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The Crescendo Reaction in Patch Testing: A Key Diagnostic Sign in Allergic Contact Cheilitis from Modern Matte Lipsticks

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A B S T R A C T

Allergic contact cheilitis (ACC) from lipsticks is a frequent diagnostic challenge, often mimicking irritant contact cheilitis (ICC). The patch test is the diagnostic standard, but its accuracy relies heavily on the interpretation of reaction dynamics over time. This report illustrates the pivotal role of the crescendo reaction pattern in confirming an allergic etiology. A 21-year-old female presented with a six-month history of debilitating pruritus, papules, and subsequent xerosis with severe post-inflammatory hyperpigmentation on her lips. The symptoms were directly correlated with the daily use of several popular commercial matte and long-lasting lipsticks. Patch testing was performed with the European Standard Series and the patient's own cosmetic products. While standard allergens were negative, three specific lipsticks elicited a classic crescendo reaction: a weak positive (+) erythema at 48 hours that intensified to a strong positive (++) reaction with papules and palpable infiltration at the 72- and 96-hour readings. This dynamic confirmed a Type IV hypersensitivity reaction and a diagnosis of ACC. Management focused on strict allergen avoidance and barrier repair, resulting in complete resolution of symptoms and significant improvement in her quality of life. In conclusion, the crescendo pattern observed in patch testing is a compelling in vivo marker of an allergic, memory T-cell-driven immune response. Its presence provides conclusive evidence for ACC, reliably distinguishing it from the decrescendo pattern characteristic of irritation. Meticulous observation of the temporal evolution of patch test reactions is paramount for accurate diagnosis and effective patient management in cheilitis.

1. Introduction

Allergic contact dermatitis (ACD) represents a significant and growing global health concern, manifesting as a T-cell-mediated, delayed-type (Type IV) hypersensitivity reaction to an expanding universe of environmental haptens.[1] The rising prevalence of ACD is intrinsically linked to the increasing chemical complexity of modern consumer products, from industrial compounds to personal care items.[2] When this immunological reaction affects the lips, it is termed allergic contact cheilitis (ACC), a condition that presents a unique and formidable clinical and diagnostic challenge.

The lips, and particularly the vermilion zone, are anatomically predisposed to both irritation and sensitization.[3] This vulnerability stems from a distinct histology: an exceptionally thin stratum corneum, a high density of superficial vasculature, and a near-complete absence of sebaceous and sweat glands.[4] This delicate structure provides a less effective barrier against external chemicals and is prone to desiccation, further compromising its integrity and increasing the

penetration of potential allergens. Consequently, the lips are a frequent site for adverse reactions to topical products.

Among the primary etiologies of ACC, cosmetic products are the most frequently implicated, with lipsticks representing the single most common culprit, especially within the female population.[5] Modern lipstick formulations are no longer simple mixtures of wax and pigment; they are sophisticated chemical systems. A single lipstick can contain dozens of ingredients, including a complex base of waxes and oils, emollients, antioxidants, preservatives, fragrances, and a vast array of organic and inorganic dyes.[6] The recent consumer demand for "long-lasting," "transfer-proof," and intensely "matte" finishes has driven the incorporation of novel film-forming polymers, volatile solvents like isododecane, and silicone elastomers. While these ingredients enhance product performance, they may also increase the risk of both irritant and allergic reactions by altering skin barrier function or introducing new potential haptens.

Clinically, ACC is a chameleon, presenting with a spectrum of morphologies.[7] Acute ACC may manifest with dramatic erythema, edema, and vesiculation, often with weeping and crusting. More commonly, it follows a subacute or chronic course characterized by persistent dryness (xerosis), scaling, painful fissuring, and lichenification. A particularly distressing and common sequela, especially in individuals with higher Fitzpatrick skin types, is post-inflammatory hyperpigmentation (PIH). This persistent discoloration can linger for months or years after the inflammation has subsided, causing significant aesthetic concern and profound psychosocial impact.[8]

The cornerstone of diagnosis in suspected ACC is the epicutaneous patch test, which remains the gold standard for identifying causative allergen(s). However, the diagnostic power of patch testing extends beyond a simple binary positive or negative result at a single time point. Its true accuracy lies in a nuanced interpretation of both the reaction's morphology, such as erythema, papules, or vesicles, and, critically, its temporal dynamics. Allergic reactions, driven by an immunological memory response, characteristically follow a crescendo pattern. In this dynamic, the inflammatory reaction observed at the first reading (typically 48 hours) intensifies at subsequent readings (72 or 96 hours), even after the allergen-containing patch has been removed. This pattern is the clinical signature of a recruiting, amplifying, T-cell-mediated immune cascade. In stark contrast, irritant contact cheilitis (ICC), the primary differential diagnosis, typically produces a decrescendo pattern. An irritant reaction, caused by direct, non-immunological cytotoxic damage to keratinocytes, is often strongest at the initial reading and fades progressively once the offending agent is removed.[9]

Despite the established diagnostic importance of these reaction dynamics, their significance can be underappreciated in clinical practice. This can lead to diagnostic ambiguity, particularly when testing with patients' own products, which may possess both irritant and allergenic potential. A definitive diagnosis is imperative, as the management of ACC hinges on the precise identification and subsequent lifelong avoidance of the causative allergen—a strategy fundamentally different from the barrier-repair and trigger-reduction approach used for chronic irritation.[10]

This case report presents a classic and illustrative example of ACC induced by multiple commercial matte lipsticks in a young woman who developed chronic, pigmented cheilitis. The aim of this report is to provide a detailed clinical and methodological illustration of the crescendo reaction pattern paramount importance in patch testing. The value of this report lies not in presenting a new phenomenon, but in reinforcing an essential diagnostic principle through a detailed, modern case. We will demonstrate how meticulous observation of this dynamic immunological signature provides conclusive evidence for an allergic etiology in a challenging clinical presentation, thereby guiding definitive management and ensuring a positive patient outcome.

