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Beyond Appearance: The Positive Mental Health Impact of Aesthetic Medicine

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INTRODUCTION

As doctors, we enter medicine with a shared purpose – to diagnose and treat diseases, alleviate suffering, restore function, do good and, where possible, save lives. The disciplines traditionally regarded as the pillars of medicine – emergency medicine, surgery, oncology, cardiology, intensive care – carry an obvious gravity because the consequences of disease are immediate and often life-threatening. It is therefore unsurprising that aesthetic medicine has, at times, found itself questioned within the wider medical community.

The public face of aesthetic medicine through popular media is often one of cosmetic enhancement and elective beautification, creating the impression that aesthetic medicine exists primarily to enhance beauty rather than to treat disease. Yet this portrayal reflects only part of what many of us do every day. In this discipline, where we manage conditions such as melasma, acne, acne scarring, hyperpigmentation, and other appearance-related disorders, our work actually extends well beyond cosmetic enhancement. We care for patients with chronic conditions that often carry a substantial yet unseen psychological burden, contributing to anxiety, depression, reduced self-esteem, impaired social functioning, strained interpersonal relationships, and a diminished quality of life.

The World Health Organization defines health as “a state of complete physical, mental and social well-being and not merely the absence of disease” [1]. Viewed through that lens, conditions that profoundly affect psychological well-being deserve the same thoughtful, evidence-based medical care that allows us to do good for our patients.

Doing Good Through Aesthetic Medicine

One question I have often reflected upon is this: if aesthetic medicine is not saving lives, how is that we, as doctors, are doing good? I believe the answer lies in recognizing that the value of medical care cannot be measured solely by mortality or disease cure. Across medicine, improving quality of life is an equally important outcome. This is particularly relevant given the growing body of evidence demonstrating that mental and physical

health are intrinsically linked, each profoundly influencing the other.

A recent systematic review of 113 studies found that more than one in four patients with skin diseases experienced depression (27.2%), and anxiety affected 36.5% of patients with acne, demonstrating the considerable psychological burden of visible skin conditions [2]. Many of the conditions we treat may not be life-threatening, but they can profoundly affect mental health, social functioning, confidence and how individuals participate in everyday life. It is these less visible consequences that we often have the privilege of treating.

Pigmentation

Take melasma, for example. Although not life-threatening, it is a chronic pigmentary disorder that affects an estimated up to 33% of the global population [3]. Numerous studies using validated instruments such as the Melasma Quality of Life Scale (MELASQoL) have consistently demonstrated significant reductions in emotional well-being, self-esteem, and social functioning [4]. Treating chronic pigmentary disorders such as melasma is not simply about lightening pigmentation, but about managing a complex chronic disease while restoring confidence and improving quality of life.

Acne and Acne Scarring

The same is true of acne. Acne vulgaris affects approximately 85% of adolescents and young adults worldwide and has consistently been associated with anxiety, depression, reduced self-esteem, and social isolation [5]. For many patients, the burden continues long after acne has resolved. Permanent acne scarring affects approximately 20-30% of individuals with moderate-to-severe acne and may influence confidence, relationships, and daily interactions for years [6]. Numerous studies also demonstrate that acne scars independently reduce quality of life and are associated with lower self-confidence and greater emotional distress than active acne alone [7].

When we treat acne and acne scarring, we are not simply improving skin appearance. We are treating

conditions that have a measurable psychological impact, allowing patients to regain confidence and participate more fully in their personal and professional lives. The visible improvement is often what others notice, but the true success of treatment is measured by happiness, confidence regained, psychological distress alleviated, and quality of life restored. Studies have consistently shown that a more positive perception of one's appearance is associated with lower rates of depression and anxiety, higher self-esteem, and better overall psychological well-being [8]. For many patients, restoring confidence means returning to work with greater assurance, engaging more comfortably in relationships, or simply feeling at ease in their own skin. That, too, is practicing good medicine.

CONCLUSION

Throughout my years in aesthetic practice, I have come to appreciate that the impact of our work extends far beyond what is visible on the skin. While many of the conditions we treat are not immediately life-threatening, the psychological burden they impose on our patients can be profound and should not be underestimated.

As doctors, our responsibility is not only to treat disease, but also to improve the lives of those entrusted to our care. Just as treating depression is measured not only by symptom relief but by restored quality of life, relieving the psychological burden of chronic skin conditions can be equally transformative for our patients.

In doing so, aesthetic medicine fulfils one of the fundamental goals shared across all medical disciplines: to meaningfully improve the lives of our patients and to truly do good by them.

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Microneedling versus Modified Jessner's Solution versus Combination Therapy for Atrophic Acne Scars in Fitzpatrick Skin Type III-V: A Randomised Control Study

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ABSTRACT: Atrophic acne scars are a common sequela of acne vulgaris and can impose a substantial cosmetic and psychosocial burden. Several treatment modalities are available, including chemical peels and microneedling; however, no single gold-standard treatment has been established. Combination approaches have been proposed to enhance treatment outcomes by targeting multiple mechanisms involved in dermal remodelling. This study aimed to compare the efficacy and safety of microneedling alone, Modified Jessner's Solution alone, and their combination in the treatment of atrophic acne scars among patients with Fitzpatrick skin types III-V. This randomised controlled study included Filipino patients aged 18–45 years with clinically diagnosed atrophic acne scars assessed using the Global Acne Scarring Grading System (GASGS). Participants were randomly assigned to three treatment groups: chemical peeling (CP), microneedling (MN), or combination therapy (MC). Treatments were administered at Weeks 0, 4, and 8, with follow-up at Week 12. Outcomes were assessed using the GASGS based on standardised clinical photographs evaluated by blinded dermatologists, as well as the Facial Acne Scar Quality of Life (FASQoL) questionnaire. Fifty-five participants completed the study: 18 in the CP group, 17 in the MN group, and 20 in the MC group. Median quantitative GASGS scores decreased progressively across all treatment groups over the 12-week study period, indicating improvement in acne scarring from baseline. The CP group demonstrated a less pronounced reduction by Week 12 compared with the MN and MC groups, although no intervention demonstrated consistent superiority. All procedures were well tolerated, with mild and transient adverse events reported. Quality-of-life scores improved across all treatment groups. Microneedling, Modified Jessner's Solution, and their combination were well tolerated and showed potential for improving atrophic acne scars among patients with Fitzpatrick skin types III-V. Combination therapy may offer additional benefits over either modality alone.

Keywords: Microneedling, Jessner, Acne scars, GASGS

INTRODUCTION

Acne vulgaris is a common inflammatory disorder of the pilosebaceous unit characterised by papules, pustules, and nodules that primarily affect the face but may also involve the trunk and upper arms [1]. It affects individuals across all age groups, with prevalence rates ranging from 42% to 74% among adolescents and young adults [2]. Acne may result in long-term sequelae, including scarring, which can cause substantial cosmetic and psychological distress. Acne scars occur in up to 95% of patients and are often associated with delayed treatment or severe inflammatory lesions [3]. The pathophysiology of acne scarring involves inflammation of the pilosebaceous unit extending into the dermis following

rupture of the follicular wall. This process leads to tissue destruction, aberrant wound healing, dermal collagen loss, and contraction of the surrounding tissue, ultimately resulting in skin indentation and the formation of atrophic scars [4]. In the Philippines, acne scarring remains a prevalent dermatological concern, with 41–60% of acne patients presenting with scarring at their initial consultation [4]. In a survey of 269 Filipino dermatologists, Handog et al. reported that scarring was most frequently observed among patients aged 26–35 years, with 78% having Fitzpatrick skin type IV. The most commonly reported scar types were hyperpigmented scars (84%), depressed scars (80%), atrophic scars (67%), hypertrophic scars (55%), and hypopigmented scars (29%) [4].

The management of acne scars remains challenging. Available treatment modalities include chemical peels, dermabrasion, microneedling, subcision, punch techniques, injectable fillers, and energy-based devices [5]. Most dermatologists report using a combination of topical therapies and procedural interventions, including chemical peels, microdermabrasion, microneedling, and energy-based devices [4]. However, no single treatment modality has been established as the gold standard.

Chemical peels are widely used in acne scar management and act by inducing controlled epidermal injury that promotes dermal remodeling and regeneration of new epidermal and dermal tissue [6,7]. Jessner's solution is a commonly used chemical peel composed of resorcinol, salicylic acid, lactic acid, and ethanol [8]. However, resorcinol has been associated with contact dermatitis and post-inflammatory hyperpigmentation, particularly in patients with darker skin types [9]. Modified Jessner's Solution replaces resorcinol with citric acid and contains higher concentrations of salicylic and lactic acids, potentially improving tolerability and reducing adverse effects [10].

Microneedling is another established modality that creates controlled micro-injuries within the epidermis and papillary dermis, stimulating collagen production through activation of growth factors and fibroblast proliferation [11,12]. The microchannels created during the procedure may also enhance transdermal drug delivery [11]. Microneedling is generally well tolerated, with adverse effects such as erythema, oedema, and pain typically resolving spontaneously [12,13].

Combination therapies may enhance treatment outcomes by targeting multiple mechanisms involved in scar remodelling [14]. Several studies have reported greater improvement in atrophic acne scars when microneedling is combined with chemical peeling compared with either treatment alone [11,15-17].

Despite the promising results reported with combination therapies, few studies have evaluated the combined use of microneedling and Modified Jessner's Solution. Most existing studies have investigated traditional Jessner's solution or other chemical peeling agents, while data among individuals with darker skin phototypes remain limited. Furthermore, few randomised controlled trials have directly compared microneedling, chemical peeling, and combination therapy within the same study population. To our knowledge, no randomised controlled trial has evaluated these treatment modalities using Modified Jessner's Solution among Filipino patients with Fitzpatrick skin types III-V. Therefore, this study aimed to compare the efficacy and safety of microneedling alone, Modified Jessner's Solution alone, and their combination in the treatment of atrophic acne scars, as assessed using clinical photography and the Goodman and Baron Global Acne Scarring Grading System (GASGS).

METHODOLOGY

Study Design and Participants

This randomized controlled trial with blinded outcome assessment was conducted between September and December 2024. The participants were recruited from patient attend outpatient dermatology clinic of the Research Institute for Tropical Medicine. The study protocol was reviewed and approved by the Institutional Ethics Review Board of the Research Institute for Tropical Medicine (Reference No.: 2024-02). Written informed consent was obtained from all participants prior to enrolment.

Participants aged 18–45 years with clinically diagnosed atrophic acne scars and Fitzpatrick skin types III–V were eligible for inclusion. Atrophic acne scars were classified using the Qualitative GASGS, and participants with mild, moderate, and severe acne scars were included in the study. Exclusion criteria included previous acne scar treatment within the preceding four months, isotretinoin therapy within the past six months, active skin infection, bleeding disorders, pregnancy, a history of keloid formation, collagen vascular disease, uncontrolled diabetes mellitus, and known hypersensitivity to Modified Jessner's Solution.

Sample Size and Randomization

Sample size estimation was based on a previous randomized study evaluating microneedling and Jessner's solution for the treatment of acne scars [8]. Assuming improvement rates of 80% with combination therapy, 30% with microneedling alone, and 25% with chemical peel alone, a minimum sample size of 18 participants per treatment arm was calculated using STATA version 17.0, with a statistical power of 80%, a significance level of 0.05, and a 20% allowance for potential loss to follow-up. Participants were randomized using block randomization stratified by acne scar severity and allocated to three treatment groups: chemical peel (CP), microneedling (MN), and combination therapy (MC).

Treatment Protocol

Participants underwent three treatment sessions at Weeks 0, 4, and 8, with the final evaluation performed at Week 12. In the microneedling group, a topical anaesthetic containing 2.5% lidocaine and 2.5% prilocaine was applied for one hour prior to the procedure. Microneedling was performed using a motorized microneedling device (M.pen Pro, Mesoestetic Pharma Group, Spain) at a needle depth of 1.5 mm, with vertical, horizontal, and diagonal passes until uniform pinpoint bleeding was achieved.

In the chemical peel group, Modified Jessner's Solution (14% salicylic acid, 14% lactic acid, and 8% citric acid) was applied evenly using a brush in three coats over the treatment area, while avoiding the periorbital and

perioral regions. Participants in the combination therapy group underwent microneedling followed immediately by application of Modified Jessner's Solution according to the same protocol. Following treatment, participants in the MN and MC groups were instructed to apply mupirocin ointment twice daily for five days. All Participants were advised to use a gentle cleanser and strict photoprotection with SPF 50 sunscreen throughout the study period.

Outcome Measures

Standardized facial photographs were obtained at baseline and at Weeks 4, 8, and 12 using the Quantificare LifeViz device (QuantifiCare, France) system under consistent lighting conditions, camera settings, and patient positioning. Photographs included frontal and bilateral lateral views. The primary outcome was the improvement in acne scar severity measured using the GASGS, which incorporates both quantitative and qualitative assessments. Acne scar severity was independently evaluated by two dermatologists blinded to treatment allocation.

For the quantitative GASGS assessment, individual acne scar lesions were counted, and numerical scores were assigned according to their number, type, and severity using a standardised grading system [3,18]. For the qualitative GASGS assessment [3,18], improvement was defined as a decrease of at least one severity grade from baseline to Week 12.

Hyperpigmented and hypopigmented lesions were also documented as part of the GASGS assessment.

Secondary outcomes included patient-reported quality of life assessed using the Facial Acne Scar Quality of Life (FASQoL) questionnaire and the incidence of treatment-related adverse events, including erythema, bleeding, and burning sensation.

Statistical Analysis

Statistical analyses were performed using Stata version 18 (StataCorp LLC, College Station, TX, USA). Descriptive statistics were used to summarize participant demographic and clinical characteristics. Continuous variables were presented as mean $\pm$ standard deviation, while categorical variables were summarized as frequencies and percentages. Baseline characteristics were compared among the three treatment groups using one-way analysis of variance (ANOVA) or the Kruskal-Wallis test for continuous variables, depending on data distribution, and the chi-square test or Fisher's exact test for categorical variables.

GASGS scores were summarized as medians and interquartile ranges (IQRs). Differences in GASGS scores among the three treatment groups at each time point were assessed using the Kruskal-Wallis test. Changes in GASGS scores over time within each treatment group were evaluated using the Friedman test for repeated measures. Statistical significance was defined as $p < 0.05$.

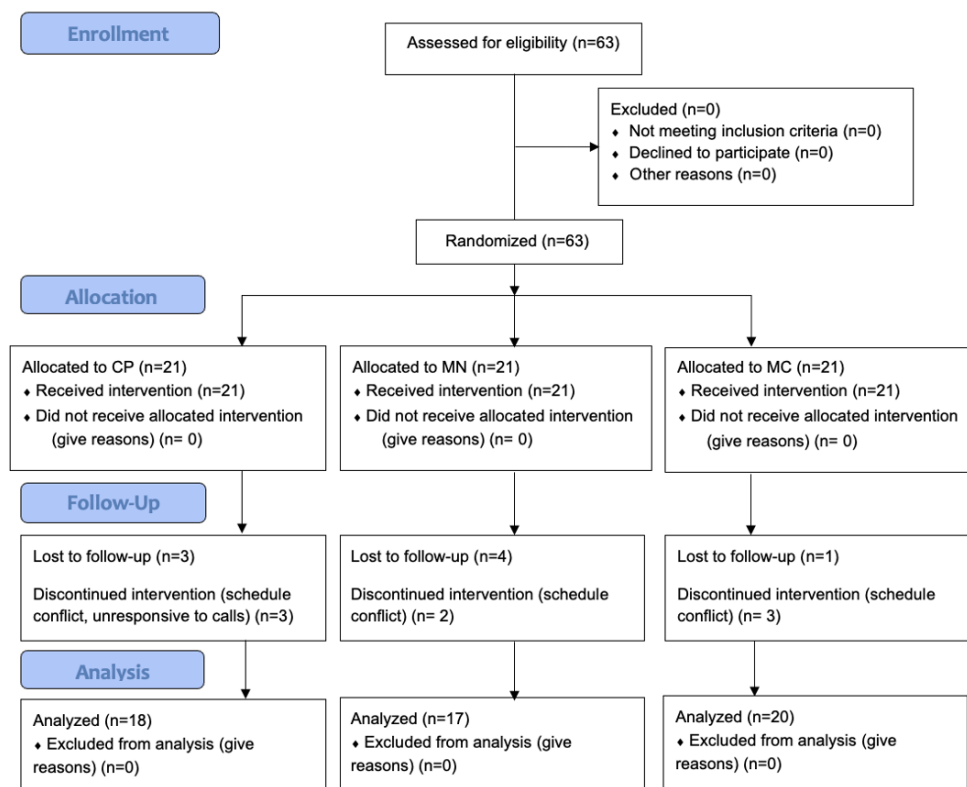


Figure 1. Participant flow diagram.

RESULTS

Demographic and Clinical Characteristics of Participants

A total of 63 participants were assessed for eligibility and randomized into CP, MN, and MC groups, with 21 participants allocated to each group (**Figure 1**). During the study period, eight participants were lost to follow-up, resulting in 55 participants included in the final analysis. The participants ranged in age from 20 to 44 years, with

a mean age of 29 years. Among the analysed participants, 36 (65%) were male and 19 (35%) were female. The duration of acne scars ranged from 4 months to 31 years. Rolling scars were the most frequently observed scar type, followed by boxcar scars. Most participants had Fitzpatrick skin type III (65%), while the remaining participants had skin type IV. Baseline demographic and clinical characteristics were comparable among treatment groups, except for age, which differed significantly between groups at $p < 0.01$ (**Table 1**).

Table 1. Descriptive Characteristics of the studied groups

Variable	CP		MN		MC		F	P
Total participants (%)	18 (32.73)		17 (30.91)		20 (36.36)			
Age (Years)							8.46	0.0036
Mean $\pm$ SD	27.556 (4.501)		29.294 (4.120)		32.600 (6.021)			
Range	20-34		24-37		23-44			
Sex							0.0109	0.9167
Female	6 (33.3)		6 (35.3)		7 (35.0)			
Male	12 (66.7)		11 (64.7)		13 (65.0)			
Variable	CP		MN		MC		H	P
Duration (Years)							2.121	0.3462
Mean $\pm$ SD	7.78(3.42)		8.21(5.59)		13.55(6.57)			
Range	4-17		0.5-22		3-31			
Variable	CP		MN		MC		χ^2	P
	n	%	n	%	n	%		
Scar Type							32.44	0.179
Boxcar	17	37.78	13	28.89	15	33.33		
Icepick	7	38.89	5	27.78	6	33.33		
Rolling	15	31.25	17	35.42	16	33.33		
Hyperpigmented	13	41.94	9	29.03	9	29.03		
Hypopigmented	4	44.44	3	33.33	2	22.22		
Skin Type							1.69	0.429
III	10	27.78	13	36.11	13	36.11		
IV	8	42.11	4	21.05	7	36.84		
V	0	0	0	0	0	0		

Improvement in Acne Scar Severity

The quantitative GASGS scores were evaluated over 12 weeks across the three treatment groups (**Table 2**). The GASGS scores decreased progressively throughout the 12-week follow-up period in all groups, indicating an improvement in acne scar severity compared with baseline. Between-group comparisons using the Kruskal-Wallis test showed no statistically significant differences in GASGS scores among the three treatment groups at baseline, Week 4, Week 8, or Week 12 (all $p > 0.05$). However, within-group analysis using the Friedman test demonstrated significant changes in GASGS scores over the 12-week study period in each treatment group (all $p < 0.001$). Post hoc pairwise comparisons were conducted using the Wilcoxon signed-rank test with exact p-values and Bonferroni correction (adjusted significance level, $\alpha = 0.0083$). After Bonferroni adjustment, no pairwise comparisons remained statistically significant in the CP and MC groups. In contrast, significant reductions in total GASGS scores were observed in the MN group between Week 0 and Week 8 (exact $p = 0.0078$), Week 0 and Week

12 (exact $p = 0.0039$), and Week 4 and Week 12 (exact $p = 0.0078$).

Table 3 presents the qualitative GASGS assessment outcomes across the three treatment groups. One participant (5.56%) in the CP group, two participants (11.76%) in the MN group, and two participants (10.0%) in the MC group demonstrated a one-grade improvement. However, the proportion of participants achieving improvement did not differ significantly among the three treatment groups based on the chi-square test ($\chi^2 = 0.439$, $p = 0.803$).

Representative Clinical Photographs

Figure 2 to **Figure 4** show representative clinical photographs of participants from each treatment group, along with their corresponding qualitative and quantitative GASGS scores over the 12-week study period. Based on the clinical photographs, noticeable improvements were observed across all treatment groups.

Table 2. Quantitative GASGS score among treatment groups over time.

