

4. Conclusion

This case report, which details the journey of a patient from five years of diagnostic futility to a rapid and successful recovery, transcends being a simple account of a common disease. It serves as a powerful argument for a more thoughtful, integrated, and pathophysiology-driven approach to inflammatory skin disease. Based on the lessons learned from this case, we propose a modern diagnostic and management algorithm for chronic annular plaques, designed to prevent the pitfalls highlighted in this report: Step 1: Privileging the History. The initial focus of the clinical encounter should be on the narrative. A detailed history exploring chronicity, the temporal relationship to life stressors, a personal or family history of atopy, and the response to previous therapies must be prioritized over a premature judgment based on morphology alone. Step 2: Mandating In-Vivo Microscopy. Dermoscopy should not be considered an ancillary test but a mandatory extension of the physical examination for any ambiguous scaly plaque. The rapid, non-invasive identification of core patterns—such as the patchy dotted vessels of dermatitis versus the uniform dots of psoriasis or the peripheral scale of tinea—can immediately avert a diagnostic misstep. A simple KOH preparation remains essential to definitively rule out an infectious etiology. Step 3: Reserving Biopsy for True Ambiguity. A skin biopsy for histopathology is the gold standard but should be reserved for cases that remain diagnostically challenging after a thorough history and microscopic evaluation, or for those that are refractory to appropriately targeted therapy. Findings must always be interpreted in the full clinical context, including prior treatments. Step 4:

Formulating a Pathophysiology-Based Management Plan. The most effective treatment plans are those that target the specific drivers of the disease in that individual. This requires a shift from a one-size-fits-all approach to a personalized strategy that may include anti-inflammatory agents, barrier-restoring emollients, symptomatic relief, and, crucially, a direct acknowledgment and management plan for the identified psychosocial and neurogenic triggers. By adopting such an algorithm, clinicians can move beyond treating the visible lesion and begin treating the underlying cause, transforming the patient experience from one of chronic frustration to one of empowered and effective healing.

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