2. Case Presentation

A 21-year-old female university student of Southeast Asian descent (Fitzpatrick skin type IV) presented to our dermatology outpatient clinic with a chief complaint of "persistent itching, swelling, and severe darkening of my lips for the past six months." The patient reported that her symptoms began approximately six months prior to the consultation. The onset was insidious but coincided with her adoption of a new cosmetic routine involving daily use of several popular, commercially available matte and "long-lasting" liquid lipsticks. She named three specific products—anonymized as Brand B, Brand C, and Brand D-as her primary daily choices.

Initially, she experienced intermittent episodes of acute inflammation. Within 12 to 24 hours of lipstick application, she would develop intense pruritus, followed by mild edema and the eruption of small, erythematous papules confined to the vermilion of both lips. These symptoms were most pronounced with the aforementioned matte formulations. For the first three months, she attempted self-management with an over-the-counter hydrocortisone 1% cream, which provided partial and temporary relief from the itching and papules. However, the symptoms invariably recurred within 24 hours of reapplying any of the implicated lipsticks. She had established a clear and consistent correlation: her lips would "flare" after using the products and would slowly improve over 3-4 days if she abstained entirely from lip cosmetics, though they never returned to her baseline state.

Over the three months leading up to her presentation, the clinical picture evolved. The acute, episodic flares of papules and edema became less frequent as her lips entered a chronic inflammatory state. They became progressively and uniformly dry, scaly, and developed a prominent, diffuse, dark brown discoloration that she found extremely distressing. The pruritus transitioned from an intense, acute itch to a persistent, low-grade, background sensation (Table 1).

To quantify the impact on her well-being, the Dermatology Life Quality Index (DLQI) was administered. Her score was 16 out of a possible 30, indicating a "very large effect" on her quality of life. She elaborated that her score was driven by feelings of embarrassment and self-consciousness about her appearance (affecting questions on clothing choice, social activities, and intimate relationships), which led her to avoid social events and being photographed. The patient had no personal or family history of atopic diseases, including atopic dermatitis, asthma, or allergic rhinitis. She reported no known allergies to medications or foods and had never experienced a similar skin condition. Her past medical history was otherwise unremarkable, and she was not taking any systemic medications. She was a non-smoker and consumed alcohol socially on rare occasions.

On examination, the patient was comfortable, with vital signs within normal limits. The dermatological examination was focused on the perioral area (Figure 1). The findings were symmetrically distributed and strictly confined to the vermilion of the labialis superior and labialis inferior (upper and lower lips). There was a subtle but clear demarcation at the vermilion border, with no significant extension onto the surrounding cutaneous lip or involvement of the oral mucosa. The most striking feature was diffuse, ill-defined macular hyperpigmentation, deep brown in color, across the entirety of both lips. This was accompanied by moderate xerosis, manifesting as fine, adherent scaling and an accentuation of the natural lip lines, creating a wrinkled appearance (lichenification). No active erythema, papules, vesicles, or erosions were present at the time of the chronic-stage examination. Palpation revealed a slightly rough, dry, and minimally indurated texture. There were no signs of angular cheilitis or herpetic lesions.

Table 1. Summary of clinical findings on admission.

Clinical Findings on Admission

A comprehensive summary of the patient's presentation and initial diagnostic workup.

PARAMETER	FINDING
Patient Demographics & History	
Age & Gender	21-year-old female
Fitzpatrick Skin Type	Type IV (Southeast Asian descent)
Chief Complaint	"Persistent itching, swelling, and severe darkening of my lips."
Duration of Symptoms	6 months
Symptom Evolution	**Initial (First 3 months):** Intermittent pruritic papules and edema after lipstick use. **Chronic (Recent 3 months):** Persistent xerosis (dryness), scaling, and progressive hyperpigmentation.
Trigger	Clear correlation with daily use of new matte & long-lasting commercial lipsticks.
Past Medical History	No personal or family history of atopy (atopic dermatitis, asthma, allergic rhinitis). No known allergies.
Impact on Quality of Life	
DLQI Score	16 / 30 (Very large effect on quality of life)
Patient-Reported Impact	Feelings of embarrassment and self-consciousness leading to social avoidance.
Physical Examination Findings	
Location	Symmetrically confined to the vermilion of upper and lower lips.
Primary Morphology	**Pigmentation:** Severe, diffuse, deep brown macular hyperpigmentation. **Texture:** Moderate xerosis, fine scaling, and lichenification (accentuated lip lines). **Palpation:** Rough, dry, and minimally indurated texture.
Absent Signs (at presentation)	No active erythema, papules, vesicles, or erosions.
Initial Laboratory Workup	
Eosinophil Count	2% (Within normal limits: 1-4%)
Total Serum IgE	45 IU/mL (Within normal limits: <100 IU/mL)
Interpretation	No laboratory evidence of an underlying atopic diathesis.