Week	CP	MN	MC	Between-group p-value ^a
Week 0	8.0 (6.0, 12.0)	7.0 (5.0, 11.0)	7.5 (4.0, 9.5)	0.595
Week 4	7.5 (6.0, 12.0)	7.0 (5.0, 9.0)	6.0 (4.0, 9.5)	0.451
Week 8	7.5 (5.0, 12.0)	6.0 (5.0, 9.0) ^c	6.0 (3.5, 9.0)	0.504
Week 12	7.5 (5.0, 12.0)	6.0 (5.0, 9.0) ^{c,d}	5.5 (2.5, 9.0)	0.377
Within-group p-value ^b	<0.001	<0.001	<0.001	

Values are presented as median (interquartile range)

^aKruskal-Wallis test

^bFriedman test

^cSignificant difference compared with Week 0.

^dSignificant difference compared with Week 4

† A Bonferroni correction was applied for six pairwise comparisons within each treatment group, resulting in an adjusted significance level of $\alpha = 0.0083$ (0.05/6). Comparisons with exact $p < 0.0083$ were considered statistically significant

Table 3. Qualitative GASGS score outcomes across treatment groups.

Improvement	CP		MN		MC		χ^2	p
	n	%	n	%	n	%		
Yes	1	5.56	2	11.76	2	10	0.4393	0.8028
No	17	94.44	15	88.24	18	90		
Total	18	100	17	100	20	100		



Figure 2. Clinical photographs of a participant who underwent CP treatment at Weeks 0, 4, 8, and 12. Dermatologist-assigned GASGS scores (qualitative grade/quantitative score): **Dermatologist 1:** Week 0 (IV/11), Week 4 (IV/11), Week 8 (IV/9), and Week 12 (IV/8); **Dermatologist 2:** Week 0 (IV/16), Week 4 (IV/13), Week 8 (III/8), and Week 12 (III/5).

Safety Evaluation

The incidence of procedure-related erythema, burning/stinging, and bleeding across the three treatment groups is presented in **Figure 5**. All adverse events were transient and resolved spontaneously following the procedures. Erythema, burning/stinging sensations, and pinpoint bleeding resolved shortly after treatment, with no cases persisting for several days. No

additional medications were required for the management of these adverse events. As part of routine post-procedure care, participants in the microneedling and combination groups were instructed to apply mupirocin ointment twice daily for five days, while all participants were advised to use a mild cleanser and SPF 50 sunscreen daily.

Erythema was observed across all treatment modalities. Most cases were mild and presented as slight

reddening. Bleeding was observed only in the MN and MC groups, likely attributable to the microneedling component, in which pinpoint bleeding served as the procedural endpoint. No bleeding events were observed in the CP group, and no cases of severe bleeding were reported. Burning or stinging sensations were reported across all treatment groups but were generally mild and tolerable. No severe adverse events were reported. Overall, all treatment modalities demonstrated favourable safety profiles (Figure 5).

Quality of Life Outcomes

Total FASQoL scores decreased from Week 0 to Week 12 across all treatment groups, indicating an improvement in quality of life (Table 4). However, the Kruskal-Wallis test showed no statistically significant differences in FASQoL scores among the CP, MN, and MC groups at Week 0 or Week 12 ($p > 0.05$).

Table 4. Total FASQoL scores across treatment groups.

	CP	MN	MC	H	p-value
Week 0	28 (17)	23 (19)	23.5 (16.5)	0.769	0.6807
Week 12	16.5 (10)	18 (9)	17.5 (8)	0.088	0.9570

Values are presented as median (interquartile range).



Figure 3. Clinical photographs of participant who underwent MN treatment at Weeks 0, 4, 8, and 12. Dermatologist-assigned GASGS scores (qualitative grade/quantitative score): **Dermatologist 1:** Week 0 (IV/15), Week 4 (IV/15), Week 8 (IV/14), and Week 12 (IV/14); **Dermatologist 2:** Week 0 (IV/15), Week 4 (IV/14), Week 8 (III/9), and Week 12 (III/9).



Figure 4. Clinical photographs of participant who underwent MC treatment at Weeks 0, 4, 8, and 12. Dermatologist-assigned GASGS scores (qualitative grade/quantitative score): **Dermatologist 1:** Week 0 (III/6), Week 4 (III/6), Week 8 (III/4), and Week 12 (III/4); **Dermatologist 2:** Week 0 (III/9), Week 4 (III/8), Week 8 (II/3), and Week 12 (II/3).

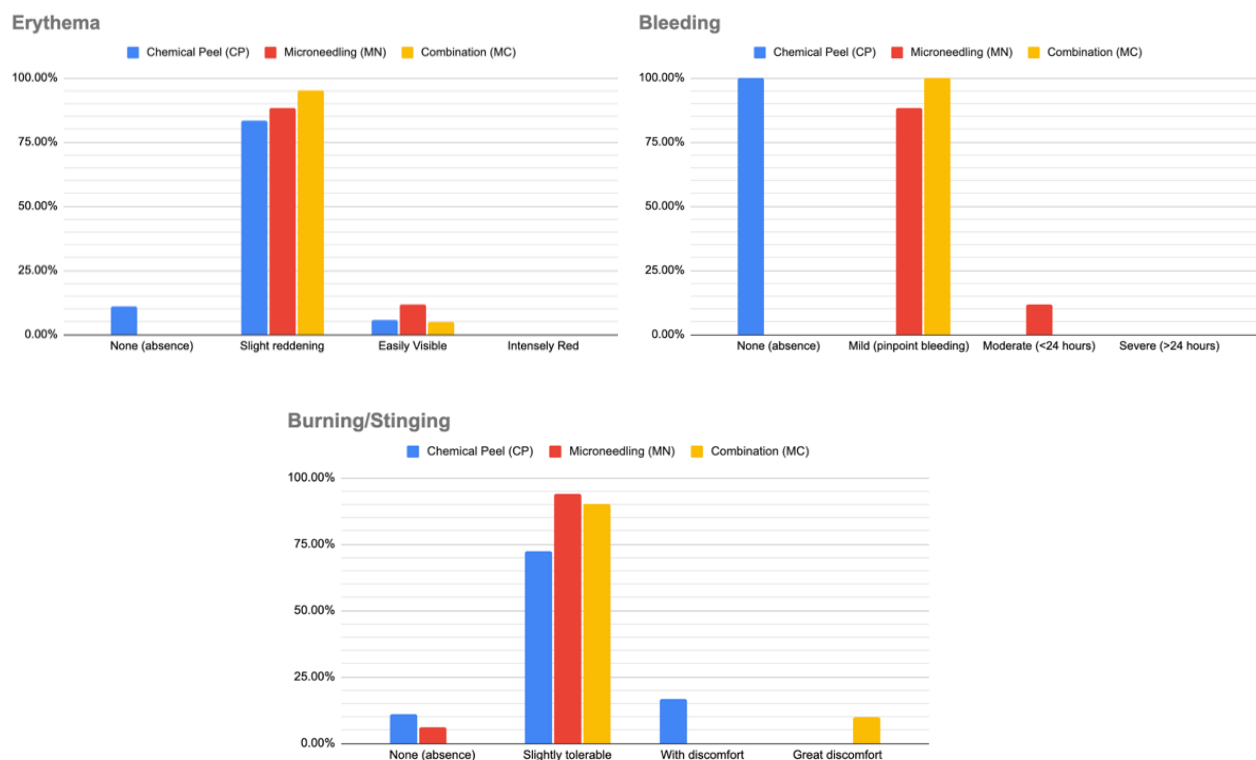


Figure 5. Safety profiles of the three treatment groups.

DISCUSSION

In this study, we evaluated the efficacy and safety of microneedling, Modified Jessner's Solution and combination treatments in participants with atrophic acne scars and Fitzpatrick skin types III–V. All three treatment modalities resulted in modest improvements

in acne scar severity from baseline to Week 12, although no single treatment modality demonstrated consistent superiority. Among the three groups, the CP group showed a less pronounced reduction in scar severity compared with the MN and MC groups, suggesting a potential benefit of microneedling-based approaches in the management of atrophic acne scars. Previous studies

have also suggested that microneedling-based approaches may provide greater clinical improvement compared with CP alone in the management of acne scars. Ali et al. reported that the combination of microneedling and Jessner's solution achieved greater clinical improvement with fewer treatment sessions compared with single-treatment approaches [8]. In another study, Ishfaq et al. demonstrated that microneedling produced better acne scar outcomes compared with 35% glycolic acid peeling in patients with darker skin types [19].

The modest improvement observed in this study may be attributed to several factors, including the limited sample size, relatively short follow-up duration, baseline variations in scar severity, heterogeneity of scar morphology, and the limited number of treatment sessions performed. As improvement in atrophic acne scars depends on gradual collagen remodelling following procedural interventions, longer treatment durations and extended follow-up periods may be necessary to identify clinically meaningful differences between treatment modalities. Accordingly, our findings suggest that, although microneedling, Modified Jessner's Solution and combination treatments may all provide short-term improvement in atrophic acne scars, microneedling and combination treatments may have greater potential for continued improvement with additional treatment sessions.

Differences in scar morphology may have also influenced treatment response among participants. Atrophic acne scars consist of various subtypes, including icepick, boxcar, and rolling scars, which may respond differently to treatment depending on scar depth, width, and degree of dermal tethering. Previous studies have reported that rolling and boxcar scars generally respond better to treatment compared with icepick scars, which are typically deeper, narrower and may require longer treatment duration or scar-specific approaches. El-Domyati et al. reported good to very good responses of rolling and boxcar scars following microneedling, whereas icepick scars demonstrated only moderate improvement [20]. Similarly, Sharad reported that combination therapy with microneedling and chemical peel improved acne scarring across different scar types, except icepick scars [16]. Therefore, the presence of mixed scar subtypes among participants in the present study may have contributed to the modest overall improvement observed across all treatment groups.

Multiple treatment sessions may be required to achieve more pronounced clinical improvement in atrophic acne scars. In a study by Ali et al., noticeable improvement was achieved following multiple treatment sessions, with a mean of 4.6 sessions for combination therapy using microneedling and Jessner's solution, 6.1 sessions for microneedling alone, and 6.75 sessions for Jessner's solution alone [8]. Similarly, Sharad et al. reported that improvements in superficial and moderately deep scars, skin texture, and post-acne pigmentation were observed following five sessions of

microneedling combined with 35% glycolic acid peel [16]. These findings suggest that additional treatment sessions and longer follow-up periods may be necessary to achieve greater clinical improvement.

In the present study, all treatment modalities demonstrated favourable safety profiles. The observed procedure-related reactions, including erythema, pinpoint bleeding, and burning sensations, were mild and transient, resolving spontaneously without additional intervention. These findings are consistent with previous studies demonstrating that microneedling and superficial chemical peel are generally well tolerated, with minimal complications when performed appropriately [8,16].

Beyond clinical assessment, patient-reported outcomes also demonstrated positive changes following treatment. The reduction in FASQoL scores across all treatment groups suggests an improvement in acne scar-related psychosocial burden, including emotional well-being, social interactions, and daily activities. This finding is consistent with previous reports indicating that improvements in acne scars, even when modest, may contribute to enhanced quality of life [17].

Evidence specifically evaluating procedural treatments for atrophic acne scars among Filipino patients remains limited. Therefore, this study provides clinically relevant insights into the management of atrophic acne scars in Filipino patients with Fitzpatrick skin types III-V. Consistent with previous literature, microneedling-based approaches appear to be well tolerated and may contribute to improvement in acne scars among darker skin populations, while combination therapy with Modified Jessner's Solution may offer additional benefits by targeting multiple aspects of acne scar pathology.

Limitations of The Study

This study has several limitations. First, participants underwent only three treatment sessions over a 12-week period. Previous studies evaluating microneedling and chemical peeling for acne scars often used a greater number of treatment sessions or longer follow-up periods, which may allow for more pronounced collagen remodeling and clinical improvement. Consequently, the modest improvements observed in the present study may be attributable to the relatively short treatment duration and follow-up period.

Second, the evaluation of treatment outcomes was primarily focused on atrophic acne scar severity using the GASGS. Other clinically relevant parameters, including post-inflammatory hyperpigmentation (PIH), post-inflammatory erythema (PIE), and changes in overall skin texture, were not formally assessed. In addition, treatment responses according to specific scar subtypes, such as icepick, boxcar, and rolling scars, were not evaluated. Third, although standardised clinical photographs and blinded dermatologists were used to minimise assessment bias, photographic evaluation

remains inherently subjective. In addition, the study was conducted at a single centre with a relatively small sample size, which may limit the generalisability of the findings.

Future studies involving larger sample sizes, additional treatment sessions, and longer follow-up periods are warranted to further evaluate the long-term efficacy and safety of these treatment modalities. Further investigations should also consider scar subtype-specific responses and include additional clinical outcomes, such as pigmentation changes and skin texture improvement.

CONCLUSION

Microneedling, Modified Jessner's Solution, and their combination were well tolerated and associated with improvements in atrophic acne scars and patient-reported quality of life over the 12-week study period. Further studies with larger sample sizes, longer follow-up periods, and additional treatment sessions are needed to further clarify the long-term efficacy of these treatment approaches, particularly among individuals with darker skin types.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest. This study received no commercial funding.

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Retinacula Cutis of Facial Ligaments as Potential Injection Targets in Facial Rejuvenation

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ABSTRACT: The increasing demand for bioremodelling in aesthetic practice has highlighted the need for standardised anatomical frameworks to ensure reproducible and safe treatment outcomes. This manuscript presents a review of an injection-point framework that specifically targets the retinacula cutis (RC) and retaining ligaments (RL) to enhance soft-tissue support. These anatomical structures form a continuous multifibrillar network that compartmentalises facial fat and distributes mechanical forces across tissues. By stimulating both papillary and reticular fibroblasts, targeted injections may promote the production of collagen and elastin, thereby reinforcing the extracellular matrix (ECM) and improving skin firmness. Ultimately, this framework serves as a reproducible foundation for clinicians to achieve more precise, anatomically sound facial rejuvenation.

Keywords: Retinacula cutis, Retaining ligaments, Extracellular matrix, Biostimulators, Bioremodelling

INTRODUCTION

The increasing demand for bioremodelling reflects the growing emphasis on regenerative approaches in aesthetic practice. Numerous commercial products, including cell-based therapies such as platelet-rich plasma (PRP) and cell therapy; cell-free approaches such as growth factors, exosomes, wound dressings, and gene therapy; and tissue-engineering approaches such as biomaterials, bioengineered skin, hydrogels, and three-dimensional (3D) bioprinting, are designed to support tissue repair, regeneration, or remodelling of the extracellular matrix (ECM) [1].

The rapid expansion of injectable treatments has not been without challenges. Different manufacturers recommend product-specific injection techniques and treatment protocols, resulting in considerable variability in clinical practice. The absence of a unified anatomical framework may complicate education, research, and the reproducibility of treatment outcomes. This review presents a clinical perspective and proposes an injection-point framework targeting the retinacula cutis (RC) and retaining ligaments (RL), with the aim of providing an

anatomically informed approach to supporting facial soft-tissue architecture.

RETINACULA CUTIS AND RETAINING LIGAMENTS

Retinacula cutis (RC) constitute a complex fibrous septal network composed of branching connective tissue extensions that originate from fixed points of the periosteum or deep fascia, traverse the superficial musculoaponeurotic system (SMAS), and terminate within the dermis. Retaining ligaments (RL) are dense condensations of fibrous connective tissue that cross through multiple facial tissue planes, anchoring the periosteum or deep fascia directly to the dermis. Collectively, these structures confer structural support to the overlying skin and SMAS, contributing to the maintenance of facial contour and soft-tissue position, while also delineating the face into discrete superficial and deep fat compartments [2].

The facial RL system and RC are consistent anatomical structures identified on intra-tissue endoscopy. They are interpreted as part of a continuous body-wide multifibrillar network of interconnected fibres

that distributes and absorbs mechanical forces across specialized tissues [3]. In a skin ultrasound study, Schelke et al. demonstrated the presence of retacula superficialis and retacula profundus as fibrous tissue separating the superficial and deep fat pad compartments respectively [4]. Similarly, Dowlatshahi et al. described thin bands of irregular dense connective tissue extending between the dermis and underlying fascia, dividing the subcutaneous tissue into small adipose-filled compartments [5]. Facial RC and RL compartmentalise facial fat, maintain tissue structure and contribute to soft tissue support.

The relationship between facial sagging, skin biomechanical properties, and RC architecture using magnetic resonance imaging (MRI) has been investigated [6]. Their findings demonstrated that increased RC density was associated with lower facial sagging scores and improved skin elasticity. As RC density increased, the degree of tissue sagging decreased, suggesting that the RC may play an important role in maintaining facial support and skin quality [6].

Anatomical Structures of the Retinacula Cutis

Dowlatshahi et al. classified the RC into functional categories, including anchors, boundaries, animators, and an accordion (elastic-band) framework. Within this framework, the RL constitute the root structures from which the RC arises, forming the fibrous network through which these functions are clinically expressed. In the present review, emphasis is placed on animators, boundaries, and anchors, as these functions are particularly relevant to the role of facial RL in transmitting forces associated with facial expression, defining aesthetic subunits, and stabilising soft tissues [5].

Animator retinacula cutis

Animator RC are fibrous septal structures that connect the mimetic muscles to the overlying skin, allowing muscular contraction to be transmitted to the dermis and producing facial expression. Facial RL, which are well recognised by aesthetic and reconstructive surgeons, contribute significantly to facial contour by providing structural support, compartmentalising soft tissues, and maintaining the relationship between the skin and underlying facial skeleton.

a. Temporal ligamentous adhesion (TLA)

The TLA is located along the palpable temporal line, extending from the lateral brow toward the superior temporal region, and may also be identified approximately 2 cm diagonally from the lateral orbital rim. In a cadaveric study by Moss et al., the ligament measured approximately 20 mm in height and 15 mm at its base [7]. Histologically, it consists of dense fibrous reticular connective tissue that firmly anchors the temporal soft tissues to the underlying deep temporal

fascia and periosteum [7]. As one of the principal RLs of the upper face, the TLA provides important structural support to the lateral brow and temporal region.

Interestingly, ultrasonographic studies evaluating the ligaments that comprise the “line of ligaments” including the TLA, have demonstrated that these structures undergo minimal time-related dimensional changes. In contrast to adjacent fat compartments and bony structures, their retentive architecture appears to remain relatively stable with age, even as surrounding soft tissues progressively lose volume and support [8]. Beyond its anatomic role, the TLA also serves as an important clinical landmark. Together with the lateral orbital thickening (LOT), zygomatic ligament, and mandibular ligament, it forms a predictable vertical alignment known as the “line of ligaments” which is commonly used to delineate safe injection planes and guide lateral facial volumisation procedures [9,10].

Given its stability and key role within the facial RL framework, the TLA has emerged as a potential target for biostimulatory therapies aimed at enhancing ECM remodelling, improving tissue integrity, and reinforcing facial support structures. Consistent with this concept, supraperiosteal and subcutaneous calcium hydroxylapatite (CaHA), injections along the course of the ligament have been described as part of full-face biostimulatory lifting protocols [9,10].

b. Lateral orbital thickening (LOT)

The LOT is a specialised condensation of the orbicularis RL measuring approximately 7×10 mm and located immediately superolateral to the insertion of the lateral canthal tendon. It inserts into and stabilises the deep surface of the orbicularis oculi muscle fascia. Clinically, its surface landmark corresponds to the lateral orbital rim at the level of the lateral canthus. Together with the orbicularis RL and zygomatic ligament, the LOT contributes to maintaining the contour and structural integrity of the lateral periorbital region. Histologically, it consists of dense fibroconnective tissue rich in RC, providing strong dermal support and facilitating force transmission between deeper structures and the overlying skin [7]. Given its dense concentration of connective tissue elements, the LOT represents an important stabilising structure within the lateral periorbital region.

With ageing, attenuation of the LOT and the adjacent orbicularis RL reduces ligamentous support, allowing inferior displacement of the lower eyelid boundary and contributing to characteristic periorbital changes, including lower eyelid laxity and festoon formation [11]. These age-related changes have also established the LOT as a relevant anatomic landmark in canthopexy and lower-eyelid suspension procedures [11]. The structural role of the LOT in maintaining lateral periorbital support has generated interest in its potential as a target for biostimulatory therapies. By promoting ECM remodelling, neocollagenesis, and tissue

reinforcement, such approaches may help restore structural support and improve age-related changes in the lateral periorbital region.

c. Tear trough ligament (TTL)

The TTL is located along the medial infraorbital rim and extends laterally across the orbital rim as the orbicularis RL. Histological evaluation has confirmed its ligamentous nature, demonstrating well-organised, densely packed collagen bundles with features comparable to those of the zygomatic ligament [12]. Functionally, the TTL serves as a fixed osteocutaneous tether through its attachment to the underlying maxilla.

As facial ageing progresses, the surrounding soft tissues undergo characteristic changes, including atrophy of the deep medial cheek fat, descent of the malar fat pad, and maxillary bone resorption. Because the ligament remains relatively fixed while adjacent structures lose support and volume, a visible depression develops along its course, contributing to the tear trough deformity [13]. Recognition of the ligament's role in the pathogenesis of this deformity has important therapeutic implications. In surgical rejuvenation procedures, release of the tear trough–orbicularis RL complex is considered a key step for effective mobilisation and restoration of the midcheek [12]. Alternatively, the resulting volume deficiency may be addressed through minimally invasive soft-tissue augmentation using dermal fillers or autologous fat transfer, whereas more advanced cases may benefit from surgical ligament release combined with volume restoration as part of periorbital rejuvenation [14].

Boundaries retinacula cutis

Boundaries RC are fibrous septal structures that separate adjacent fascial planes and contribute to the formation of visible contour lines.

a. Buccal-maxillary ligament (BML)

The BML is located lateral to the upper nasolabial fold at the level of the pyriform aperture, where it serves as a fixed boundary between the mobile cheek tissues and the pyriform aperture. Histologically, it has been described as a fibrous septal condensation that separates the superficial medial cheek fat compartment from the nasolabial fat compartment. Although detailed histological characterisation remains limited compared with other facial RLs, it is consistently identified as a dense fibrous structure that delineates these adjacent fat compartments [15].

Functionally, the maxillary ligament acts as an important anatomical restraint that limits the mobility of surrounding soft tissues and maintains compartmental boundaries. As ageing occurs, progressive loss of soft-tissue supports and elasticity leads to descent of the malar fat pad and adjacent superficial cheek fat

compartments, while tissues anchored by the maxillary ligament remain relatively stable, creating differential tissue mobility between the cheek and paranasal regions. This differential pattern of movement creates a transition zone in which descending malar tissues drape over the more firmly supported paranasal region, contributing to the formation, accentuation, and progressive deepening of the nasolabial fold [16].

The biomechanical significance of this ligamentous boundary has important therapeutic implications. For example, barbed thread-lifting techniques utilise the maxillary and zygomatic ligaments as anchoring structures to reposition descended tissues toward the paranasal region and reduce the prominence of the nasolabial fold. Consequently, the maxillary ligament is increasingly recognised as a functional support structure and potential target for volumising and thread-based approaches to nasolabial fold correction [17].

b. Zygomatic ligament

The zygomatic ligament originates from the periosteum along the inferior border of the zygomatic arch and extends superficially to insert into the overlying dermis. It is distributed along the Frankfort horizontal plane, spanning from the lateral orbital region toward the infraorbital rim. Histologically, Duan et al. demonstrated that the ligament consists of a continuous fibroelastic tissue network extending from the periosteum to the skin of the anteromedial midface, supporting its classification as a true osteocutaneous RL [18].

Functionally, the zygomatic ligament plays a critical role in stabilising the midfacial soft tissues and maintaining cheek projection and contour. It helps preserve the structural relationship between the malar fat pads and the zygomatic framework by anchoring the overlying soft tissue envelope to the underlying skeleton. As ageing progresses, attenuation of adjacent soft tissues and progressive loss of support may alter the biomechanical balance maintained by the ligament, contributing to the descent of midfacial structures. These changes are associated with the development of characteristic ageing features, including malar mound formation, festoons, lower eyelid bags, and diminished cheek definition. Consequently, the zygomatic ligament represents a key anatomical structure in the evaluation of midfacial ageing and age-related contour changes.

The central role of the zygomatic ligament in facial support has also led to increasing interest in its therapeutic applications. In particular, placement of collagen biostimulators in proximity to the ligament has been proposed as a means of enhancing structural support and soft-tissue suspension. By reinforcing the ligamentous framework and promoting ECM remodelling, such treatments may attempt to help reposition the malar fat pads against age-related descent, thereby improving midface projection and reducing the appearance of jowling, nasolabial folds, and marionette lines [19].

c. Mandibular septum

The mandibular septum is located along the middle portion of the mandibular border, extending from the jowl region toward the mandibular angle and blending anteriorly with the mandibular ligament. Originally described as a posterior extension of the mandibular ligament arising approximately 1 cm superior to the mandibular border, subsequent cadaveric and histological investigations have provided an alternative interpretation, identifying a platysma-mandibular ligament that functions as a hammock-like structure limiting further descent of jowl fat into the neck. These differing observations highlight the ongoing debate regarding the precise anatomical and histological boundaries of this region [20].

Despite these variations in description, the mandibular septum is generally recognised as an important boundary structure that compartmentalises the lower facial soft tissues and contributes to the maintenance of jawline contour. It is also considered a key fixation point of the platysma to the mandible, playing a significant role in the structural relationships involved in jowl formation [20]. Age-related changes affecting the mandibular septum and surrounding support structures may alter the stability of the lower face. Attenuation, laxity, or dehiscence of this ligamentous framework can facilitate descent of adjacent soft tissues, contributing to the development of jowls in combination with volume loss, SMAS laxity, and mandibular bone resorption. As a result, the mandibular septum has become an important anatomical landmark in lower facial rejuvenation and jawline recontouring procedures [20]. However, more recent cadaveric studies have suggested that jowl formation may be driven predominantly by descent of the cheek fat compartments rather than by true laxity of the adjacent mandibular osteocutaneous ligament. This distinction has important implications for surgical planning and selection of appropriate corrective strategies [21].

Reflecting these anatomical considerations, therapeutic approaches are often tailored to the underlying mechanism of deformity. When ligament release is indicated, dissection is typically confined to the subcutaneous plane to address skin tethering while preserving deeper structural support. Alternatively, or in conjunction with surgical intervention, volumisation of the deflated prejowl sulcus has been proposed as an effective approach for restoring jawline continuity and improving jowl contour [21].

Anchor retinacula cutis

Anchor RC are dense attachments between the dermis and deep fascia or periosteum that act as semi-rigid anchoring points and support soft-tissue contours.

a. Auriculoplatysmal ligament

The auriculoplatysmal ligament (also referred to as the platysma-auricular ligament) arises from the SMAS at the posterior border of the platysma and is located inferior to the earlobe near the angle of the mandible. It functions as an anchoring structure, securing the platysma-SMAS complex to the dermis and fascial tissues of the inferior parotid and auricular regions. Cadaveric dissection and histological studies have demonstrated that the auriculoplatysmal ligament is anatomically distinct from the adjacent Lore's fascia, with both structures consistently identified as dense fibrous connective tissue suitable for fixation during platysmal suspension procedures [22].

Functionally, the auriculoplatysmal ligament serves as an important boundary ligament between the face and neck, contributing to stabilisation of the lateral jawline and upper cervical region through the transmission and distribution of mechanical forces, thereby helping to maintain the continuity and contour of the cervicomandibular junction. With ageing, progressive laxity of the surrounding soft tissues may diminish the effectiveness of this ligamentous support, contributing to blunting of the jawline and loss of cervical contour. Consequently, the auriculoplatysmal ligament has become increasingly relevant in the evaluation of age-related changes affecting the lower face and neck [23].

Given its strategic location and biomechanical function, this ligamentous region has also attracted interest as a potential therapeutic target. Interventions aimed at reinforcing the auriculoplatysmal ligament complex may help restore structural support, improve jawline definition, and enhance neck contour as part of comprehensive facial rejuvenation strategies [23].

b. Masseteric ligament

The masseteric ligament is a fibrous condensation arising from the parotidomasseteric fascia along the anterior border of the masseter muscle and extending superficially to connect with the SMAS and the overlying connective tissue network. Recent anatomical investigations have suggested that the upper and lower masseteric ligaments are more accurately classified as fascio-SMAS ligaments, as they primarily connect the parotidomasseteric fascia to the SMAS rather than inserting directly into the dermis. Dissection-based studies have demonstrated that these ligaments originate from the parotidomasseteric fascia and interweave within the thickness of the SMAS, distinguishing them from true osteocutaneous RL such as the zygomatic ligament [24].

Functionally, the masseteric ligament contributes to stabilisation of the lateral cheek and helps

define the transition between the mobile anterior cheek and the relatively fixed parotid-masseteric region. Through its integration with the SMAS and adjacent retinacular connective tissue network, it plays an important role in maintaining lateral facial contour and soft-tissue support. Unlike osteocutaneous RL, age-related changes affecting this region appear to be driven primarily by the combination of SMAS attenuation, progressive soft-tissue descent, and underlying skeletal remodelling rather than by intrinsic failure of the ligament itself. These changes may alter the balance between mobile and fixed facial compartments, contributing to the contour changes characteristic of facial ageing.

Accordingly, the masseteric ligament region is an important consideration in the assessment of age-related changes affecting the lateral face. Its anatomical relationship with the SMAS and surrounding connective tissue framework has also generated interest in potential therapeutic applications. In particular, biostimulatory injectables placed within this region may help strengthen the retinacular connective tissue network and support the SMAS, thereby contributing to improved structural support and restoration of lateral facial contour [24].

c. Mandibular ligament

The mandibular ligament is a true osteocutaneous RL that originates from the anterior border of the mandible and extends directly into the overlying dermis. It is typically located approximately 5 cm lateral to the facial midline and 1 cm superior to the inferior mandibular border. Cadaveric dissection and histological studies have characterised the mandibular osteocutaneous ligament as a thick fibrous structure arising from the mandible, with mean dimensions of approximately 51.1 ± 4.5 mm lateral to the midline and 9.1 ± 3.1 mm superior to the inferior mandibular border. Histological evaluation confirmed a direct ligamentous connection between the periosteum and the overlying dermis, supporting its classification as a true RL [19].

Functionally, the mandibular ligament provides critical structural support to the lower lip, chin, and prejowl region by anchoring the skin and superficial soft tissues to the underlying mandible. Through this attachment, it helps limit inferior displacement of the overlying tissues and contributes to preservation of jawline definition and lower facial contour. As facial ageing progresses, the interplay of soft-tissue descent, volume loss, and mandibular bone resorption alters the biomechanical environment of the lower face. In this setting, attenuation or laxity of the mandibular ligament may further reduce soft-tissue support, contributing to the development of jowls and progressive loss of jawline definition. These changes underscore the ligament's importance in maintaining lower facial architecture and contour.

Given its central role in lower facial support, the mandibular ligament serves as an important anatomical

landmark in lower facial rejuvenation and jawline contouring procedures [19]. Its strategic position within the RL framework has also led to its recognition as a potential therapeutic target in interventions designed to restore structural support, improve jawline definition, and address age-related changes of the lower face.

d. Mental ligament

The mental ligament is a true osteocutaneous RL located approximately 1 cm lateral to the midline and 1 cm superior to the inferior border of the mandible, extending from the mandibular periosteum to the overlying dermis. Histological evaluation of cadaveric specimens has confirmed its ligamentous nature, demonstrating a dense fibrous connection between the periosteum and the skin. Anatomically, the mental ligament is situated approximately 9.1 ± 2.8 mm lateral to the midline and 8.4 ± 3.8 mm superior to the inferior mandibular border, distinguishing it from the more laterally positioned mandibular osteocutaneous ligament [19].

Functionally, the mental ligament contributes to stabilisation of the chin, lower lip, and prejowl region by anchoring the skin and subcutaneous tissues to the underlying mandible. As part of the lower facial RL framework, it helps maintain the structural relationship between the soft tissues and the facial skeleton, thereby supporting lower facial contour and definition. With advancing age, progressive soft-tissue remodelling, volume loss, and skeletal changes may alter the supportive function of the mental ligament. Attenuation or laxity of this ligamentous attachment can facilitate soft-tissue descent, contributing to deepening of the labiomental crease, loss of chin support, and the development of prejowl fullness. These changes highlight the ligament's role in preserving lower facial architecture and contour.

In conjunction with the mandibular ligament, the mental ligament forms an integral component of the RC support system of the lower face. Consequently, it has become increasingly relevant in the assessment of lower facial ageing and in planning rejuvenation procedures targeting the chin and jawline. Its structural importance also makes it a potential target for regenerative and biostimulatory interventions aimed at restoring tissue support, improving contour, and enhancing lower facial definition [19].

Retinacula Cutis and Retaining Ligaments in Facial Rejuvenation

Certain strategic facial injection points may be used to target the RC and facial RL system, with the potential to improve facial contour, structural support, and the integrity of the ECM. This approach may target both papillary and reticular fibroblasts within the dermis, potentially stimulating collagen and elastin production while promoting tissue remodelling.

Papillary fibroblasts, characterised by the expression of NTN1, PDPN, and CCRL1, play a role in maintaining the rete ridge-like architecture of the dermal-epidermal junction (DEJ). Through the secretion of specialised ECM components, they support epidermal-dermal attachment and contribute to the regeneration of skin appendages during wound healing. On the other hand, the reticular fibroblasts express TGM2, ACTA2, MGP, and peroxisome proliferator-activated receptor gamma (PPAR- γ), which are involved in the production of collagen and elastin fibres that maintain dermal ECM homeostasis. In addition, they facilitate the early phases of wound healing and tissue repair, contributing to tissue remodelling and scar formation [25]. By potentially influencing both papillary and reticular fibroblast populations, treatment targeting the RC may contribute to improved dermal architecture, skin quality and firmness, while potentially providing additional structural support to the facial RL and contributing to facial rejuvenation and contour enhancement.

In facial surgery, specific anatomical structures such as the septa, adhesions, and RLs serve as anchoring points for tissue lifting and support. Similarly, subcutaneous injections of biomaterials may provide local support within the RC network and enhance soft-tissue support, while supraperiosteal injections may provide a deeper structural scaffold that supports these ligamentous attachments and contributes to facial lifting. Furthermore, targeting the RC and RL systems in conjunction with other treatment modalities may provide a more comprehensive approach to facial rejuvenation. While deep supraperiosteal injections address the underlying structural framework, treatment targeting the

RC may provide a superficial "anchor" that may contribute to resisting gravity and maintaining tissue position.

From a treatment-planning perspective, if volumisation is the primary treatment objective, biomaterials may be placed at the periosteal level. If improvement in skin quality is the primary objective, a biostimulatory filler may be considered within the superficial fat layer to target the RC network. By combining these approaches, clinicians may achieve synergistic effects, where deep volumisation and superficial treatment of the RC network work together to optimise both facial contour and skin quality.

Casabona et al. described the concept of the "line of ligaments" which consists of the temporal ligamentous adhesion, lateral orbital thickening, zygomatic ligament, and mandibular ligament. These structures represent key anatomical landmarks in facial rejuvenation. In general, treatment placed lateral to the line of ligaments has been described as being associated with a lifting effect, whereas treatment medial to this line primarily contributes to volumisation [26]. Building on this anatomical concept, we propose a structured injection-point framework that integrates the surface anatomy of the RC and RL system with strategic treatment points to facilitate more systematic and anatomically informed treatment planning. Surface anatomical landmarks corresponding to these structures are summarised in **Table 1**, while the proposed injection points are illustrated in **Figure 1**. Utilisation of these anatomical reference points may assist in standardising injection planning and identifying relevant anatomical structures.

Table 1. Surface anatomy of key facial RL and related structures.

Retinacula cutis	Points	Target ligament	Surface anatomy
Animator	1	Temporal ligamentous adhesion	Palpable temporal line extending from the lateral brow towards the superior temporal region, approximately 2 cm diagonally from the lateral orbital rim
	2	Lateral orbital thickening of the orbicularis retaining ligament	Lateral orbital rim at the lateral canthus
	3	Tear trough ligament	Medial infraorbital rim extending laterally across the orbital rim
Boundaries	4	Buccal maxillary ligament	Upper nasolabial fold and pyriform aperture region
	5	Zygomatic ligament	Straight line extending from the upper margin of the tragus to the infraorbital rim (Frankfort horizontal line)
	6	Mandibular septum	Mandibular border extending from the jowl towards the mandibular angle
Anchor	7	Auriculoplatysmal ligament	Below the inferior border of the earlobe, near the angle of the mandible
	8	Masseteric cutaneous ligament	Anterior border of the masseter muscle
	9	Mandibular ligament	Pre-jowl sulcus and anterior border of the jowl
	10	Mental ligament	Approximately 1 cm lateral to the chin midline and superior to the inferior mandibular border

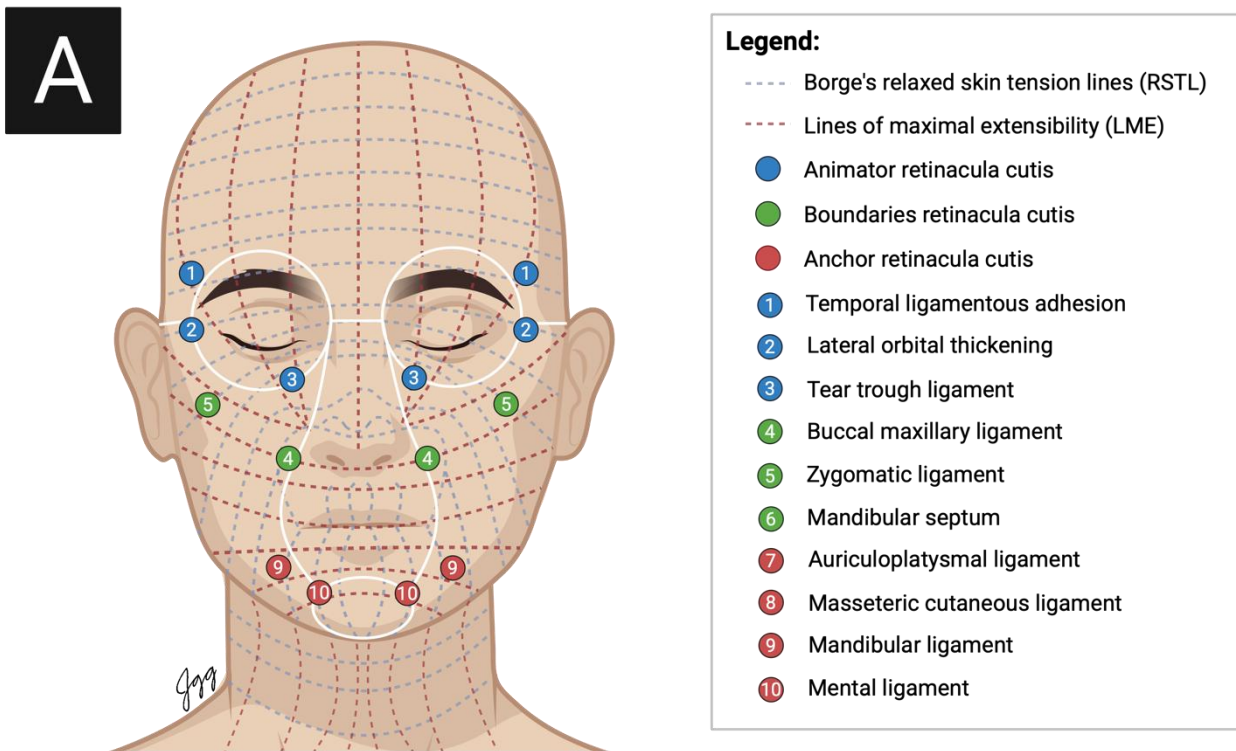
CLINICAL IMPLICATIONS OF THE PROPOSED ANATOMICAL FRAMEWORK

To translate the proposed anatomical framework into clinical practice, the selection of an appropriate biostimulatory agent should take into consideration the anatomical location, tissue plane, and physicochemical properties of the product. The proposed reference points may provide a systematic approach for treatment planning; however, the extent to which a biostimulatory agent can modify or reinforce the RC and RL network is likely to depend on its composition, rheological properties, biodegradability, and biological mechanism of action. Therefore, product selection should be tailored to the anatomical and structural characteristics of the intended treatment site.

Synthetic biodegradable materials include polymers such as poly-L-lactic acid (PLLA), poly-D,L-lactic acid (PDLLA), polydioxanone (PDO), and polycaprolactone (PCL), which undergo gradual degradation and may stimulate ECM remodelling and collagen production. In contrast, non-biodegradable materials, such as polymethyl methacrylate (PMMA), and

other formulations with distinct physicochemical characteristics, as well as ceramic-based agents such as CaHA, zirconia, and alumina, exhibit different degradation profiles and tissue interactions that may influence tissue integration, longevity, adverse event profiles, and the management of complications [27]. These factors are not only critical when navigating high-risk, anatomically delicate zones such as the periorbital/infraorbital, glabellar, and nasolabial regions, where the risks of product migration and vascular complications must be minimised, but are also important considerations when optimising the potential efficacy and safety of the specific injection points for facial rejuvenation proposed in this framework.