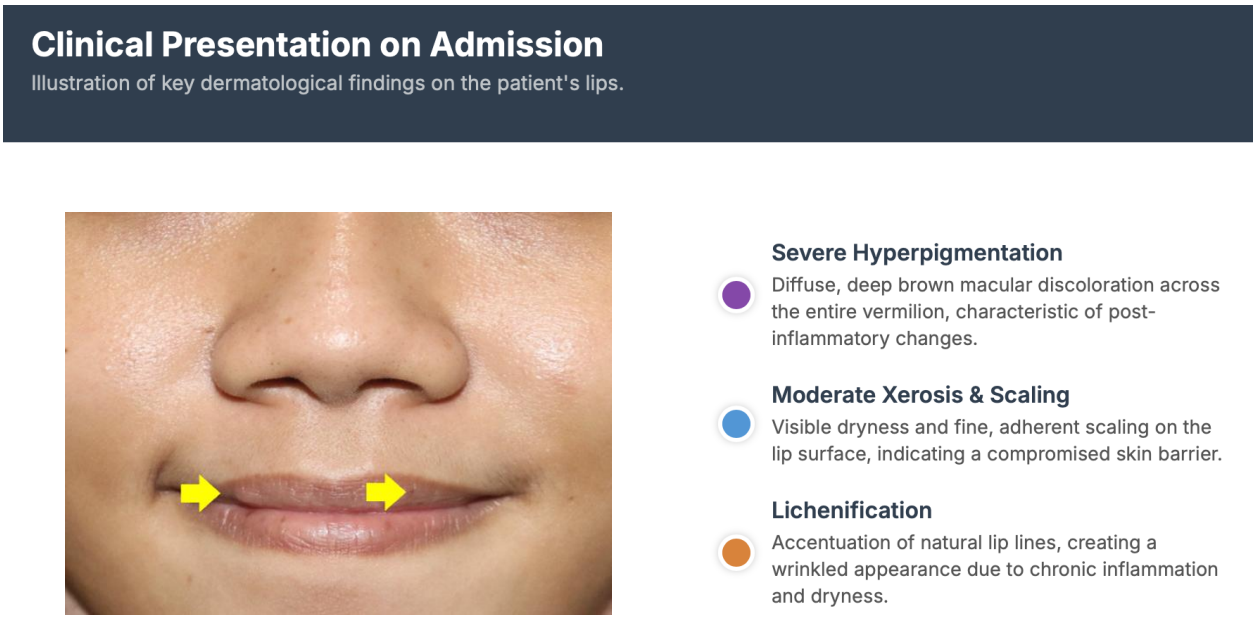


Figure 1. Clinical presentation.

Based on the compelling history of a cosmetic trigger and the chronic clinical findings, the primary differential diagnoses were: (1) Allergic Contact Cheilitis (ACC): Considered the leading diagnosis given the history of pruritic papules and a clear correlation with specific products, now in a chronic, pigmented phase; (2) Irritant Contact Cheilitis (ICC): A crucial differential, as long-lasting matte lipsticks contain potentially irritating solvents and film-formers. Clinically, chronic ICC can be indistinguishable from ACC; (3) Pigmented Contact Dermatitis: A variant of contact dermatitis where hyperpigmentation is the most prominent feature, often disproportionate to the degree of visible inflammation. This was considered a strong possibility; (4) Other forms of cheilitis: Actinic cheilitis was ruled out by the patient's age and the distribution of changes. Cheilitis exfoliativa was less likely given the clear external trigger.

To establish a definitive diagnosis, a comprehensive workup was initiated. To exclude underlying systemic or atopic predispositions, basic laboratory tests were performed; (1) Complete Blood Count (CBC) with Differential: Results were within normal limits. The eosinophil count was 2% (Normal range: 1-4%); (2) Total Serum Immunoglobulin E (IgE): The level was 45 IU/mL (Normal range: <100 IU/mL). The normal eosinophil and IgE levels made a significant underlying atopic diathesis, which can lower the threshold for irritation and allergy, less likely, further pointing towards a primary contact-driven process.

Comprehensive patch testing was performed to identify the specific causative agent(s). The European Standard Series was applied, along with the patient's personal products. The personal products included the three implicated lipsticks (Lipstick B, C, D in Table 2), two other lipsticks she used infrequently (Lipstick A, E), and her regular lip balm (Petrolatum Jelly A). The lipsticks were prepared by scraping the product and creating a 10% dilution in white petrolatum, a standard practice for testing finished cosmetic products to minimize irritancy while preserving antigenicity. The petrolatum jelly was tested "as is." The allergens were applied using Finn Chambers® on Scanpor® tape to the patient's upper back. The patient received detailed instructions to avoid all systemic