As illustrated in **Figure 1** and **Table 1**, each proposed strategic point corresponds to a specific ligamentous attachment or RC system. By selecting agents with rheological profiles that favour appropriate tissue distribution and integration, clinicians may potentially optimise product placement around the relevant ligamentous and RC structures while appropriately considering the safety requirements of high-risk facial regions.



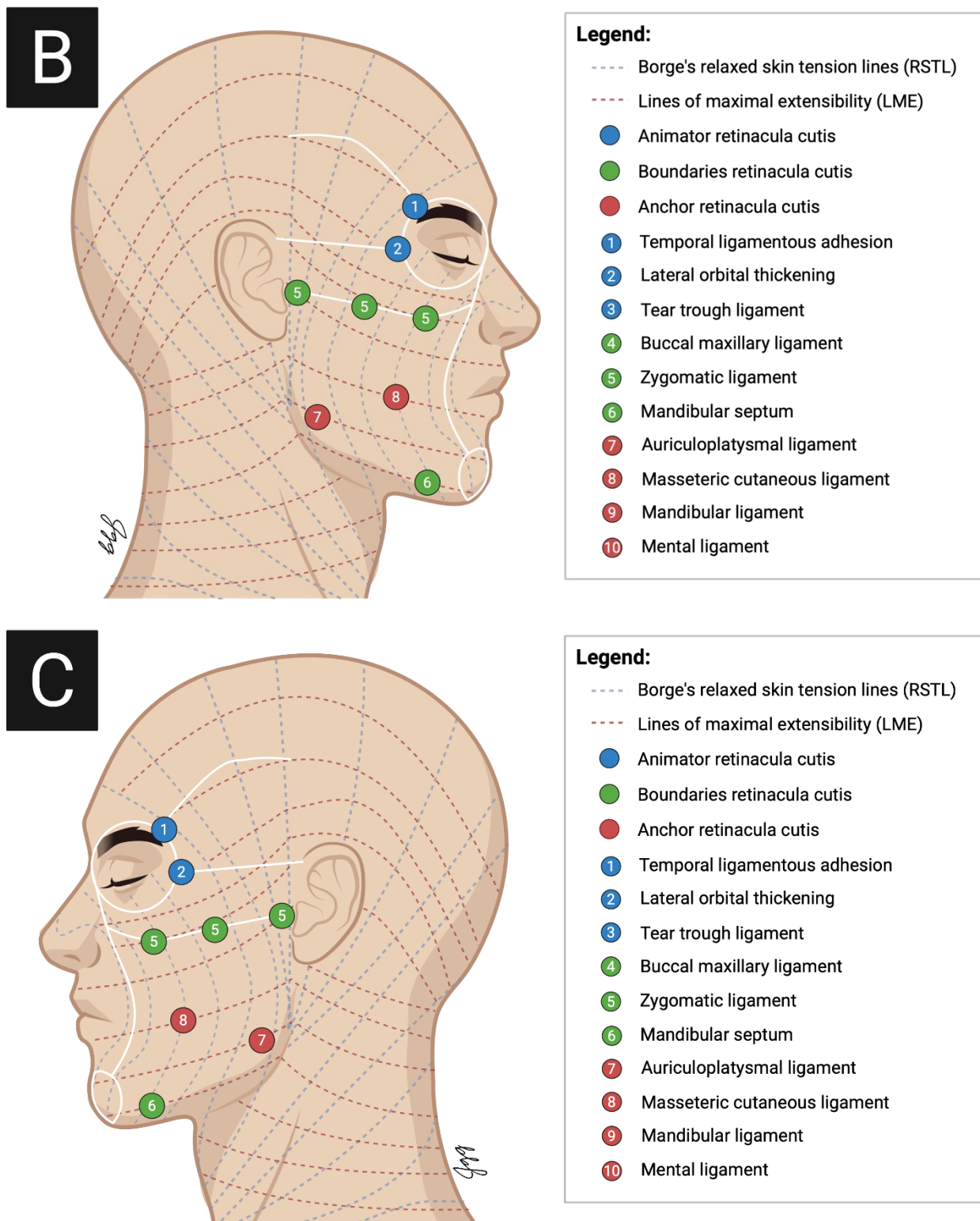


Figure 1. Schematic illustration of facial biomechanical vectors and key RL attachments in the frontal (A), right lateral (B), and left lateral (C) views. Blue dashed lines represent Borge's relaxed skin tension lines (RSTLs), indicating the natural orientation of resting skin tension, while red dashed lines denote the lines of maximal extensibility (LMEs), representing the direction of greatest skin stretch. The panels illustrate the frontal and lateral facial views, highlighting the regional distribution of RSTLs and LMEs across the face and neck. Numbered markers indicate the following structures: (1) temporal ligamentous adhesion, (2) lateral orbital thickening, (3) zygomatic ligament, (4) tear trough ligament, (5) buccal-maxillary ligament, (6) masseteric cutaneous ligament, (7) mandibular ligament, (8) auriculoplatysmal ligament, (9) mandibular septum, and (10) mental ligament. White contours delineate major facial aesthetic subunits and zones influenced by these RL. Understanding the relationship between skin tension lines and RL anatomy may inform surgical incision planning, flap design, scar camouflage, and facial rejuvenation procedures. Created in BioRender. Gatmaitan, J. (2026) <https://BioRender.com/4hl5d8t>

CONCLUSION

In conclusion, this manuscript presents a clinical perspective and proposes a new injection-point framework that targets the RC and RL to potentially enhance soft-tissue support and facial rejuvenation. By shifting the focus towards the anatomical support provided by this retinacula system, we propose a strategic approach to facial rejuvenation that prioritises anatomical anchoring over simple volumisation.

While numerous injectable products utilise these anatomical regions, respect for the underlying structures remains essential to ensure predictable outcomes and patient safety. Standardising techniques in aesthetic and procedural dermatology must be grounded in this practical anatomy. Specifically, clinicians should account for the RC and the RL system whenever performing volumisation or lifting treatments to optimise the “optical lift” and tissue integrity.

Future investigations should focus on quantifying the mechanical effects of interventions targeting these ligamentous structures, for instance, using high-resolution MRI to measure changes in RC density post-injection or 3D surface imaging to evaluate the longevity of the lifting effect. Furthermore, these studies could consider the contribution of age-related skeletal remodelling and bone resorption, as these deep structural changes may alter the tension of the RL and consequently influence the stability of the superficial and deep RC network. Ultimately, this framework may provide an anatomically informed foundation for clinicians to achieve more precise and anatomically sound facial rejuvenation.

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CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest or patents associated with this research.

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Hydroquinone-Induced Exogenous Ochronosis in Filipino Patients: Clinical and Therapeutic Insights from a Case Series

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ABSTRACT: Exogenous ochronosis (EO) is an uncommon but increasingly recognised acquired pigmented disorder associated with prolonged topical hydroquinone use, particularly among individuals with darker skin phototypes. Despite the widespread availability of skin-lightening agents in the Philippines, local data on hydroquinone-induced EO remain limited. This case series describes the clinical, dermoscopic, histopathologic, and therapeutic findings in eight Filipino women with Fitzpatrick skin type IV diagnosed with hydroquinone-induced EO between November 2021 and April 2025. Patients, aged 43–65 years (mean, 55.4 years), presented with progressive bluish-grey to black facial hyperpigmentation after 10–25 years of unsupervised use of 2% hydroquinone, commonly in combination with tretinoin 0.05%. Dermoscopy consistently demonstrated reddish-brown reticular structures obliterating follicular openings, black dots, and elongated serpentine vessels, while histopathology in 6 biopsied cases revealed characteristic ochre-colored globules, degenerated collagen fibers, dilated vessels, and prominent solar elastosis. Following diagnosis, hydroquinone was discontinued, strict photoprotection was advised, and treatment with safer skin lightening agents was initiated. In addition, selected patients underwent laser therapy, with significant clinical improvement observed in one patient following sequential 1064 nm Q-switched Nd:YAG (QSNY) laser and 675 nm laser treatment, whereas 577 nm laser produced modest responses. These findings highlight the characteristic clinical, dermoscopic, and histopathological features of hydroquinone-induced EO in Filipino women, which may facilitate early recognition of EO in individuals with darker skin phototypes. Dermoscopy may aid clinical recognition, while histopathology can provide confirmation when indicated. Prompt discontinuation of hydroquinone and an individualised approach to treatment may be considered in the management of EO.

Keywords: Exogenous ochronosis, Hydroquinone, Clinical, Dermoscopy, Histopathology, Therapy

INTRODUCTION

Exogenous ochronosis (EO) is a facial pigmented disorder characterised by asymptomatic blue-black and grey-black hyperpigmented macules and patches with changes in skin texture, typically on the face, neck, back, and extensor surfaces of the extremities [1]. It is more commonly reported in individuals with darker skin phototypes, particularly among Black South Africans and Hispanic populations with prolonged use of hydroquinone-containing skin-lightening products. However, increasing numbers of cases have also been reported in individuals with lighter skin tones, including those of East Indian, Asian, and European descent [2]. Historically, the condition has been primarily associated with prolonged use of high-concentration hydroquinone

and other compounds, such as phenol, resorcinol, and quinine, with unprotected sun exposure as an exacerbating factor [1]. Dermoscopy and skin biopsy can aid in diagnosis, with histopathological examination providing confirmation when indicated. Management involves discontinuing the offending agent, strict photoprotection, and considering topical or procedural therapies; however, no treatment has been consistently proven to be universally effective [1].

An earlier case series of EO in Filipino patients has been reported, with a focus on describing the dermoscopic findings of EO to aid diagnosis [3]. In contrast, the present case series describes the clinical, dermoscopic, and histopathological findings, as well as treatment approaches. The patients in the present case series are distinct from those in the previously published

cohort, with no overlap between the two patient populations. This case series provides additional local clinical experience with EO associated with prolonged, unsupervised hydroquinone use, including its diagnosis and management.

METHODOLOGY

Participants

We present a series of eight Filipino female patients diagnosed with EO and Fitzpatrick skin type IV who consulted at our institution between November 2021 and April 2025 (**Table 1**). Written informed consent for publication, including the use of clinical photographs, was obtained from all patients. Patients were aged 43 to 65 years, with a mean age of 55.4 years. None exhibited features of endogenous ochronosis, such as dark urine, arthralgia, scleral hyperpigmentation, or thickened cartilage. All cases presented with progressive facial hyperpigmentation persisting for 2 to 10 years. Notably, all had a history of prolonged use, ranging from 10 to 25 years, of 2% hydroquinone in solution and cream formulations, some of which also contained tretinoin 0.05%. Photoprotection practices varied. The baseline severity of facial hyperpigmentation, assessed using the mMASI [4], ranged from 4.2 to 18.0 (mean, 10.5).

Dermoscopic Assessment

The patients presented with bluish-grey to black facial hyperpigmentation, most pronounced over the malar and zygomatic areas. Closer inspection revealed caviar-like papules (2 cases) and confetti-like depigmentation (all cases) (**Figure 1**). Dermoscopy (HEINE Optotechnik GmbH & Co. KG, Herrsching, Germany) showed reddish-brown reticular structures obliterating follicular

openings, black dots, interspersed elongated serpentine vessels, and confetti-like depigmentation. Histopathology was performed in 6 cases, while 2 patients declined biopsy. The biopsied cases showed ochre-coloured globules, dilated vessels, degenerated collagen fibres, and prominent solar elastosis. Histopathological examination of areas with confetti-like depigmentation revealed fibrosis and ochronotic pigment, suggesting scarring (**Figure 2**).

Management and Outcome

All patients were prescribed topical therapy aimed at promoting collagen remodelling and skin repair and were advised to discontinue hydroquinone and use daily photoprotection. One patient (case 5) underwent sequential treatment with a 1064 nm Q-switched Nd:YAG (QSNY) laser (Tri-Beam Premium, Jeisys Medical, Korea), under topical anaesthesia with 10.56% lidocaine occlusion (fluence, 4–6 J/cm²; spot size, 4 mm; frequency, 2–6 Hz; pulse duration, 5–10 ns; single pass), followed by a 675 nm laser (RedTouch, DEKA Laser, Italy; power, 6.0 W; dwell time, 150 ms; spacing, 2000 µm). Significant clinical improvement was observed after 4 sessions of QSNY laser followed by 6 sessions of the 675 nm laser. The mMASI score decreased from 14.7 at baseline to 5.4 after treatment, representing a 63.3% reduction (**Figure 3**). Two patients (cases 1 and 2) were treated with the 577 nm ProYellow laser (Asclepion Laser Technologies GmbH, Jena, Germany), resulting in mild improvement after 2 to 3 sessions. Laser therapy was administered at monthly intervals. No adverse effects were reported during treatment. The remaining five patients who received conservative management without additional laser therapy, were subsequently lost to follow-up; therefore, treatment outcomes could not be assessed.

Table 1. Demographic and clinical characteristics of the patients.

Patient no.	Sex	Age (years)	Hydroquinone use (2%)	Sun protection	mMASI (baseline)	Additional treatment
1	Female	55	10 years	Yes	5.4	577 nm ProYellow laser (3 sessions)
2	Female	54	10 years	Occasional	6.9	577 nm ProYellow laser (2 sessions)
3	Female	60	25 years	None	16.2	-
4	Female	43	10 years	Occasional	4.2	-
5	Female	61	10 years	Occasional	14.7	1064 nm QSNY laser (4 sessions) 675 nm laser (6 sessions)
6	Female	54	20 years	Occasional	9.9	-
7	Female	51	10 years	None	18.0	-
8	Female	65	10 years	Yes	9.0	-

Modified MASI total score = 0.3 A(f) D(f) + 0.3 A(lm) D(lm) + 0.3 A(rm) D(rm) + 0.1 A(c) D(c)

The range of the total score is 0 to 24.

Area and darkness are scored as follows:

Area of involvement: 0 = absent, 1 = <10%, 2 = 10%-29%, 3 = 30%-49%, 4 = 50%-69%, 5 = 70%-89%, and 6 = 90%-100%;

Darkness: 0 = absent, 1 = slight, 2 = mild, 3 = marked, and 4 = severe.

Area (A), Darkness (D), forehead (f), left malar (lm), right malar (rm), and chin (c).



Figure 1. Clinical presentation of eight cases at baseline showing bluish-grey to black facial hyperpigmentation, most prominent over the malar and zygomatic areas, with confetti-like depigmentation. **A**, case 1; **B**, case 2; **C**, case 3 with additional caviar-like papules; **D**, case 4; **E**, case 5; **F**, case 6; **G**, case 7 with additional caviar-like papules; **H**, case 8.

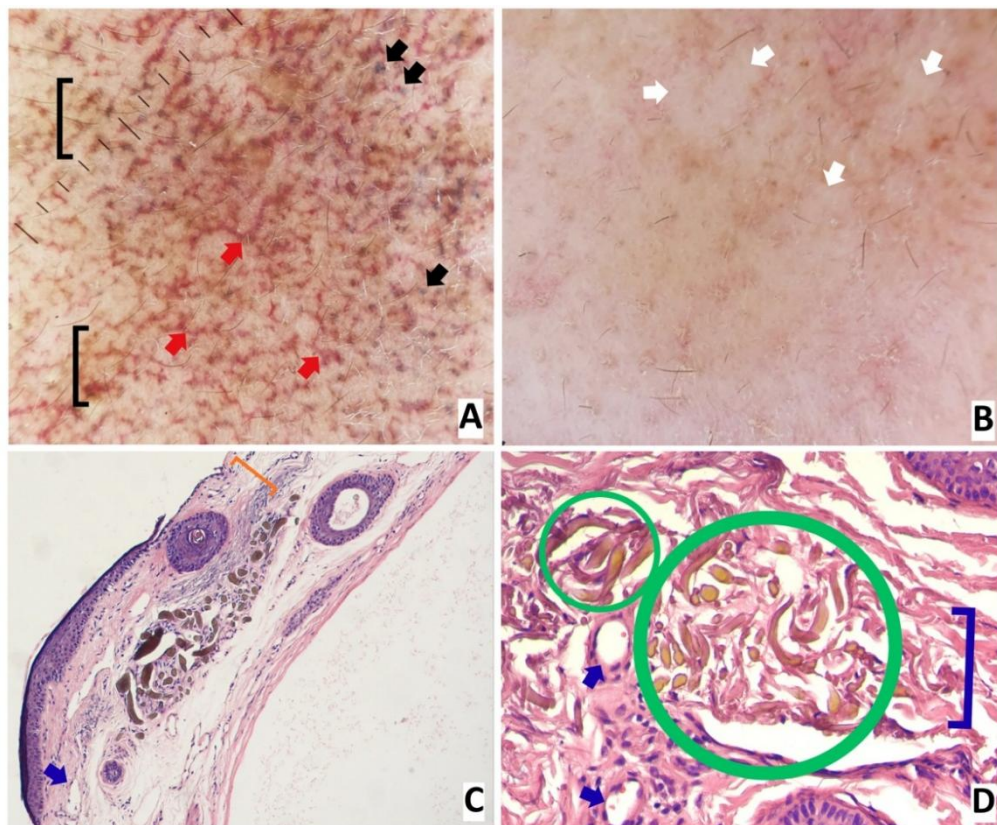


Figure 2. Dermoscopy showing (A) reddish-brown reticular structures obliterating follicular openings (black bracket), black dots (black arrow), interspersed elongated serpentine vessels (red arrow), and (B) confetti-like depigmentation (white arrow). Histopathology showing (C) prominent solar elastosis (orange bracket) [case 3, H&E, $\times 10$] and (D) ochre-coloured globules (green circle), dilated vessels (blue arrow), and degenerated collagen fibres (blue bracket) [case 4, H&E, $\times 40$]. Biopsy site: preauricular area. The preauricular area was selected to minimise visible scarring.



Figure 3. Case 5. Clinical response to sequential laser therapy in hydroquinone-induced EO. (A) Baseline showing grey-black facial hyperpigmentation involving the forehead, malar, zygomatic, and chin regions (mMASI, 14.7); (B) Improvement in facial pigmentation after 4 sessions of 1064 nm QSNY laser treatment; (C) Further reduction in facial hyperpigmentation following 6 sessions of a 675 nm laser as sequential treatment (mMASI, 5.4).

DISCUSSION

EO is a rare but potentially irreversible complication of prolonged topical hydroquinone use. The proposed pathogenesis of EO is prolonged hydroquinone exposure inhibits homogentisate 1,2-dioxygenase, resulting in localized accumulation and polymerization of homogentisic acid within altered elastic fibers [2], a process that may further be potentiated by chronic ultraviolet exposure. Although traditionally associated

with high-concentration formulations, accumulating evidence indicates that EO may also develop following long-term use of 2% hydroquinone [2,5,6], particularly in the absence of physician supervision and consistent photoprotection. Earlier reports likewise proposed that extended duration of hydroquinone exposure, rather than concentration alone, is the principal factor in the development of EO. It has also been suggested that the percentage of hydroquinone stated by a manufacturer may not necessarily represent the actual concentration

of hydroquinone in a product [7]. Similarly, in the present case series, all patients had prolonged exposure to products labelled as containing 2% hydroquinone before disease onset. These observations are consistent with earlier findings that chronic exposure to hydroquinone-containing regimens, together with inconsistent photoprotection, may contribute to the development of EO. However, the actual hydroquinone concentrations of the products used by our patients were not independently verified and may have differed from the concentrations stated by the manufacturers.

The clinical, dermoscopic, and histopathological findings in the present series were consistent with those previously reported in Filipino patients with EO [3]. Dermoscopy in our patients demonstrated reddish-brown reticular structures with obliteration of follicular openings, black dots, elongated serpentine vessels, and confetti-like depigmentation. Similarly, Belizario et al. reported blue-gray and black dots, annular and arcuate structures, reddish- to dark-brown curvilinear structures, obliteration of follicular openings, telangiectasias, and confetti-like depigmentation [3]. The similarity of these dermoscopic features, particularly the presence of pigmentary structures associated with obliteration of follicular openings, may aid in distinguishing EO from other facial hyperpigmentation disorders such as melasma. Histopathologically, our findings of ochre-colored deposits, solar elastosis, and degenerated collagen were likewise consistent with previous reports, in which ochre-colored bodies in the papillary dermis were the characteristic finding, accompanied variably by solar elastosis, telangiectasia, and inflammatory changes [3].

Management of EO remains challenging because no therapy has consistently demonstrated predictable efficacy. Current treatment is centred on discontinuation of the offending agent, strict photoprotection, and individualised use of topical or procedural therapies [2,5]. In the present case series, in addition to discontinuation of hydroquinone and photoprotection, selected patients received laser therapy, including sequential treatment with a 1064 nm QSNY laser followed by a 675 nm laser in one patient and a 577 nm yellow laser in 2 patients. Laser therapy was offered as an additional treatment; however, only three patients proceeded because the remaining patients were unable to afford the associated costs and opted for topical therapy.

The 1064 nm QSNY laser has been reported to improve pigmentation in EO through fragmentation of dermal ochronotic pigment, thereby facilitating its clearance by dermal macrophages and lymphatics [8]. In our patient, the subsequent use of the 675-nm laser was intended to promote dermal collagen remodelling and improve skin texture after pigment reduction, as this wavelength has been shown to stimulate neocollagenesis and extracellular matrix remodelling [9]. To our knowledge, there are currently no published reports describing the use of the 675 nm laser for the treatment

of EO, particularly as a sequential modality following QSNY laser therapy. The 577 nm yellow laser was selected to target associated facial erythema and telangiectasia [10]. Although favourable clinical improvement was observed in the patient who received sequential QSNY and 675-nm laser treatment, further studies are warranted to evaluate the therapeutic efficacy, safety profile, optimal treatment parameters, and long-term outcomes of this sequential laser approach in the management of EO. In case 5, mMASI was used to assess treatment outcome. Although mMASI was developed and validated for melasma and has not been validated for EO, it was used as an exploratory measure to provide standardised quantitative documentation of facial pigment burden, despite the absence of a disease-specific severity index for EO.

Recent clinical guidance continues to recognise hydroquinone as the gold-standard topical depigmenting agent when prescribed appropriately [6]. Rather than discouraging its use, current recommendations advocate limiting treatment duration, ensuring physician supervision, and transitioning to maintenance regimens or alternative depigmenting agents, including azelaic acid, tranexamic acid, thiamidol, kojic acid, niacinamide, and arbutin, when clinically appropriate [6]. More recently, 2-mercaptopyridine-5-yl glycine has emerged as a promising non-hydroquinone depigmenting agent, demonstrating efficacy comparable to 4% hydroquinone with improved tolerability in patients with facial melasma, making it a potential alternative for long-term pigmentary management [11].

Collectively, our findings highlight the potential association between prolonged, unsupervised hydroquinone use and the development of EO, underscoring the importance of patient education, regular follow-up, and clinical monitoring for early signs of EO to minimise irreversible pigmentary sequelae. This study is limited by its small sample size, uncontrolled design, and incomplete follow-up, while the actual hydroquinone concentrations of products labelled as 2% were not independently verified and may have differed from the manufacturers' stated concentrations.

CONCLUSION

EO is a potentially preventable complication of prolonged, unsupervised use of topical hydroquinone-containing skin-lightening products. Our findings show that hydroquinone-induced EO may occur following long-term use of products containing 2% hydroquinone and highlight the value of dermoscopy and histopathology for clinical recognition and diagnosis. Prompt discontinuation of hydroquinone, strict photoprotection, and individualised management remain the cornerstones of treatment, although therapeutic outcomes, including those with laser therapy, are variable. Physician oversight, patient education, and adherence to recommended treatment duration may help reduce the risk of EO.

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None

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest.

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Combined 308-nm Excimer Lamp and Tacrolimus Therapy for Linear Morphea Evaluated by Clinical Assessment and Ultrasound (USG): A Case Report of a 38-Year-Old Woman

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ABSTRACT: Morphea is a chronic inflammatory disease characterised by skin sclerosis. The disease has several proposed classifications, one of which is linear morphea, characterised by lesions that may follow Blaschko lines and can involve the dermis, subcutaneous tissue, muscle, and underlying bones. Various treatments have been explored for linear morphea, including phototherapy, immunomodulators, and topical treatments like tacrolimus and imiquimod. This case report presents a novel treatment approach using 308-nm excimer lamp therapy in combination with tacrolimus 0.1% ointment for a 38-year-old woman diagnosed with linear morphea. The patient had a linear depressed plaque on her forehead that evolved over four years. After 20 weeks of combined treatment, significant improvement was observed, including a reduction in lesion length, improved dermal thickness and complete resolution of pruritus. The treatment was well tolerated with no major adverse effects, suggesting that this combination may represent a promising alternative to traditional therapies for linear morphea.

Keywords: Linear morphea, Excimer lamp therapy, Tacrolimus, Phototherapy

INTRODUCTION

Linear morphea is a subtype of localised scleroderma, characterised by band-like, linear lesions that can involve the dermis, subcutaneous tissue, muscles, and even underlying bones. It is the most common form of localised morphea in children and adolescents, typically presenting as a unilateral lesion on the extremities, face, or scalp, often following the lines of Blaschko [1]. When it affects the head and neck, the condition can result in skin atrophy, growth retardation, muscle atrophy, and may resemble a “cut of the sword”, known as “en coup de sabre” [1]. The etiology of morphea remains poorly understood and is thought to involve an interplay between genetic susceptibility and environmental triggers. Its pathogenesis is believed to be autoimmune-mediated, progressing from an initial inflammatory phase to fibrosis and cutaneous sclerosis [1].

Various treatment options have been explored for linear morphea, including phototherapy (especially narrowband UVB and UVA), as well as oral and topical therapies such as Vitamin D derivatives and immunomodulators like methotrexate with or without corticosteroids [1]. Additionally, case reports have

suggested other therapeutic options, including topical treatments like tacrolimus and imiquimod, as well as oral agents such as mycophenolate mofetil, cyclosporine, hydroxychloroquine, azathioprine, retinoids, intravenous immunoglobulin (IVIG), rituximab, and infliximab. Other approaches, including anti-fibrotic drugs, JAK inhibitors, autologous stem cell transplantation, and supportive therapies like physiotherapy and massage, have also been proposed [2].

Excimer lamp therapy is a targeted phototherapy modality that delivers high-intensity monochromatic UVB light at 308-nm directly to affected skin, thereby limiting irradiation of uninvolved tissue. Given its anti-inflammatory and immunomodulatory effects, as well as its ability to provide localised treatment with minimal exposure to surrounding healthy skin, excimer lamp therapy may represent a promising therapeutic option for localised forms of morphea such as en coup de sabre [3]. However, to our knowledge, the combined use of 308-nm excimer lamp therapy and topical tacrolimus for the treatment of linear morphea has not been previously reported. Herein, we report the use of this combination therapy in a 38-year-old woman with linear morphea.

CASE PRESENTATION

A 38-year-old woman presented with a solitary, hyperpigmented, linear depressed plaque on her forehead that had gradually developed over four years. The lesion initially appeared as an erythematous, asymptomatic patch and subsequently evolved into an intermittently pruritic (Visual Analogue Scale (VAS) score: 3/10), hyperpigmented, linear depressed plaque that with progressive extension. The lesion was non-painful, and no other associated symptoms were reported. Prior to presentation at our institution, the patient had sought consultation elsewhere and received topical antifungal therapy as well as three sessions of unspecified laser treatment at an aesthetic clinic, without clinical improvement. There was no history of preceding trauma, infection, radiation exposure, or other identifiable triggering factors. The patient had no known comorbidities and no family history of autoimmune or connective tissue disorders. On examination, the lesion was consistent with linear morphea (en coup de sabre), with no signs of systemic involvement.

MANAGEMENT

A 4-mm skin punch biopsy was performed from the active border of the lesion. Histopathological examination revealed early dermal sclerosis characterised by thickened collagen bundles, reduced spacing between collagen fibres, and a mild superficial and periadnexal lymphohistiocytic infiltrate. The presence of preserved adnexal structures surrounded by thickened collagen fibres supported a diagnosis of early-stage morphea. Although similar histopathological findings may occur in systemic sclerosis, the diagnosis of linear morphea was established based on the characteristic linear morphology of the lesion and the absence of clinical features suggestive of systemic

sclerosis, including Raynaud phenomenon, sclerodactyly, or internal organ involvement (**Figure 1**). Following clinicopathological correlation, a diagnosis of linear morphea (en coup de sabre) was established.

Given the location of the lesion, a skull radiograph was performed to assess potential underlying osseous involvement, which demonstrated no evidence of bony abnormalities. Laboratory investigations for connective tissue diseases were not performed due to financial constraints. Prior to treatment initiation, high-frequency ultrasonography (LOGIQ P9, GE HealthCare, USA) of the affected area was performed using a 12-MHz linear transducer to assess the extent of cutaneous involvement and measure dermal thickness.

The patient was initiated on 308-nm excimer lamp therapy at an initial dose of 100 mJ/cm² administered twice weekly. The dose was increased by 50 mJ/cm² per session for the first four sessions, followed by increments of 10 mJ/cm² per session for the subsequent 35 sessions. In addition, tacrolimus 0.1% ointment, a topical calcineurin inhibitor, was applied to the lesion twice daily for its anti-inflammatory and immunomodulatory effects. The patient completed 40 sessions of excimer lamp therapy over 20 weeks, with a cumulative dose of 17,800 mJ/cm² while continuing topical tacrolimus 0.1% ointment throughout the treatment period.

Treatment response was assessed using both subjective and objective measures. Subjective symptoms, including tightness and pruritus, were evaluated using the VAS. Objective clinical evaluation included serial measurements of lesion dimensions, while dermal thickness and echogenicity were assessed using high-frequency ultrasonography to monitor tissue remodelling. Serial measurements were obtained at the centre of the lesion using the same anatomical site, standardised patient positioning, and a single operator

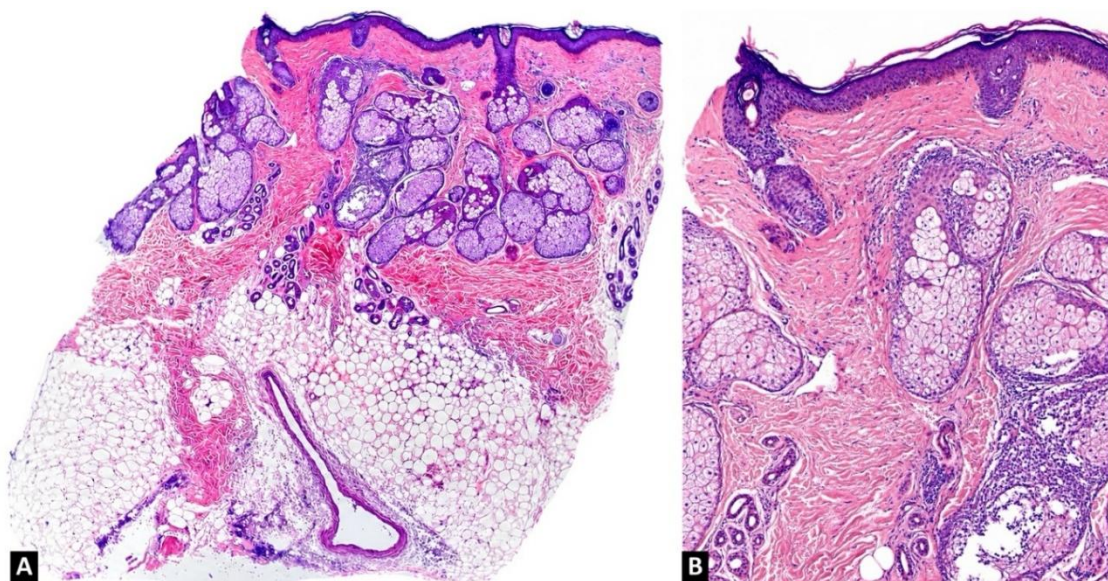


Figure 1. Hematoxylin and eosin (H&E) staining demonstrates a squared silhouette with mild spongiosis and prominent basal layer hyperpigmentation of the epidermis. Dermal changes are characterised by a mild superficial lymphohistiocytic infiltrate and reduced spacing between thickened collagen bundles. (A) $\times 4$ magnification; (B) $\times 10$ magnification.

to ensure measurement consistency. Disease activity and damage were further assessed using the Localised Scleroderma Cutaneous Assessment Tool (LoSCAT) [4]. All assessments were performed at baseline and after every 10 sessions of excimer lamp therapy.

CLINICAL OUTCOME

Following initiation of therapy, the patient demonstrated progressive clinical improvement throughout the treatment course. After 20 weeks, high-frequency ultrasonography revealed evidence of dermal remodelling with increased dermal thickness and improved sonographic appearance. Subjective symptoms improved significantly, with complete resolution of pruritus (VAS score: 0/10) and absence of residual tightness. Clinically, the lesion length decreased from 4.4 cm at baseline to 3.4 cm after treatment, representing a 22.73% reduction in lesion size (**Figure 2**). No new lesions developed during the treatment period.

Assessment using the LoSCAT demonstrated complete resolution of disease activity (**Table 1**), with the modified Localized Scleroderma Skin Severity Index (mLoSSI) score improving from 3 at baseline to 0 after treatment. The Localized Scleroderma Skin Damage Index (LoSDI) remained stable at 3, indicating persistent

but non-progressive cutaneous damage, with improvement in dermal atrophy and residual dyspigmentation. Both therapies were well tolerated, and no notable adverse effects were observed during the treatment period.

Table 1. Disease activity and damage assessed using the Localised Scleroderma Cutaneous Assessment Tool (LoSCAT).

LoSCAT component	Baseline	Post-treatment
mLoSSI		
Erythema	0	0
Skin thickness	0	0
New lesion extension	3	0
Total mLoSSI	3	0
LoSDI		
Dermal atrophy	2	1
Subcutaneous atrophy	0	0
Dyspigmentation	1	2
Total LoSDI	3	3

Scores were assigned as follows: 0=absent, 1=mild, 2=moderate, and 3=severe.

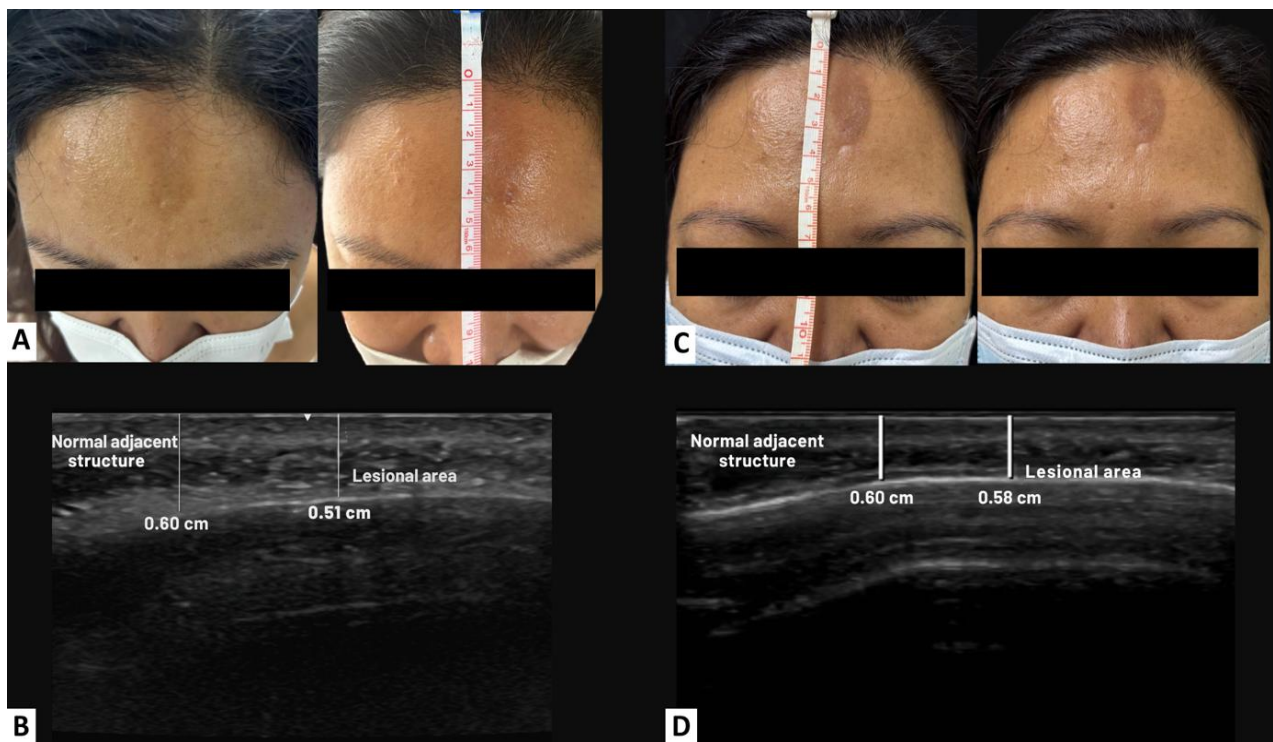


Figure 2. Clinical and ultrasonographic findings before and after treatment. **(A)** Clinical photograph showing a solitary, ill-defined, irregularly shaped hyperpigmented linear depressed plaque on the forehead at baseline (lesion length, 4.4 cm); **(B)** Baseline high-frequency ultrasonography demonstrating reduced dermal thickness; **(C)** Clinical photograph after 40 treatment sessions, showing a reduction in lesion length to 3.4 cm; **(D)** Follow-up ultrasonography after 40 treatment sessions demonstrating increased dermal thickness, consistent with dermal remodelling.

DISCUSSION

This case demonstrates a favorable clinical response to the combined use of 308-nm excimer lamp therapy and topical tacrolimus in a patient with linear morphea, highlighting the potential role of targeted localised treatment approaches. Morphea is a chronic autoimmune condition characterised by inflammation-driven fibrosis and excessive collagen deposition in the dermis and subcutaneous tissues [2], which may explain the reduction in dermal thickness observed in this patient.

Most studies have described narrowband UVB and UVA1 as effective phototherapy options for morphea. A local case series by Frez et al. demonstrated favorable clinical outcomes with narrowband UVB in patients with morphea, further supporting the utility of phototherapy in disease management [5]. However, conventional phototherapy involves irradiation of both affected lesions and surrounding normal skin [6]. In contrast, targeted modalities such as excimer light deliver high-intensity UVB directly to affected areas, potentially enhancing treatment efficacy while minimising unnecessary exposure to uninvolved skin [6,7]. These findings suggest that targeted excimer phototherapy may represent an effective tissue-sparing alternative for localised facial morphea.

Previous reports have described the use of 308-nm excimer light in localised scleroderma, with improvements in skin texture observed in most treated lesions [7]. Subsequent reviews have highlighted 308-nm excimer lamp phototherapy as a targeted UVB modality that selectively delivers high-dose irradiation to affected skin while sparing surrounding healthy tissue, supporting its potential role in the management of superficial morphea [3,8]. Consistent with these findings, our patient demonstrated marked clinical improvement, including complete resolution of pruritus, reduction in lesion length, ultrasonographic evidence of dermal remodelling, and complete resolution of disease activity based on LoSCAT assessment following combined excimer lamp and topical tacrolimus therapy.

Early treatment remains important in morphea to minimise disease progression and prevent irreversible damage [9]. As this patient presented during the active sclerotic phase, and excimer phototherapy has been suggested as a potential treatment option for early, localised sclerotic lesions [10], timely intervention was considered appropriate. The treatment regimen was well tolerated, with only minimal, transient erythema following excimer therapy sessions that resolved within 24 hours, and no significant adverse effects associated with either modality. This favourable safety profile is consistent with previous reports indicating that phototherapy and topical immunomodulators are generally well tolerated in localised morphea [2].

The combination therapy may exert complementary therapeutic effects. Phototherapy

induces immunomodulatory effects in lesional skin, including modulation of cytokines involved in fibrogenesis, alteration of immune cell activity through mechanisms such as apoptosis of pathogenic T lymphocytes, and regulation of pro-fibrotic mediators, including transforming growth factor- β and matrix metalloproteinases, thereby reducing inflammation and fibrosis [11]. Tacrolimus, meanwhile, suppresses calcineurin-mediated T-cell activation, reducing immune-driven inflammation [12]. Given that morphea involves both immune dysregulation and fibroblast activation, targeting these inflammatory and fibrotic pathways concurrently may contribute to the favourable clinical response observed in this case.

Despite the favourable outcome, several limitations should be acknowledged. As a single-case report, the findings cannot be generalised and lack comparative analysis. Furthermore, because topical tacrolimus was administered concomitantly with excimer lamp therapy, the individual contribution of each treatment modality to the observed clinical response cannot be definitively determined. Additionally, the absence of long-term follow-up limits assessment of disease recurrence, which may occur due to the chronic and relapsing nature of morphea [13]. This consideration is particularly relevant, as retrospective data in adults with linear morphea have shown that although approximately 70% of patients achieve disease improvement or stabilisation following treatment, recurrence may occur in up to 40% of cases after treatment completion [14]. Further studies involving larger cohorts, controlled designs, and extended follow-up are warranted to validate these observations and establish standardised treatment approaches.

CONCLUSION

This case highlights the potential benefit of combining 308-nm excimer lamp therapy with topical tacrolimus in the management of linear morphea. Early intervention remains important to minimise disease progression and irreversible tissue damage. The combination of targeted UVB therapy and topical immunomodulation was associated with a favourable clinical response in this patient. Further studies involving larger cohorts, comparative designs, and extended follow-up are warranted to further evaluate the efficacy and long-term safety of this combined therapeutic approach.