corticosteroids for four weeks, topical corticosteroids on her back for two weeks, and systemic antihistamines for 72 hours prior to testing. She was advised to keep the test area dry and avoid strenuous exercise for the duration of the test. The patches were removed after 48 hours (Day 2). Readings were performed by a trained dermatologist at 48 hours (30 minutes after patch removal), 72 hours (Day 3), and 96 hours (Day 4). Reactions were graded according to the International Contact Dermatitis Research Group (ICDRG) criteria (Figure 2). The detailed results are presented in Table 2. The European Standard Series was entirely negative, with the exception of two reactions of questionable relevance. A weak positive (+) reaction to Lanolin Alcohol (30%) and Hydroquinone (1%) was noted at the 48-hour reading. However, at the 72-hour reading, both reactions had faded significantly to doubtful (?+), and they were completely resolved by 96 hours. This decrescendo pattern is characteristic of a mild irritant reaction or is of questionable clinical relevance, especially given the patient's lack of known exposure to these specific allergens in products she used. The products labeled Lipstick A, Lipstick E, and Petrolatum Jelly A were consistently negative at all readings. The most significant and diagnostically conclusive findings were the reactions to Lipstick B, Lipstick C, and Lipstick D. At the 48-hour reading, all three sites showed a weak positive (+) reaction, characterized by well-demarcated faint erythema and minimal, barely palpable infiltration. At the 72-hour reading, these reactions had dramatically intensified. They were now graded as strong positive (++) reactions, presenting with marked erythema, easily palpable and well-defined infiltration, and scattered, discrete micropapules within the patch area. At the 96-hour reading, the strong positive (++) reactions persisted with no sign of fading. The erythema remained vibrant, and the infiltration and papulation were still prominent. This dynamic intensification from a weak (+) reaction at 48 hours to a strong (++) reaction at 72 and 96 hours is the classic crescendo pattern, providing compelling evidence of a Type IV delayed-type hypersensitivity reaction.

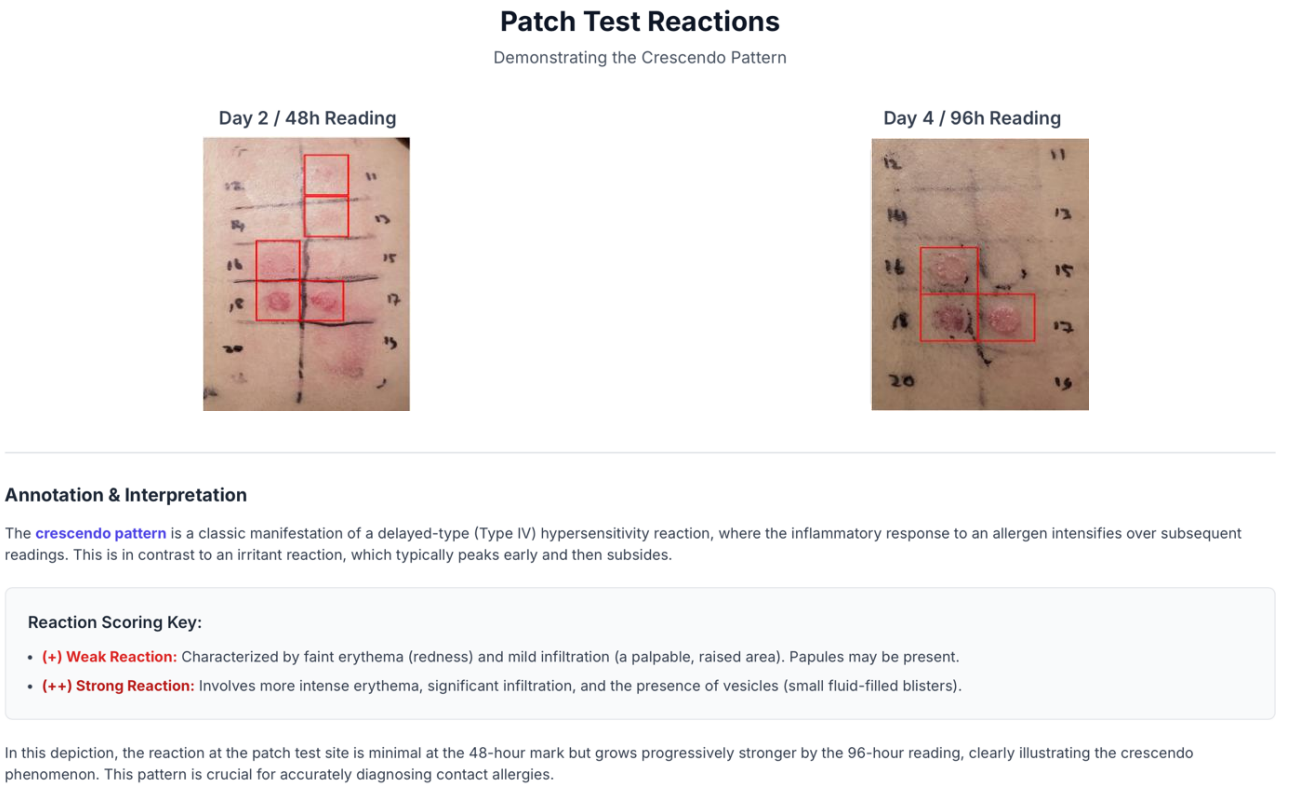


Figure 2. Patch test reactions demonstrating the crescendo pattern.

Table 2. Patch Test Results

Comprehensive Allergen Panel with ICDRG Scoring

NO.	ALLERGEN NAME	CONC.	VEHICLE	48 HOURS (D2)	72 HOURS (D3)	96 HOURS (D4)
European Standard Series						
1	Mercaptobenzothiazole	2%	Pet.	-	-	-
2	Cobalt chloride	1%	Pet.	-	-	-
3	Para-phenyldiamine	0.1%	Pet.	-	-	-
4	Balsam Peru	25%	Pet.	-	-	-
5	Benzocaine	5%	Pet.	-	-	-
6	Potassium Dichromate	0.5%	Pet.	-	-	-
7	Ethylenediamine	1%	Pet.	-	-	-
8	Benzophenone	3%	Pet.	-	-	-
9	Colophony	20%	Pet.	-	-	-
10	S-chloro-7-iodo-8-hydroxy quinoline	5%	Pet.	-	-	-
11	Lanolin	30%	Pet.	+	?+	-
12	Nickel sulfate	5%	Pet.	-	-	-
13	Hydroquinone	1%	Pet.	+	?+	-
14	Quarterium	1%	Pet.	-	-	-
Patient's Own Products						
15	Lipstick A	10%	Pet.	-	-	-
16	Lipstick B	10%	Pet.	+	++	++
17	Lipstick C	10%	Pet.	+	++	++
18	Lipstick D	10%	Pet.	+	++	++
19	Lipstick E	10%	Pet.	-	-	-
20	Petrolatum Jelly A	As is	-	-	-	-

Annotation & Interpretation

ICDRG Scoring Key

- `-` : Negative reaction
- `?+` : Doubtful (faint macular erythema)
- `+` : Weak positive (erythema, infiltration)
- `++` : Strong positive (papules, vesicles)

Key Observations

- **Crescendo Pattern:** Lipsticks B, C, and D show a classic allergic response, intensifying from a weak (+) to a strong (++) reaction.
- **Decrescendo Pattern:** Lanolin and Hydroquinone show reactions that fade over time, suggesting minor irritation or questionable relevance.
- **Negative Controls:** The patient's other products were negative, confirming the specificity of the reaction to the three implicated lipsticks.

Based on the compelling history, clinical morphology, and the conclusive crescendo pattern on patch testing, a final diagnosis was established: Allergic Contact Cheilitis caused by one or more shared ingredients in Lipstick B, Lipstick C, and Lipstick D, with severe secondary post-inflammatory hyperpigmentation. The management plan was multifactorial: (1) Allergen Avoidance: This was the cornerstone of therapy. The patient received extensive counseling to immediately and permanently cease using the three implicated lipsticks. She was provided with a detailed handout on how to read cosmetic ingredient labels (INCI lists) and counseled that, since the specific hapten was not identified, she should be cautious with all new colored lip products. (2) Pharmacotherapy: For acute inflammation control: A short course of a mid-potency topical corticosteroid, Desonide 0.05% ointment, was prescribed for twice-daily application for a maximum of 7-10 days, to be used only for any potential flares; For daily maintenance and barrier repair: She was advised to use a simple, inert emollient like pure petrolatum jelly multiple times daily to restore the integrity of the lip barrier; (3) Management of PIH: She was educated on the importance of strict sun protection to prevent further darkening. Daily use of a broad-spectrum SPF 30+ lip balm was mandated; (4) Follow-up: The patient was scheduled for follow-up appointments at 4 weeks and 3 months.

At the 4-week follow-up, the patient reported complete adherence to the avoidance strategy and was using the petrolatum jelly regularly (Table 3). The pruritus, scaling, and xerosis had entirely resolved. She had not needed to use the topical corticosteroid. At the 3-month follow-up, she remained completely symptom-free of active cheilitis. The post-inflammatory hyperpigmentation had visibly lightened by an estimated 50%. Most importantly, her DLQI score had improved dramatically from 16 to 3 ("small effect on patient's life"), reflecting a profound improvement in her confidence and social functioning.