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None.

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest.

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Rosacea and *Demodex* folliculitis Treated with 5% Permethrin and Low Dose Isotretinoin: A Case Report

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ABSTRACT: Demodicosis is a skin disease caused by mites of the genus *Demodex*. Some evidence suggests that these mites may contribute to flare-ups in patients with rosacea. We report a case of a 32-year-old healthy male who developed multiple, irregularly shaped, well-defined erythematous macules, papules, pustules and scaly plaques infested with *Demodex* mites over the zygomatic and centrofacial (glabellar) areas of the face. The patient was treated with permethrin 5% cream twice daily for two months, and low-dose oral isotretinoin at 10 mg/day for two months, resulting in significant clinical improvement. The combination of permethrin 5% cream and low-dose oral isotretinoin may be considered a potential treatment option for *Demodex* folliculitis on a background of rosacea.

Keywords: Demodicosis, Rosacea, Isotretinoin, Permethrin, *Demodex*

INTRODUCTION

Demodicosis is a cutaneous disorder caused by *Demodex* mites [1]. The pathogenesis of demodicosis is not completely understood. Proposed mechanisms include blockage of hair follicles and sebaceous ducts due to reactive epithelial hyperplasia and hyperkeratinization; bacterial transmission by *Demodex*; a foreign-body reaction to the mites or their waste products; delayed hypersensitivity to mite antigens; and an irritant reaction caused by fatty acids released by mite-derived lipase [2]. Increased *Demodex* density has been implicated in papulopustular rosacea, and the distinction between rosacea-like demodicosis and papulopustular rosacea remains an area of ongoing debate [3]. Although ivermectin is generally considered the treatment of choice for human demodicosis, other off-label therapies have also been used [4]. This case report aims to describe a case of *Demodex* folliculitis complicating papulopustular rosacea in a patient with a darker skin phototype, including its clinical and histopathological features and the observed response to topical permethrin and low-dose oral isotretinoin.

CASE PRESENTATION

A 32-year-old Filipino man presented with plaques on the zygomatic and centrofacial (glabellar) areas of the face.

Five years prior to consultation, he developed pruritic macules and papules over the zygomatic areas that gradually increased in size and number. The lesions were aggravated by sun exposure and progressed into multiple, well-defined, erythematous, plaques studded with pustules. He was initially diagnosed as seborrheic dermatitis by a private physician and treated with a topical corticosteroid and antifungal, with minimal improvement. Three years prior to consultation, the lesions recurred and extended to the glabellar area and periorbital rim (**Figure 1**), accompanied by scaling, dryness, and blepharitis. He received no further consultation or treatment until the persistence and progression of the lesions prompted consultation at our institution. Written informed consent was obtained for publication of the clinical details and photographs.

MANAGEMENT AND OUTCOME

The patient had Fitzpatrick skin phototype IV with persistent centrofacial redness, mild phyma, transient facial erythema, inflammatory papules and pustules, mild facial edema, and ocular involvement. Dermoscopy revealed matted telangiectasia, linear curved vessels, and white lines corresponding to *Demodex* tails (**Figure 2A**). Skin punch biopsy demonstrated numerous *Demodex* mites within the follicular infundibula, some surrounded by dense neutrophilic infiltrates

(microabscess). Enlarged sebaceous lobules with focal lysis and moderate perivascular and periadnexal lymphocytic infiltrates were also observed (**Figure 2B**), confirming the diagnosis of rosacea with *Demodex* folliculitis. Baseline laboratory investigations were performed prior to treatment and were within normal limits. The patient was treated with permethrin 5% cream twice daily and oral isotretinoin 10 mg/day (0.17 mg/kg/day) for two months, resulting in clinical

improvement, as documented by Quantificare® photographs obtained at baseline and after 2 months of treatment (**Figure 3A**). No adverse events were reported during the treatment period. The post-treatment images demonstrated reduced facial erythema, with a corresponding reduction in the extent and intensity of erythema on the Quantificare® erythema maps (**Figure 3B**). Isotretinoin was initially planned for a five-month course; however, the patient was lost to follow-up.



Figure 1. Clinical photograph showing multiple, irregularly shaped, well-defined erythematous macules, papules, and scaly plaques involving the forehead (glabella), periorbital rim, and bilateral zygomatic areas.

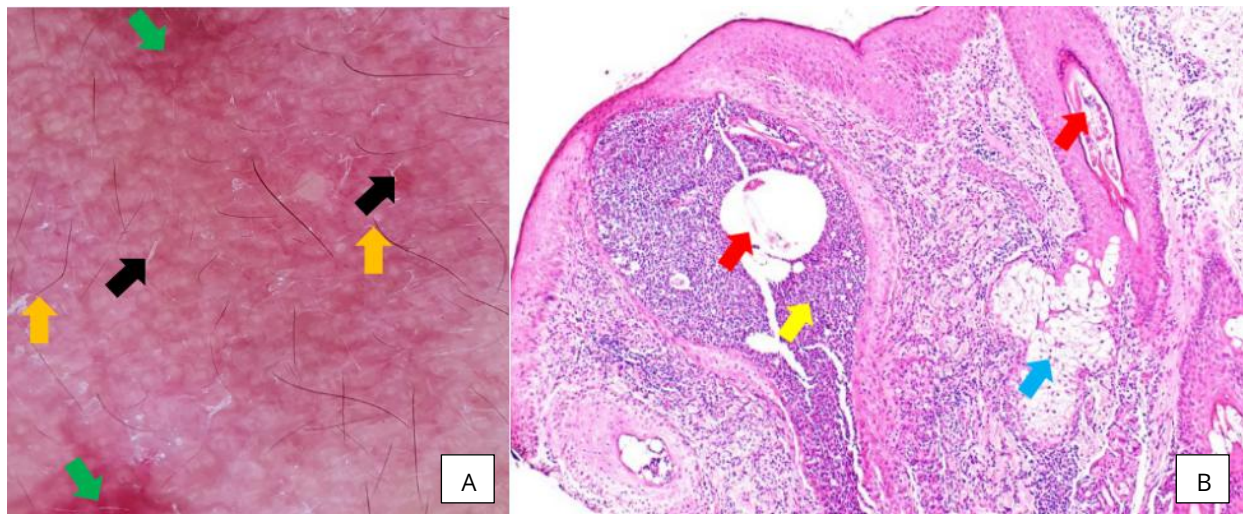


Figure 2. Diagnostic findings. **(A)** Dermoscopy showing a network of blood vessels. Specific features include dilated blood vessels (green arrows), white dots corresponding to dilated follicular infundibula (orange arrows), and white lines (*Demodex* tails) (black arrows); **(B)** Histopathological findings showing numerous *Demodex* mites within the follicular infundibula (red arrows), associated with dense neutrophilic infiltrates (yellow arrow), enlarged sebaceous lobules with focal lysis (blue arrow), and moderate perivascular and periadnexal lymphocytic infiltrates (H&E, ×400).

DISCUSSION

Demodicosis, or demodicidosis, is a cutaneous disorder caused by *Demodex* mites, which are common commensals of the pilosebaceous unit. Demodicosis has traditionally been classified into pityriasis folliculorum, rosacea-like demodicosis, and demodicosis gravis. Among these, rosacea-like demodicosis shares clinical features with papulopustular rosacea, and current evidence suggests that the two may represent phenotypes within the same disease spectrum rather than distinct disorders [3]. Although the relationship

between *Demodex* and rosacea remains controversial [1], increasing evidence supports its role in the pathogenesis and exacerbation of papulopustular rosacea. In the present case, the characteristic clinical features of rosacea, together with dermoscopic findings suggestive of *Demodex* infestation and histopathological demonstration of numerous intrafollicular mites with perifollicular inflammation, supported the diagnosis of rosacea complicated by *Demodex* folliculitis.

Demodicosis may be diagnosed by examining scales using 40% potassium hydroxide (KOH) or by standard skin surface biopsy (SSSB), which samples the

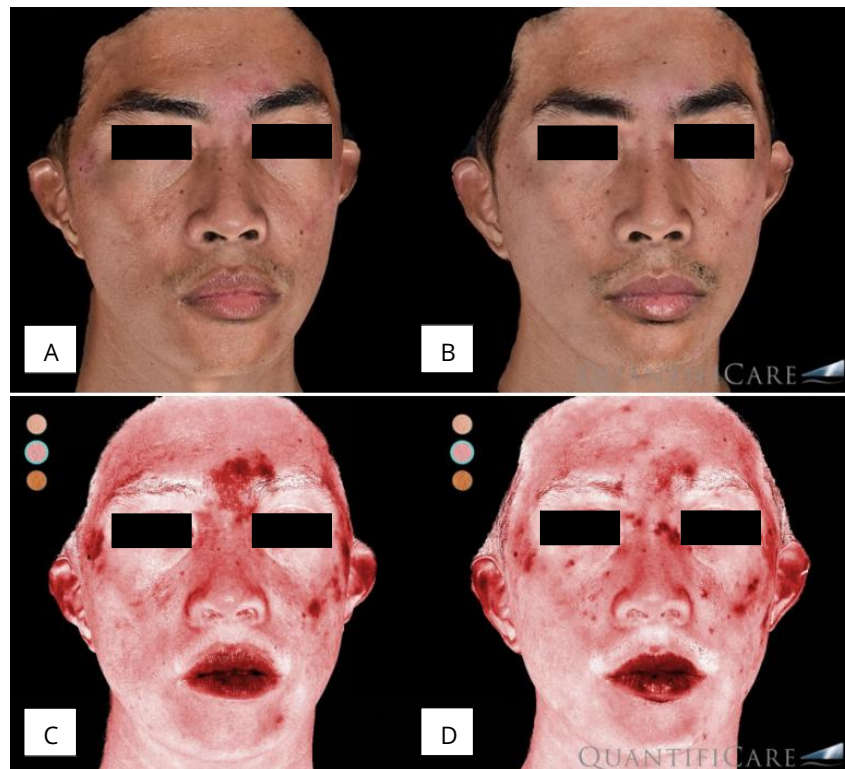


Figure 3. Quantificare® 3D photographic images obtained at baseline (A) clinical photographs; (C) erythema maps; and after two months of treatment (B) clinical photographs; (D) erythema maps. Photographs demonstrate clinical improvement, with reduced facial erythema and a corresponding reduction in the extent and intensity of erythema following treatment with topical permethrin 5% cream and low-dose oral isotretinoin.

superficial stratum corneum and follicular contents using cyanoacrylate glue [2]. Although the presence of ≥ 5 mites in a single low-power field on KOH examination or >5 mites/cm² on SSSB is considered significant, *Demodex* mites may also be found in healthy skin. Therefore, diagnosis should rely on increased mite density or extrafollicular localisation together with compatible clinical findings.

In the present case, KOH and SSSB were not performed due to limited availability. Dermoscopy and skin biopsy were chosen to further characterise the lesions and provide histopathological confirmation of the suspected diagnosis. Dermoscopy serves as a useful, non-invasive adjunct, with characteristic findings including white “*Demodex* tails” protruding from follicular openings and reticular or linear telangiectatic vessels [5]. Histopathological findings may vary but generally demonstrate *Demodex* mites within pilosebaceous units accompanied by perifollicular inflammatory infiltrates, with neutrophilic or granulomatous inflammation in more severe forms [2]. In our patient, the characteristic dermoscopic findings, together with skin biopsy demonstrating numerous intrafollicular *Demodex* mites associated with neutrophilic folliculitis and perifollicular inflammatory infiltrates, supported the diagnosis of *Demodex* folliculitis on a background of rosacea.

Treatment of demodicosis is supported by limited evidence [1]. Ivermectin, a purely acaricidal agent, is considered the treatment of choice for human

demodicosis and may be administered as a single oral dose of 0.2 mg/kg or topically as a 1% cream, with reported therapeutic effects [1,6]. Other off-label therapies, including topical permethrin and low-dose oral isotretinoin, have also been used for demodicosis and for the treatment of papules and pustules associated with rosacea and *Demodex* infestation [4]. In a randomised, double-blind, placebo-controlled study, permethrin 5% cream applied twice daily for two months was comparable to metronidazole 0.75% gel in improving erythema and papules and showed greater efficacy against *Demodex folliculorum* [7]. Permethrin 5% cream has also been reported to reduce *Demodex* mite burden and improve blepharitis [8]. Meanwhile, low-dose isotretinoin (10–20 mg administered one to five times weekly) has been reported to be effective and well tolerated in patients with mild-to-moderate papulopustular rosacea [9].

Greater benefit may be achieved when permethrin 5% cream is combined with other systemic and/or topical therapies [7]. In this case, clinical improvement was documented after two months of treatment with permethrin 5% cream and low-dose oral isotretinoin. A similar combination was reported by Paichitrojjana et al. in a patient with ivermectin-refractory demodicosis, who was treated with permethrin 1% cream and low-dose oral isotretinoin, with marked clinical improvement after eight weeks [10].

Our case adds to the limited literature describing this combination treatment in a Filipino

patient with Fitzpatrick skin phototype IV. The clinical improvement observed after two months suggests that combined topical permethrin and low-dose oral isotretinoin may represent a potential treatment option for *Demodex* folliculitis associated with rosacea in patients with darker Asian skin. Further studies are needed to determine the efficacy and safety of this approach in this population.

CONCLUSION

This case illustrates the clinical overlap between papulopustular rosacea and rosacea-like demodicosis, which may represent two phenotypes within the same disease spectrum. Dermoscopy may aid in the clinical diagnosis of *Demodex* folliculitis, while skin biopsy provides histopathological confirmation, particularly in cases complicating papulopustular rosacea. Treatment with topical 5% permethrin and low-dose oral isotretinoin was associated with clinical improvement during the treatment period, suggesting that this combination may represent a potential treatment option for selected patients with rosacea associated with *Demodex* infestation.

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None

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest.

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Structural Rhinoplasty for Congenital Aplasia of the Cartilaginous Nasal Septum: A Case Report

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ABSTRACT: Congenital aplasia of the cartilaginous nasal septum is an extremely rare condition. We present the case of a 22-year-old Filipina who sought aesthetic correction of a flattened nose. Physical examination revealed a short nose with a low dorsum, lack of tip projection, right alar notching, perialar flattening, a shortened columella, and an acute nasolabial angle. Cranial radiography showed no congenital craniomaxillofacial anomaly. The patient underwent open structural rhinoplasty with a septal extension graft (SEG) using polycaprolactone (PCL) mesh and dorsal augmentation using expanded polytetrafluoroethylene (ePTFE). Intraoperatively, complete absence of the cartilaginous nasal septum was confirmed. Postoperatively, the patient demonstrated satisfactory aesthetic and functional outcomes, with improved patient-reported quality-of-life scores. This case highlights the surgical management of congenital cartilaginous nasal septal aplasia using structural rhinoplasty with a PCL mesh SEG and reports the resulting aesthetic and patient-reported outcomes.

Keywords: Congenital aplasia, Cartilaginous nasal septum, Structural rhinoplasty, Septal extension graft, Case report

INTRODUCTION

Congenital aplasia of the cartilaginous nasal septum is an exceedingly rare craniofacial anomaly, with only a limited number of cases reported in the literature [1-3]. The nasal septum is composed of cartilaginous, bony, and membranous components that collectively provide structural support to the external nose, maintain dorsal projection, and contribute to nasal airflow and function. Absence of the cartilaginous septum may result in loss of nasal support, leading to deformities such as saddle-nose deformity, tip underprojection, and functional impairment [1].

Most septal defects encountered in clinical practice are acquired and may result from prior septal surgery, trauma, infection, inflammatory disease, chemical injury, or substance abuse [1]. In contrast, congenital defects of the nasal septum are exceptionally uncommon, occurring as isolated anomalies or in association with other craniofacial abnormalities. Diagnosis relies on careful clinical evaluation and may be

supplemented by imaging studies to assess associated craniofacial anomalies and guide surgical planning [4].

Reconstruction of congenital septal deficiencies remains challenging because of the need to restore both structural support and nasal aesthetics. Reported surgical approaches include the use of autologous cartilage or bone grafts [2], such as costal cartilage and iliac bone grafts, as well as allogenic and alloplastic materials for septal reconstruction and dorsal augmentation. The choice of reconstruction depends on the extent of the defect, available donor tissue, and surgeon preference. We present the case of a 22-year-old Filipina with congenital aplasia of the cartilaginous nasal septum who underwent structural rhinoplasty using polycaprolactone (PCL) mesh as a septal extension graft and expanded polytetrafluoroethylene (ePTFE) for dorsal augmentation. This report highlights the diagnosis and surgical management of this rare anomaly and contributes to the limited literature on reconstructive options for congenital absence of the cartilaginous nasal septum.

CASE PRESENTATION

A 22-year-old Filipina presented to our clinic seeking aesthetic correction of a flattened nose (**Figure 1**). Her medical history revealed no maxillofacial trauma, nasal or midfacial surgery, previous medical treatment for nasal problems, intranasal substance use, or recreational drug use. She was born via uncomplicated vaginal delivery. Her family reported that the nasal deformity had been present since birth and persisted throughout her growth. There was no family history of a similar condition.

General physical examination revealed a healthy-appearing adult in no apparent distress, with no evident craniofacial abnormalities. External nasal examination showed a short nose with supratip depression, a lack of tip projection, right alar notching, perialar flattening, vertical shortening of the columella, a low dorsum, and an acute nasolabial angle. Cranial x-ray showed no congenital anomalies in the craniomaxillofacial region (**Figure 2**). Computed

tomography (CT) was requested but was not performed because of financial constraints.

The patient completed two questionnaires to assess her quality of life in relation to her nasal condition. Her score on the Nasal Obstruction Symptom Evaluation (NOSE) scale was 0, indicating no self-reported nasal obstruction symptoms (**Supplementary Table S1**). Her score on the Rhinoplasty Outcomes Evaluation (ROE) questionnaire was 29.2%, indicating substantial dissatisfaction with her nasal appearance and rhinoplasty-related quality of life (**Supplementary Table S2**, See Supplementary). The NOSE and ROE score were scheduled for reassessment at the 6-month follow-up.

The preoperative diagnosis was overhanging ala, right alar notching, an underprojected nasal tip, and a low dorsum. Structural rhinoplasty using PCL mesh and dorsal augmentation with ePTFE through an open approach was planned. Informed consent for the publication of this case report and the accompanying clinical photographs was obtained from the patient.



Figure 1. Preoperative views: frontal (A), lateral (B,C), basal (D), and oblique (E,F).



Figure 2. Cranial X-rays: anteroposterior (A) and lateral (B) views.

MANAGEMENT AND OUTCOME

Preoperative preparation

The patient was placed in a supine position (Supplementary Figure S1), and intravenous sedation was administered. Standard aseptic and antiseptic preparation was performed, followed by sterile draping. Local anaesthesia was infiltrated. Vestibular hair removal and topical mupirocin application were performed. Conchal cartilage was harvested from the right ear via a posterior approach while preserving the perichondrium.

Surgical Procedure

Overhang correction was performed via sail excision of the right ala. An inverted-V columellar incision and bilateral marginal incisions were made. Intraoperative findings revealed a thin and weak right lower lateral cartilage (Figure 3A & 3B) and complete absence of the cartilaginous nasal septum (Figure 3C). Two pieces of PCL mesh were inserted to construct a septal extension graft (SEG) and secured to the anterior nasal spine and vomer (Figure 3D & 3E). The mesh was covered with conchal cartilage. Tip reconstruction and dorsal augmentation

with ePTFE were subsequently performed, followed by placement of supportive grafts (Figure 3F). Figure 4A & 4B illustrate the SEG design using PCL mesh and its attachment to the anterior nasal spine and vomer.

Post-procedure

The incisions were closed using Vicryl and nylon sutures, and a Denver splint was applied. The patient tolerated the procedure well. The postoperative course was uneventful, with no complications observed (Figure 5). The patient returned seven days after surgery for removal of the external splint and sutures (Figure 6). At the six-month follow-up, improvements in nasal projection, length, and contour were observed (Figure 7). The patient completed the NOSE and ROE questionnaires again postoperatively to assess her quality of life and satisfaction after surgery. The NOSE score remained at 0 before and after surgery, indicating no self-reported nasal obstruction symptoms (Supplementary Table S3). The ROE score improved from 29.2% preoperatively to 87.5% postoperatively, indicating improved patient satisfaction with the surgical outcomes (Supplementary Table S4).