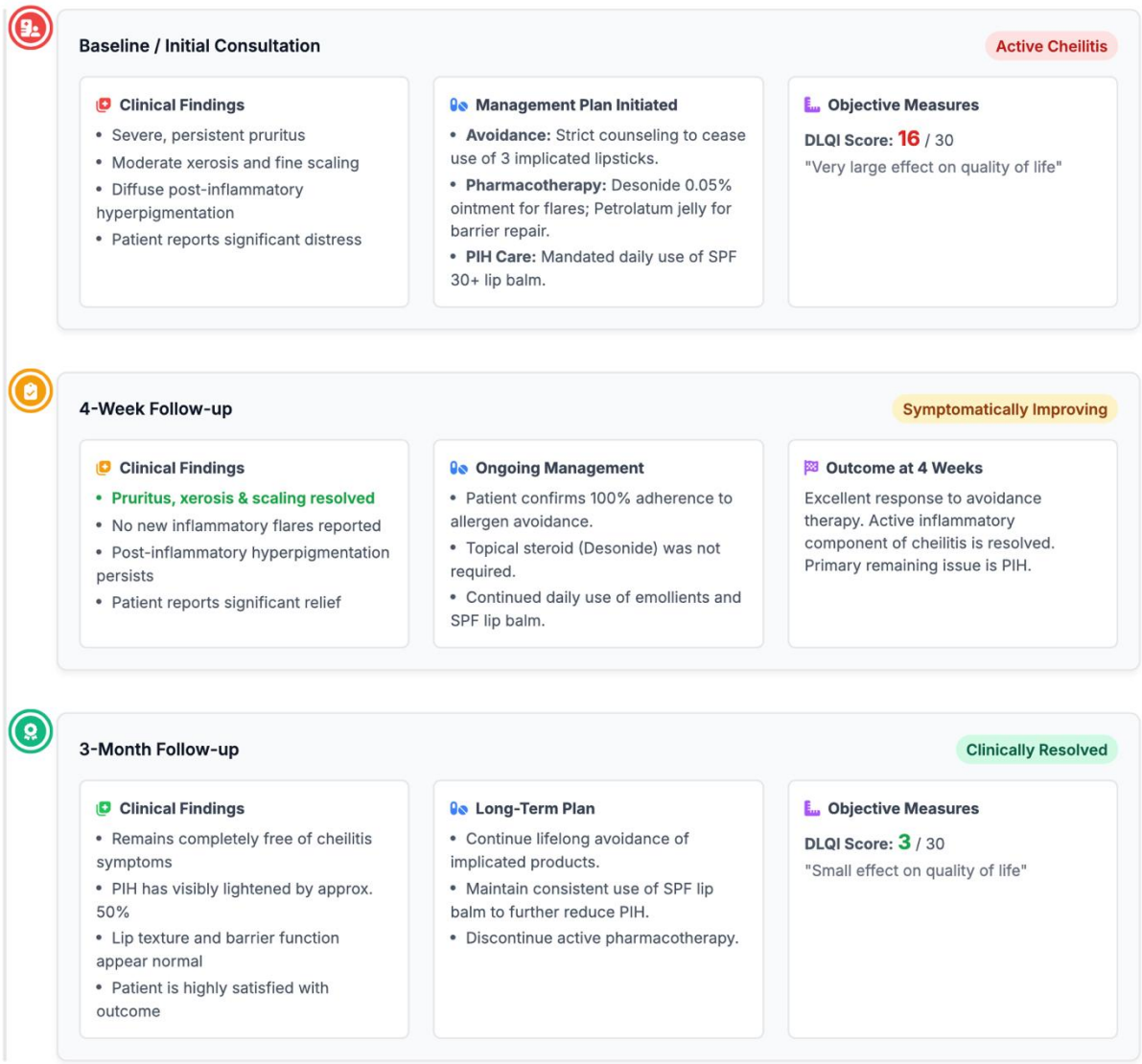
3. Discussion

This case report provides a detailed illustration of allergic contact cheilitis (ACC), where the diagnostic journey culminates in the unequivocal interpretation of a crescendo reaction pattern on patch testing.[11] The following discussion will explore the intricate pathophysiology of ACC, the immunological basis of the crescendo phenomenon as a conclusive diagnostic marker, a speculative analysis of likely culprits in modern lipsticks, and the mechanisms driving the development of PIH. ACC is a canonical example of a Type IV, or delayed-type, hypersensitivity reaction. This complex immunological process is orchestrated not by antibodies, but by antigen-specific T-lymphocytes, and unfolds in two distinct phases (Figure 3).

The Sensitization Phase (Induction): This initial phase is clinically silent and begins when a low-molecular-weight chemical, known as a hapten, from a product like lipstick penetrates the thin stratum corneum of the lips. Potential haptens in lipsticks are numerous and include fragrance molecules, preservatives, antioxidants, or even certain dyes. These haptens are too small to be immunogenic on their own. To trigger an immune response, they must first bind covalently to endogenous carrier proteins within the epidermis, forming a stable hapten-protein complex.[12] This process of "haptenation" renders the self-protein immunologically "foreign". These neoantigens are then recognized and internalized by local antigen-presenting cells (APCs), primarily the Langerhans cells that form a dense network in the epidermis.[13] Upon capturing the antigen, Langerhans cells undergo maturation, downregulate their epidermal adhesion molecules, and migrate from the epidermis via afferent lymphatics to the regional draining lymph nodes, including the submandibular and submental nodes. Within the paracortex of the lymph node, they process the complex and present the haptenic epitope, nestled within the groove of Major Histocompatibility Complex (MHC) molecules (Class II for exogenous antigens), to naive CD4+ T-helper (Th) lymphocytes.

Table 3. Treatment Plan and Follow-up Timeline

A Chronological Overview of Patient Management and Outcomes



This interaction, along with co-stimulatory signals, induces the clonal expansion and differentiation of these T-cells into allergen-specific memory Th1 and Th17 cells. This entire sensitization process typically takes 10-14 days, after which the individual is considered sensitized for life.[14] The Elicitation Phase (Challenge): Once sensitization has occurred, any subsequent exposure of the lips to the same hapten will trigger the elicitation phase, leading to the clinical manifestations of ACC. Upon re-exposure, the hapten again forms complexes with epidermal proteins. This time, however, they are recognized directly in the tissue by the now-abundant, circulating hapten-specific memory T-cells that patrol the skin and mucosal surfaces. This recognition leads to rapid T-cell activation within the lip tissue itself, typically within 24-48 hours of exposure. Activated T-cells unleash a potent cocktail of pro-inflammatory cytokines and chemokines.[15] Th1 cells release interferon-gamma (IFN-γ), which activates keratinocytes and macrophages, and tumor necrosis factor-alpha (TNF-α), which promotes inflammation.