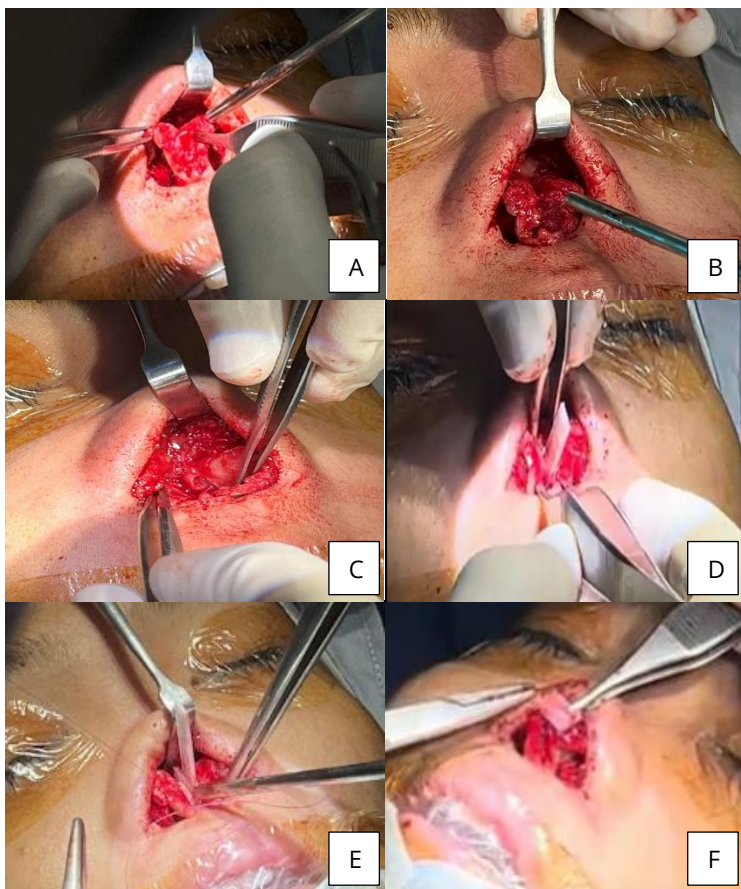


Figure 3. Intraoperative findings of thin and weak right lower lateral cartilage (A), exposure of the upper lateral cartilage (B), absence of the cartilaginous nasal septum (C), placement of PCL mesh as a SEG (D), fixation of PCL mesh using 5-0 polydioxanone (PDS) sutures (E), and placement of an onlay graft (F).

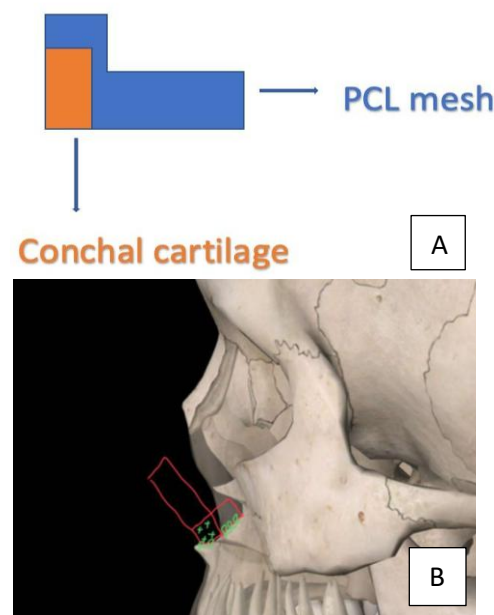


Figure 4. Schematic illustration of the SEG design using PCL mesh (A), and its attachment to the anterior nasal spine and vomer (B).

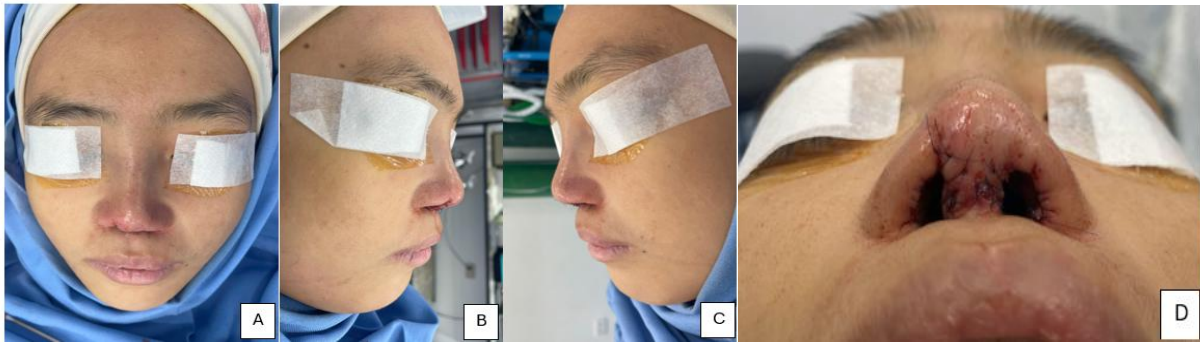


Figure 5. Immediate postoperative views: frontal (A), lateral (B,C), and basal (D).

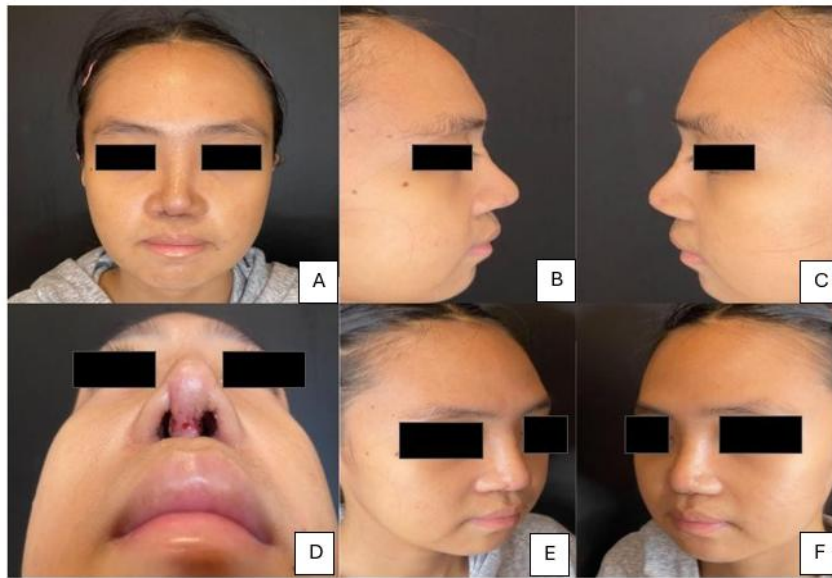


Figure 6. Removal of sutures: frontal (A), lateral (B,C), basal (D), and oblique (E,F) views.

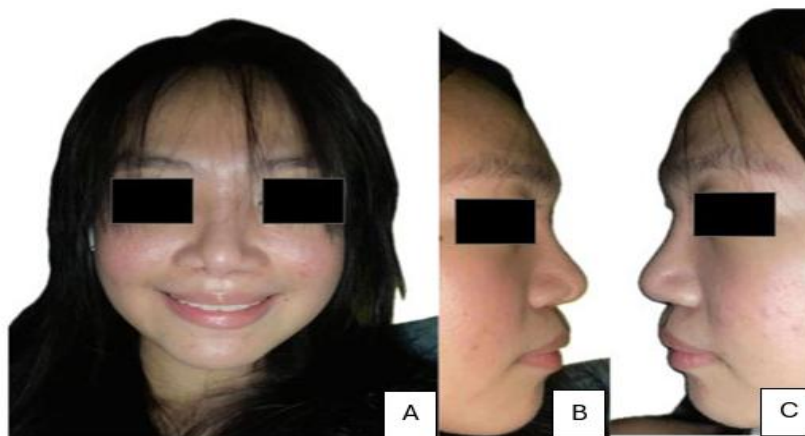


Figure 7. Six-month postoperative views: frontal (A) and lateral (B,C).

DISCUSSION

The nasal septum develops through a complex sequence of midfacial embryogenesis. Nasal development begins during the fourth week of gestation with the formation of the nasal placodes adjacent to the frontonasal prominence. By the fifth week, the medial and lateral nasal processes surround the developing nasal pits, which deepen to form the primitive nasal cavities. As the nasal cavities expand, the frontonasal process contributes to the formation of the nasal septum. By approximately the eighth week, the cartilaginous nasal

capsule surrounds the developing nasal cavity and subsequently undergoes partial endochondral ossification. The quadrangular septal cartilage and upper and lower lateral cartilages remain cartilaginous in the mature nose [1,5]. Disruption of this developmental process may result in a spectrum of congenital nasal abnormalities, ranging from partial nasal hypoplasia to absence of individual nasal structures or, in severe cases, complete arhinia [2].

Congenital defects of the nasal septum are particularly uncommon, and current knowledge is derived largely from isolated case reports and small case

series. Hammoud et al. noted that only a small number of cases involving the absence of the quadrangular cartilage had been reported and described an adult patient with an absent cartilaginous septum and bilateral middle turbinates [1]. Fijałkowska and Antoszewski likewise identified three patients with absence of the quadrangular septal cartilage among 13 patients with isolated nasal underdevelopment [2]. These reports underscore both the rarity and variable clinical expression of congenital septal aplasia.

The differential diagnosis includes congenital and acquired causes of septal deficiency and saddle-nose deformity. Acquired defects may occur following nasal surgery, trauma, cauterization or chemical injury, intranasal drug use, infection, and inflammatory or granulomatous disease [1,3]. In the present patient, the deformity had been noted since birth and persisted during growth, with no history of maxillofacial trauma, nasal or midface surgery, intranasal substance use, or treatment for a previous nasal disorder. No clinical or intraoperative findings suggested prior destructive or inflammatory disease. Binder syndrome was also considered because it may present with a flattened nasal profile; however, the patient's normal maxillary and nasal bone development and balanced midface profile were not consistent with the characteristic maxillary hypoplasia and midfacial retrusion described in Binder syndrome [2]. Taken together with the intraoperative finding of complete absence of the cartilaginous septum, these findings supported a congenital rather than acquired etiology.

Congenital absence of nasal structures may occur in isolation or alongside other congenital abnormalities. Abdulkadir et al. described congenital absence of the nasal septum in a newborn with multiple craniofacial and systemic anomalies, suggesting a possible syndromic association [6]. These findings highlight the importance of considering possible syndromic associations, particularly when additional craniofacial, developmental, or systemic abnormalities are present. In such cases, multidisciplinary assessment, including genetic evaluation when clinically indicated, may be considered. The present patient had no evident associated craniofacial abnormalities or family history of a similar condition. However, genetic testing was not performed; therefore, an underlying genetic association cannot be excluded.

Diagnosis relies on a detailed history and careful external and intranasal examination, supplemented by imaging when available. CT helps define the extent of septal absence, assess the bony septum and other internal nasal structures, identify associated craniofacial abnormalities, and guide surgical planning [1]. In the present case, cranial radiography showed no evident craniomaxillofacial anomaly. CT was requested but not performed because of financial constraints. In the absence of CT imaging, the patient's clinical history and physical examination findings guided the preoperative assessment and surgical planning, while the complete

absence of the cartilaginous nasal septum was confirmed intraoperatively. Although this represents a limitation of the preoperative evaluation, the case demonstrates how clinical findings can guide surgical planning when cross-sectional imaging is unavailable.

Management is usually individualized and ranges from observation to substantial structural reconstruction, depending on symptoms, anatomy, degree of deformity, functional impairment, patient age, aesthetic concerns, and available reconstructive tissue. Because congenital cartilaginous septal aplasia is exceptionally rare, there is no standardized reconstructive protocol. Previously reported surgical cases have used autologous cartilage or bone grafts for nasal reconstruction [2,3]. In contrast, Hammoud et al. described a patient who was managed conservatively for recurrent epistaxis, her main complaint, as she had no breathing difficulties and was satisfied with her nasal appearance [1].

Structural rhinoplasty aims to reinforce and reposition existing nasal structures to achieve stable aesthetic and functional correction, an approach particularly relevant in Southeast Asian noses, in which the lower lateral cartilages may provide limited structural support [7]. When substantial tip repositioning is required, a SEG can provide a stable framework for controlling projection, rotation, and length, as illustrated in **Figure 8**, alongside the spreader graft [7]. In the present case, complete absence of the cartilaginous septum eliminated the usual source of septal cartilage for the SEG, and the patient also required substantial tip projection and elongation. Therefore, two pieces of PCL mesh were used to construct the SEG, secured to the anterior nasal spine and vomer, and covered with conchal cartilage. The thin and weak right lower lateral cartilage was additionally reinforced with an alar conchal batten graft, while ePTFE was used for dorsal augmentation to harmonise the dorsum with the newly projected tip.

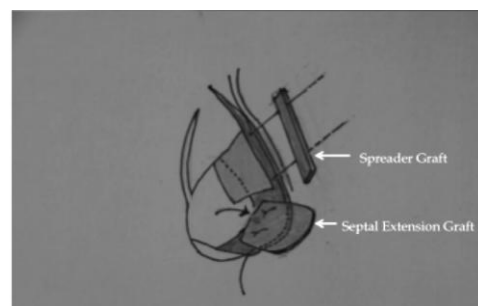


Figure 8. Schematic illustration of two structural support grafts used in rhinoplasty: the spreader graft and the SEG [7].

PCL mesh is a bioabsorbable scaffold that can provide temporary structural support while permitting tissue ingrowth. Its porous architecture allows ingrowth of chondroblastic and mesenchymal tissue, and coverage with cartilage has been recommended to reduce the risk of mucosal erosion and provide additional long-term support as the material is absorbed

[8]. PCL has been described in structural rhinoplasty when septal cartilage is small, weak, or inadequate for reconstruction [8]. In this case, this principle was extended to a patient in whom the cartilaginous septum was congenitally absent. At six months, improvements in nasal projection, length, and contour were observed, with no postoperative complications reported. Nevertheless, the relatively short follow-up period is an important limitation, particularly when evaluating a bioabsorbable implant, and longer follow-up is necessary to assess maintenance of structural support after progressive PCL degradation.

Patient-reported outcome measures provide an important complement to the surgeon's assessment after rhinoplasty because aesthetic procedures are strongly influenced by patient perception, expectations, and quality of life [9,10]. The NOSE scale is a validated patient-reported measure of nasal obstruction and has been used to evaluate functional outcomes following septorhinoplasty [11]. The patient's NOSE score remained at 0 before and six months after surgery, indicating no patient-reported nasal obstruction following the reconstructive procedure. The ROE questionnaire evaluates aesthetic satisfaction, nasal function, and psychosocial aspects of rhinoplasty outcomes [9,12]. In the present case, the ROE score increased from 29.2% preoperatively to 87.5% at six months, representing a 58.3-percentage-point increase and indicating greater patient-reported satisfaction after surgery.

CONCLUSION

Congenital aplasia of the cartilaginous nasal septum is an exceptionally rare anomaly that requires careful clinical assessment and individualized surgical planning. This case adds to the limited literature by documenting structural rhinoplasty using PCL mesh as a SEG in the absence of native septal cartilage. At six months, improvements in nasal projection, length, and contour were observed, with preserved self-reported nasal function. The substantial improvement in the ROE score further demonstrated increased patient-reported satisfaction following reconstruction. These findings suggest that PCL mesh may be a useful reconstructive option in carefully selected patients with inadequate or absent septal cartilage. However, a single case with six months of follow-up cannot establish long-term durability or comparative effectiveness. Longer follow-up is necessary to evaluate the maintenance of structural support following progressive PCL degradation.

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CONFLICT OF INTEREST

The authors declare no financial or personal conflicts of interest that could have influenced the work reported in this case report.

Supplementary Material

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Acquired Brachial Cutaneous Dyschromatosis with Coexisting Pigmentary Disorders: A Report of Two Cases with Clinicopathologic Findings

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ABSTRACT: Acquired brachial cutaneous dyschromatosis (ABCD) is an uncommon pigmentary disorder characterised by reticulated hyperpigmented and hypopigmented macules and patches on the dorsal forearms. It predominantly affects postmenopausal women and has been associated with chronic ultraviolet (UV) exposure and, in some reports, the use of angiotensin-converting enzyme inhibitors (ACEis). We report two cases of ABCD in the Philippines, highlighting its coexistence with facial iatrogenic dyschromia in one patient and idiopathic guttate hypomelanosis (IGH) in the other, raising the possibility of shared contributory factors despite these conditions representing distinct disease entities. Two 60-year-old Filipino women with Fitzpatrick skin phototype (FSP) IV presented with progressive reticulated hyperpigmented and hypopigmented macules and patches on the bilateral dorsal forearms. Histopathological evaluation of the forearm lesions in both patients demonstrated marked basal layer hyperpigmentation, sparse superficial perivascular lymphocytic infiltrates, and prominent solar elastosis, consistent with ABCD and chronic UV-induced photodamage. In Case 1, Melan-A immunohistochemistry of the forearm lesion demonstrated increased melanocyte density with an irregular distribution along the basal layer, supporting melanocytic hyperactivity. Concomitant facial pigmentary changes were consistent with iatrogenic dyschromia. In Case 2, concomitant discrete hypopigmented macules on the lower extremities were clinically and histopathologically consistent with IGH. These cases highlight the importance of correlating clinical, histopathological, and immunohistochemical findings in the evaluation of ABCD and provide further clinical observations supporting its proposed etiopathogenesis and possible association with concomitant pigmentary disorders. Management may focus on melanogenesis-modulating therapy and strict photoprotection to address pigmentary changes and minimise further pigmentary alterations.

Keywords: Acquired brachial cutaneous dyschromatosis, Dyschromia, Cutaneous pigmentary disorders, Idiopathic guttate hypomelanosis, Facial dyschromia

INTRODUCTION

Acquired brachial cutaneous dyschromatosis (ABCD) is an uncommon pigmentary disorder characterised by asymptomatic, reticulated hyperpigmented and hypopigmented macules on the dorsal forearms, most often observed in postmenopausal women [1,2]. ABCD has been associated with chronic sun exposure and, in several reports, angiotensin-converting enzyme inhibitor (ACEi) use [1]. Its distribution on the extensor upper arms, which are frequently exposed to ultraviolet (UV) radiation, may be consistent with a photoinduced mechanism. The proposed association between ABCD and hypertension or antihypertensive therapy may reflect the high prevalence of hypertension in middle-

aged and postmenopausal individuals, along with the common use of these medications in this population [1]. Although ACEis are known to cause various cutaneous adverse reactions such as photosensitivity, they have not previously been implicated in causing pigmentary disorders [3].

Histopathologically, ABCD is characterised by an atrophic epidermis, basal layer hyperpigmentation, telangiectases, and prominent solar elastosis [1]. UV radiation induces keratinocyte and melanocyte injury, resulting in dysregulated melanin synthesis and transfer, which may be associated with histological findings of basal layer hyperpigmentation, epidermal thinning, superficial telangiectases, and solar elastosis. We report two cases of ABCD in the Philippines and document its

coexistence with facial iatrogenic dyschromia in one patient and with idiopathic guttate hypomelanosis (IGH) in another. Although ABCD, IGH, and depigmenting agents-induced dyschromia are pathogenetically distinct entities, their coexistence in our patients raises the possibility of shared contributory factors, including chronic photodamage. This case report aims to contribute to the limited literature on ABCD by highlighting its clinical and histopathological features that may aid in its recognition and differentiation from other pigmentary disorders with similar presentations, thereby supporting accurate diagnosis and appropriate patient management. Furthermore, given the challenges in treating ABCD and the lack of established optimal management strategies, this report aims to provide a basis for further investigation into its etiopathogenesis, potential contributing factors, and therapeutic approaches.

CASE PRESENTATION

Case 1: A 60-year-old Filipino woman with Fitzpatrick skin phototype (FSP) IV presented with a 20-year history of progressive hyperpigmentation over both malar areas. She reported intermittent use of various over-the-counter depigmenting creams containing tretinoin (0.25%) and hydroquinone (2%). Within 2–3 days of application, she developed mild erythema without associated stinging, burning, pruritus, or scaling. The reaction was observed shortly after application and was self-limited but was not consistently observed with each

depigmenting cream used by the patient. Despite continued use over several years, the malar hyperpigmentation showed minimal improvement, and she subsequently developed multiple irregularly shaped, non-scaly hypopigmented macules and patches on both malar areas (**Figure 1**). One year prior to consultation, she noted the development of multiple irregular, geometric tan-to-brown patches interspersed with hypopigmented macules and scattered telangiectatic vessels on the bilateral dorsal forearms (**Figure 2**). The lesions gradually progressed with chronic sun exposure. She had no history of hypertension, denied use of ACEis, and reported no recent intake of phototoxic medications. No similar lesions were reported in her family.