Th17 cells produce interleukin-17 (IL-17) and IL-22, which recruit neutrophils and amplify the inflammatory response. This cytokine cascade orchestrates the recruitment of a secondary, non-specific inflammatory infiltrate of monocytes, macrophages, and other lymphocytes to the site of exposure.[16] This cellular influx and the effects of the cytokines on the local vasculature cause the characteristic clinical signs of acute ACC: erythema (vasodilation), edema (increased vascular permeability), and papulovesicles (spongiosis and cellular infiltrate).

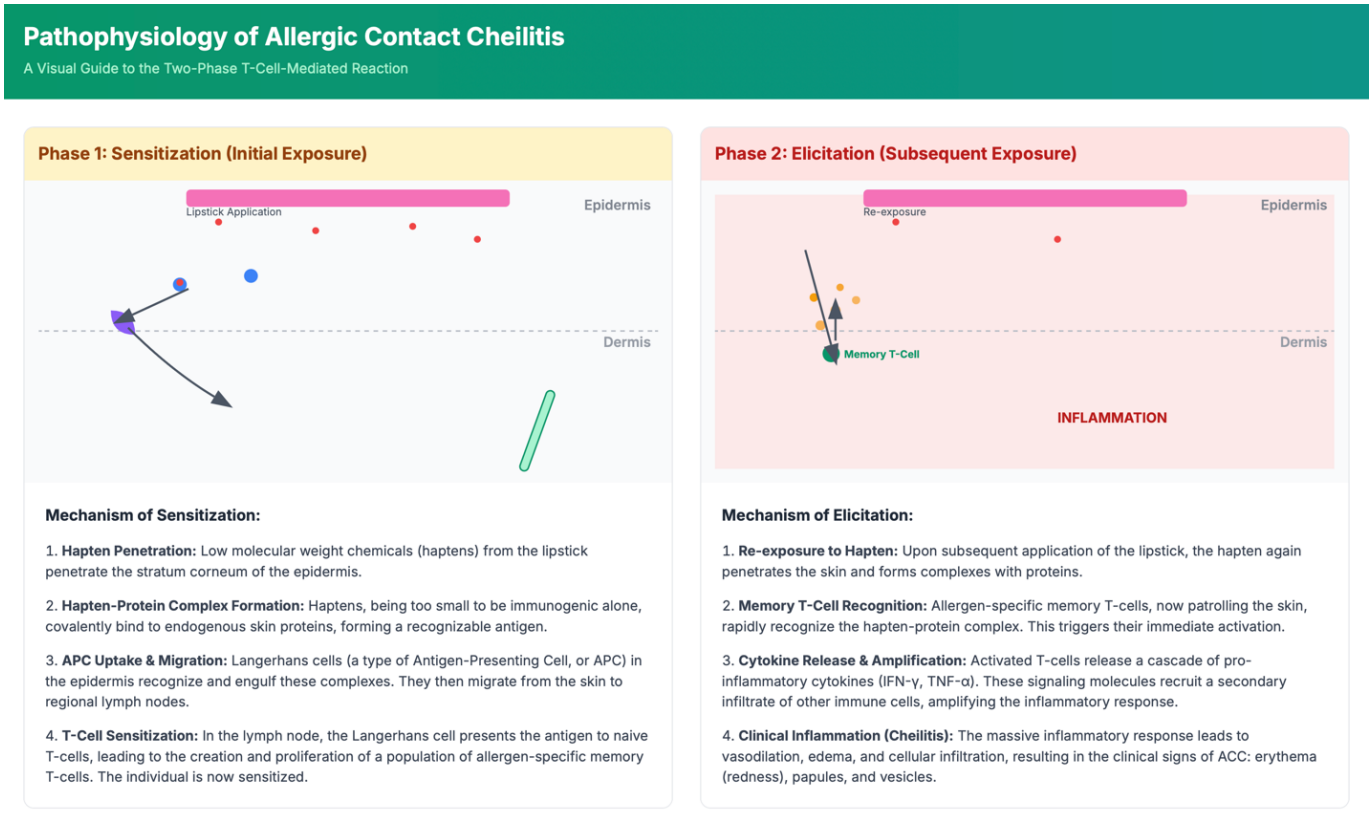


Figure 3. Pathophysiology of allergic contact cheilitis.

The most compelling aspect of this case is the clear demonstration of the crescendo reaction, which serves as a direct clinical window into the pathophysiology of the elicitation phase.[17] Its dynamics are what definitively separate a true memory-driven allergic response from a direct toxic-irritant effect. When an allergen is applied under a patch, the elicitation phase is initiated. By the 48-hour reading, the initial wave of memory T-cell recognition and activation has occurred. The first volley of cytokine release has begun, recruiting an early inflammatory infiltrate and causing vasodilation. Clinically, this manifests as the initial reaction—in this case, a weak positive (+) response of erythema and minimal infiltration.

The key insight is why the reaction intensifies after the patch is removed. This is due to the self-amplifying and sustained nature of the T-cell-mediated immune cascade. A sufficient amount of hapten has already penetrated the skin and bound to epidermal proteins during the 48-hour application, creating a local depot of antigen that continues to stimulate T-cells for several days.[18] The initial cytokine release, including TNF-α and chemokines like CXCL8, acts as a powerful beacon, actively recruiting more inflammatory cells—

monocytes, macrophages, and additional T-cells—from the circulation into the patch test site. This secondary wave of cellular infiltration peaks between 48 and 96 hours, significantly amplifying the local inflammation and leading to more pronounced clinical signs like intense erythema, palpable infiltration (edema and cellular infiltrate), and papule formation. This escalating inflammatory response, driven by a positive feedback loop of continued antigen presentation and cellular recruitment, is the immunological basis of the crescendo pattern.

This stands in stark contrast to an irritant reaction. An irritant causes direct, non-immunological damage to keratinocytes, leading to the release of pre-formed inflammatory mediators like IL-1α. The inflammation is maximal while the irritant is present and begins to resolve through innate repair mechanisms as soon as the irritant is removed.[19] This results in the characteristic decrescendo pattern, as was observed with the Lanolin and Hydroquinone reactions in our patient, thus highlighting the diagnostic power of comparing reaction dynamics on the same patient.

A limitation of this case is that while the vehicles (the lipsticks) were identified, subsequent patch testing with their individual components was not performed. Therefore, the identification of the specific causative hapten remains speculative. However, the fact that three different matte lipstick products from different brands elicited a positive reaction strongly suggests they share a common, cross-reacting allergenic ingredient. An analysis of common ingredients in such formulations reveals several plausible culprits. (1) Tocopherol (Vitamin E) and its Esters (Tocopheryl Acetate): Widely used as antioxidants to prevent rancidity of oils in cosmetics, tocopherol is a well-documented, albeit uncommon, contact allergen. The oxidation of tocopherol itself can form allergenic derivatives, and its presence in all three implicated products makes it a prime suspect; (2) Fragrance Ingredients: Fragrances are among the most common causes of cosmetic allergy worldwide. While a lipstick may be marketed as "unscented," it often contains aromatic essential oils or synthetic chemicals to mask the base odor of waxes and oils. Common sensitizers, such as cinnamal, geraniol, or hydroxycitronellal, could be shared across the product lines; (3) Phenoxyethanol: This is a globally ubiquitous preservative that has largely replaced parabens in many cosmetic formulations. While generally considered to have a low sensitization potential, reports of allergic contact dermatitis to phenoxyethanol are increasing as its use becomes more widespread. Its presence in all three reactive products warrants suspicion; (4) Dyes and Pigments: While classic dye allergens like D&C Red 27 are well-known, other organic pigments used to achieve vibrant matte shades could also be responsible.

A significant component of the patient's clinical presentation and distress was the chronic, severe hyperpigmentation. PIH is a common consequence of any inflammatory process in the skin, particularly in individuals with skin of color.[20] The link between the Type IV reaction of ACC and melanogenesis is multifactorial. The pro-inflammatory cytokines released during ACC, such as TNF-α and interleukins, along with other mediators like prostaglandins and leukotrienes, directly stimulate melanocytes, upregulating the activity of tyrosinase, the rate-limiting enzyme in melanin synthesis. This leads to increased production of melanin (melanogenesis) and enhanced transfer of melanosomes to surrounding keratinocytes, resulting in epidermal hyperpigmentation.

More significantly, the intense inflammation can damage the dermo-epidermal junction and disrupt the integrity of the basal keratinocyte layer. This damage allows melanin pigment to "drop" from the epidermis into the papillary dermis, where it is engulfed by macrophages (termed melanophages). This process is known as pigmentary incontinence and results in dermal hyperpigmentation. Dermal pigment is cleared very slowly by the lymphatic system, which explains the deep brown color and the persistence of PIH for many months or even years, long after the active cheilitis has resolved. The thinness of the lip's

epithelium may make it particularly susceptible to this process.

This case underscores the necessity of a meticulous approach to patch testing. Simply noting a positive reaction at 48 hours is insufficient. The readings at 72 or 96 hours are not optional; they are essential for observing the reaction dynamics that differentiate allergy from irritation. Furthermore, while identifying the patient's own product is the crucial first step for management via avoidance, the ideal diagnostic conclusion involves identifying the specific hapten. This allows for broader and more precise avoidance counseling, empowering the patient to screen all future products. In a clinical setting where this is not feasible, resources like online cosmetic allergen databases can be invaluable tools for patients to find products free from common sensitizers suspected in their case.

4. Conclusion

This case of lipstick-induced allergic contact cheilitis serves as a powerful clinical reminder of the paramount importance of the crescendo reaction pattern in patch testing. This dynamic finding is a highly reliable in vivo marker of a Type IV hypersensitivity reaction, providing conclusive evidence that distinguishes allergy from irritation. In the evaluation of chronic cheilitis, where the clinical picture can be ambiguous, clinicians must meticulously observe the temporal evolution of patch test reactions. This detailed interpretation is the cornerstone of an accurate diagnosis, which in turn enables the most effective management strategy: targeted allergen avoidance, leading to symptom resolution and a restored quality of life.

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