Histopathological examination of a 4-mm punch biopsy from the facial lesion demonstrated epidermal thinning, basal layer hyperpigmentation, and sparse superficial perivascular lymphocytic infiltrates (**Figure 3A**), findings consistent with iatrogenic dyschromia. In contrast, the forearm biopsy revealed marked basal layer hyperpigmentation, an ill-defined grenz zone, prominent solar elastosis, pigment-laden macrophages, hemosiderin deposition, and fibrillar collagen changes (**Figure 3B**), consistent with ABCD. Immunohistochemical staining with Melan-A of the facial lesion demonstrated irregularly-shaped and apoptotic melanocytes (**Figure 3C**), whereas the forearm specimen showed epidermal melanocytosis consisting of irregularly-shaped melanocytes along the basal layer and increased melanin in the basal cell layer (**Figure 3D**).



Figure 1. Facial iatrogenic dyschromia showing multiple hypopigmented macules and telangiectasias against a background of hyperpigmented patches over the malar areas.



Figure 2. Reticulated hyperpigmented and hypopigmented macules and patches over the bilateral dorsal forearms, consistent with ABCD.

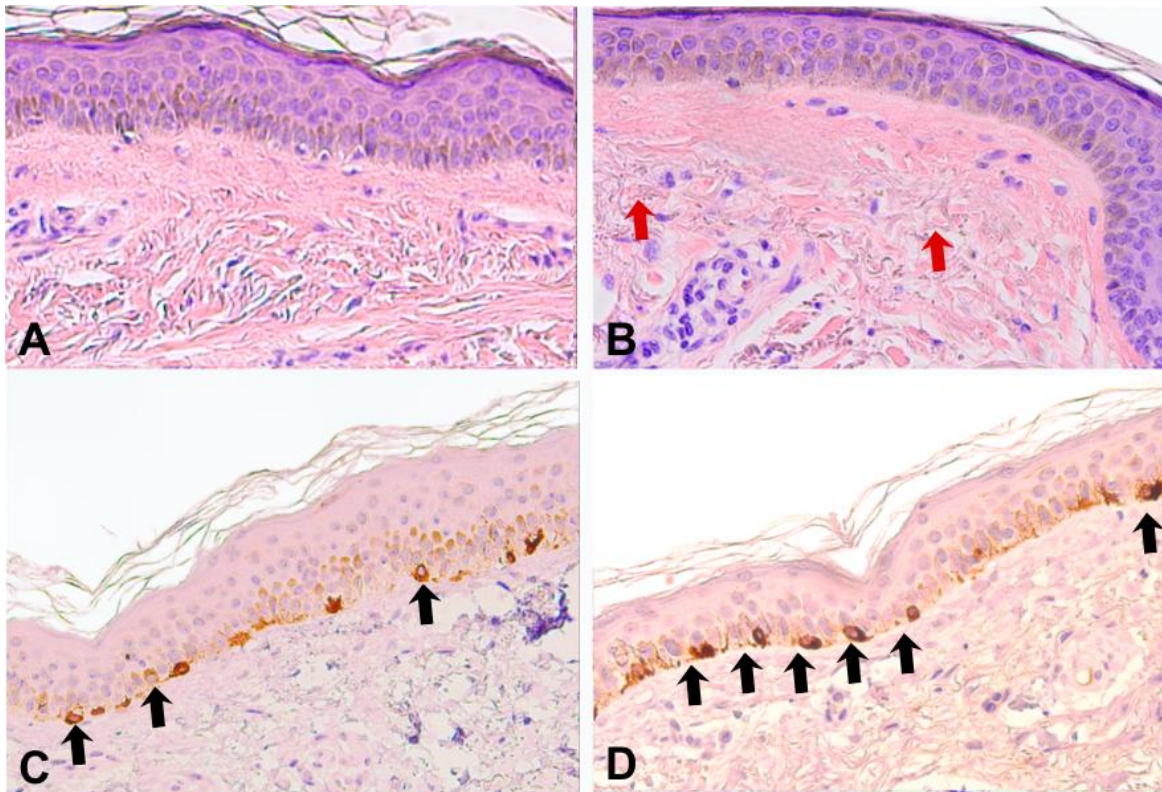


Figure 3. (A) Facial iatrogenic dyschromia showing epidermal atrophy and basal layer hyperpigmentation and sparse superficial perivascular lymphocytic infiltrates (H&E, $\times 400$). (B) ABCD of the dorsal forearm showing epidermal atrophy, basal layer hyperpigmentation, and prominent solar elastosis (red arrow) (H&E, $\times 400$). (C) Melan-A immunostaining of the facial lesion demonstrating irregularly-shaped and apoptotic melanocytes (black arrow) ($\times 400$). (D) Melan-A immunostaining of the forearm lesion highlighting epidermal melanocytosis, with irregularly shaped melanocytes and increased melanin in the basal layer (black arrow) ($\times 400$).

Case 2: A 60-year-old Filipino woman with FSP IV, presented with 2-year history of gradual development of hypopigmented and depigmented macules involving the lower extremities (**Figure 4**). Over time, multiple, reticulated, brown patches interspersed with hypopigmented macules also became apparent on the bilateral dorsal forearms (**Figure 5**). The lesions were asymptomatic and progressively more noticeable. The patient denied any history of topical hypopigmenting agent use, including hydroquinone, retinoids, or other depigmenting creams, and reported no prior cosmetic procedures. There was no history of hypertension or intake of ACEis, and no recent use of phototoxic medications. The patient reported no significant history of excessive sun exposure and denied any family history of similar pigmentary disorders.

Histopathological examination using a 4mm punch biopsy of the hypopigmented macule on the thigh revealed epidermal atrophy with basal layer hyperpigmentation with skipped areas of retained melanin in the basal layer alternating with large areas of melanin loss, and a sparse perivascular lymphocytic infiltrate, findings consistent with IGH (**Figure 6A**). In contrast, the forearm lesion demonstrated epidermal acanthosis with basal layer hyperpigmentation, prominent solar elastosis, and a sparse perivascular lymphocytic infiltrate, consistent with ABCD (**Figure 6B**). Although Melan-A immunostaining was not performed

for Case 2 due to financial constraints, the H&E findings supported the diagnosis of ABCD in the appropriate clinical context.

MANAGEMENT AND OUTCOME

Both patients were initiated on a topical serum (Neotone®) containing niacinamide, trimethoxybenzyl acetylsinapate, licorice root extract, and diacetyl boldine, applied twice daily, with the aim of addressing pigmentary changes. They were also counselled on strict daily broad-spectrum photoprotection and advised to avoid potential irritant topical agents and bleaching agents, particularly hydroquinone. Neither patient showed clinical improvement after six months of Neotone serum use, resulting in limited patient satisfaction due to the lack of visible response.

Subsequent management would emphasize continued photoprotection and individual treatment strategies. Potential options include melanogenesis-modulating agents, such as topical tacrolimus or bimatoprost, and targeted phototherapy for focal hypopigmented areas, although their efficacy and safety in ABCD have yet to be established. Procedures targeting dermal photodamage, including microneedling, chemical peels, and mild ablative resurfacing, may also be cautiously considered.



Figure 4. Multiple hypopigmented to depigmented macules on the lower extremities, consistent with IGH.



Figure 5. Reticulated hyperpigmented and hypopigmented macules and patches over the bilateral dorsal forearms, consistent with ABCD.

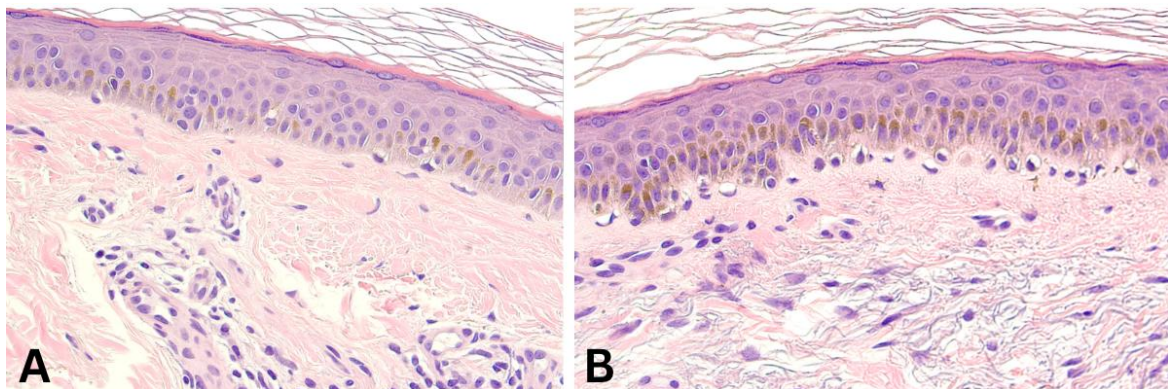


Figure 6. (A) IGH showing epidermal atrophy and basal layer hyperpigmentation with skipped areas of retained melanin (H&E, ×400). (B) ABCD of the dorsal forearm showing epidermal atrophy, basal layer hyperpigmentation, and prominent solar elastosis in the dermis (H&E, ×400).

DISCUSSION

ABCD is characterised by asymptomatic, irregular grey-brown patches with geographic borders, sometimes accompanied by hypopigmented, slightly atrophic macules, most commonly on the bilateral forearms. We present two cases in which ABCD coexisted with facial iatrogenic dyschromia in Case 1 and IGH on the lower extremities in Case 2. In Case 1, facial iatrogenic dyschromia was associated with the use of topical skin-lightening agents. Histopathological findings may include basal layer hyperpigmentation, dermal pigment incontinence with melanophages, and mild perivascular lymphocytic infiltrates; epidermal atrophy or vacuolar degeneration may also be present, depending on the causative agent [4]. ABCD, on the other hand, shows focal increased pigmentation of the epidermal keratinocytes against a background of solar elastosis in the dermis [5]. Melan-A staining in facial iatrogenic dyschromia revealed an irregular but patchy pattern, which may correlate with melanocytopenia [4], compared with melanocytosis or increased melanocyte density [5] in ABCD.

In case 2, IGH is a distinct hypopigmentary disorder characterised clinically by multiple, small, sharply demarcated, round to oval, hypopigmented to depigmented, porcelain-white macules on sun-exposed extremities [6]. This condition may be associated with senile degeneration, UV exposure, genetic factors, local trauma, autoimmune factors, and local inhibition of melanogenesis [7]. Histopathological findings include an atrophic epidermis with effacement of the rete ridges, a thick basket-weave orthokeratotic stratum corneum, and focal areas of retained melanin, which are relatively specific histopathological features of IGH [8]. These findings contrast with the increased melanin density in the basal layer and prominent solar elastosis in the papillary dermis of ABCD [5].

There is currently no established standard treatment for ABCD. Management primarily involves a multimodal therapeutic approach in conjunction with strict photoprotection [9]. Broad-spectrum sunscreen use, photoprotective clothing, and behavioral sun avoidance are recommended as first-line measures. In these cases, we used a topical serum formulation

(Neotone®), which has been investigated for other pigmentary disorders such as melasma, with good results [10,11]. However, no visible improvement was observed in our patients for these cases at the six-month follow-up. Alternative therapeutic approaches need to be explored.

Therapies aimed at melanocyte modulation, including topical tacrolimus, bimatoprost, or targeted phototherapy such as excimer laser treatment, may be considered for focal hypopigmented areas. These therapies have been used to treat hypopigmentation in other pigmentary disorders, such as IGH [12], and could potentially be explored for focal hypopigmented areas of ABCD. Topical retinoids [1], including tretinoin and other vitamin A derivatives, may improve epidermal turnover and contribute to dermal remodelling, particularly in the setting of photodamage. Procedural interventions targeting dermal photodamage, such as microneedling, chemical peels, or mild ablative resurfacing [1], may be considered cautiously in selected cases, particularly where solar elastosis is prominent. Adjunctive topical therapies, such as antioxidants including vitamin C [10], may theoretically help mitigate oxidative stress.

CONCLUSION

These cases aim to highlight the clinical and histopathological features of ABCD to facilitate its recognition and differentiation from other pigmentary disorders with similar clinical presentations. They also contribute to the limited literature on ABCD and may provide a basis for further research into its underlying pathogenesis, potential risk factors, and therapeutic approaches. The concomitant presence of iatrogenic dyschromia and IGH illustrates the diverse pigmentary changes that may occur in chronically sun-exposed skin. These coexisting disorders may reflect different melanocyte responses associated with chronic UV exposure and other contributing factors. Clinicians should maintain a broad differential diagnosis when evaluating ABCD in chronically sun-exposed skin and avoid attributing all pigmentary changes to a single process. Accurate recognition of these distinct entities is important to prevent misdiagnosis and inappropriate pigment-targeted therapy. Management should prioritise sustained photoprotection, with treatment strategies tailored to the underlying pigmentary disorder.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest related to the publication of this case report. Informed consent was

obtained from the patients for publication of the case report, including the clinical photographs.

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Circumscribed Juvenile Pityriasis Rubra Pilaris in a 4-Year-Old Filipino Female Treated with Acitretin: A Case Report

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ABSTRACT: Pityriasis rubra pilaris (PRP) Type IV, the circumscribed juvenile form, is a rare, chronic papulosquamous disorder in children. It can be misdiagnosed as paediatric psoriasis because both share erythematous plaques and hyperkeratotic lesions. PRP Type IV typically presents with well-demarcated, scaly, reddish-orange plaques on the elbows, knees, hands, and feet. Orange-hued palmoplantar keratoderma may also accompany the condition. Diagnosis relies on clinicopathological correlation, with biopsy typically revealing hyperkeratosis and a checkerboard pattern of alternating parakeratosis and orthokeratosis. Management is challenging because no standardised treatment guidelines exist, although combined topical and systemic therapy may be effective. In this report, a 4-year-old girl presented with a 2-year history of persistent pruritic plaques. She was treated with low-dose oral acitretin, topical emollients, corticosteroids, and vitamin D analogues, resulting in marked clinical improvement and good treatment tolerance. This case highlights the importance of accurate diagnosis and demonstrates that early, appropriately tailored therapy can achieve favourable outcomes and improve quality of life in paediatric patients with PRP Type IV.

Keywords: Acitretin, Pityriasis rubra pilaris, PRP, Treatment, Type IV

INTRODUCTION

Pityriasis rubra pilaris (PRP) is a rare, chronic papulosquamous disease of keratinisation. Its aetiology remains unknown [1]. The incidence is estimated at 1 in 5000, with an equal male-to-female ratio [2]. PRP is classified into six types, each characterised by distinct clinical features, age of onset, and disease progression [1]. PRP Type IV, the circumscribed juvenile form, usually occurs in prepubertal children. Its primary characteristics include hyperkeratotic perifollicular papules that develop into sharply defined erythematous plaques, usually on the elbows, knees, and the dorsal aspects of the hands and feet. Orange-hued palmoplantar keratoderma often accompanies the condition [1]. Because this dermatosis is rare, with limited evidence from clinical studies and a lack of clinical practice guidelines, treatment remains challenging. This report describes the clinical, histopathological, and therapeutic features of PRP Type IV in a 4-year-old girl, as well as the clinical response to a combined topical and systemic treatment approach.

CASE PRESENTATION

We report the case of a 4-year-old Filipino girl with a 2-year history of persistent, very itchy, scaly, reddish-orange-colored plaques on the palms, soles, and both knees. The patient consulted a private physician with unrecalled diagnosis and was only given moisturising lotion, with no other treatment. Review of systems was unremarkable. No similar cases were reported in the family. The parents are non-consanguineous. Dermatological examination revealed multiple, well-defined, irregularly shaped, erythematous, scaly plaques and patches on the hands, feet, and knees, some with excoriations (**Figure 1**). Hair and nail examinations were normal. Systemic examination was unremarkable, and laboratory examinations including complete blood count, liver enzymes, serum chemistries, and chest radiograph were all within normal limits.

A 4-mm skin punch biopsy was taken from the erythematous plaque on the right sole. Histopathological examination with hematoxylin and eosin (H&E) staining

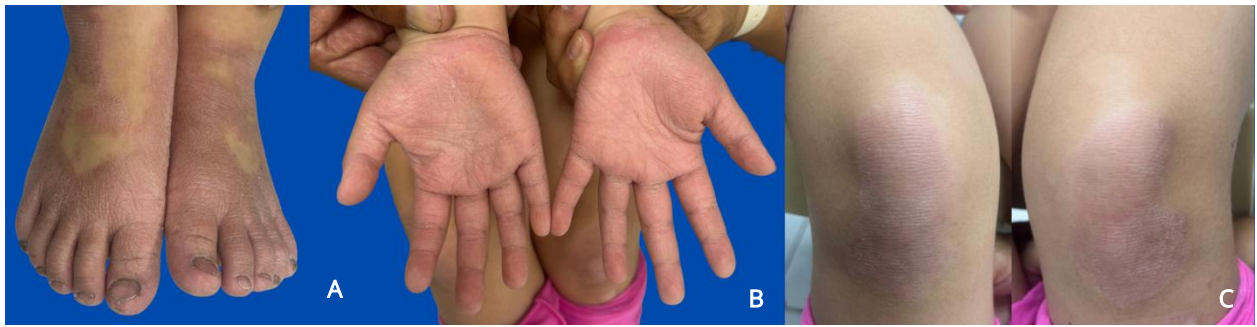


Figure 1. Erythematous, scaly plaques with follicular accentuation on the feet (A), hands (B), and knees (C) and diffuse symmetrical orange, waxy palmoplantar keratoderma with fissures.

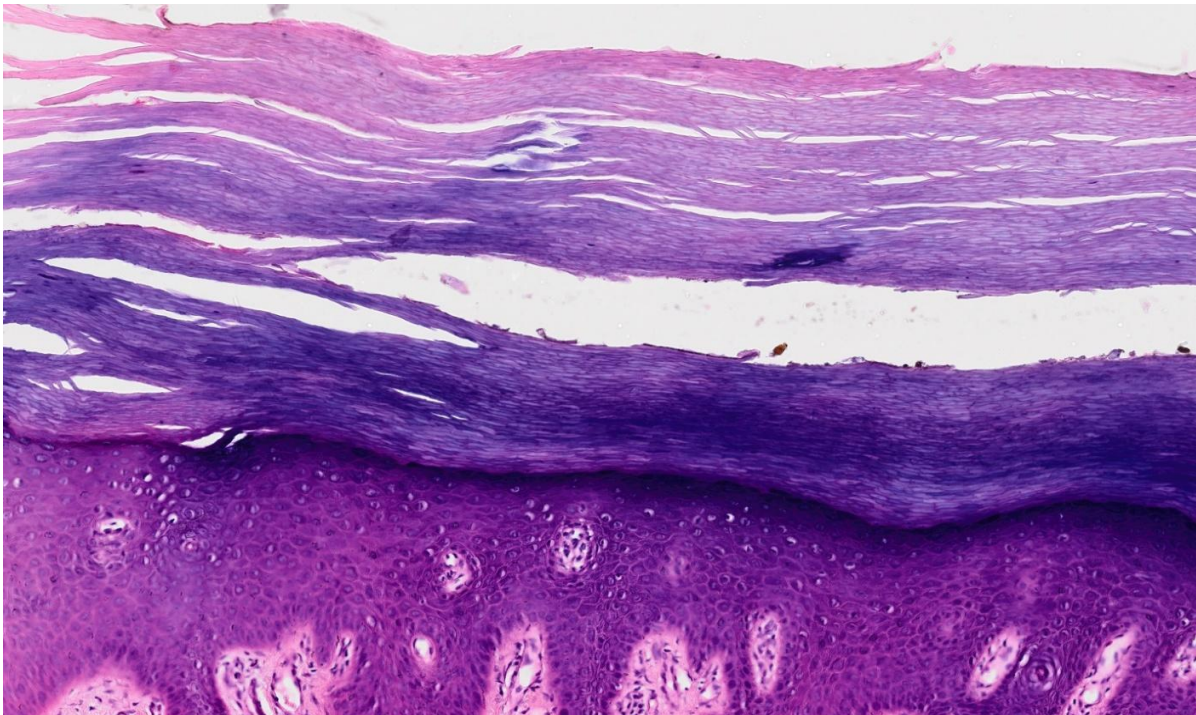


Figure 2. H&E staining of a skin punch biopsy specimen obtained from the right sole shows prominent acanthosis of the epidermis, hyperkeratosis, and alternating vertical and horizontal parakeratosis of the stratum corneum in a checkerboard pattern.

revealed hyperkeratosis and alternating vertical and horizontal parakeratosis in a checkerboard pattern in the stratum corneum. There was prominent acanthosis of the epidermis. The dermis showed a mild, superficial perivascular lymphocytic infiltrate (**Figure 2**). Based on the aforementioned clinicopathological findings, the diagnosis of PRP (Type IV) was established. Written informed consent for publication was obtained from the patient's legal guardian.

MANAGEMENT AND OUTCOME

The patient was started on acitretin 10 mg once daily (0.43 mg/kg/day), together with 30% urea lotion and clobetasol propionate 0.05% ointment applied twice daily for 2 weeks. After 2 weeks, clobetasol propionate was changed to calcipotriol, while the other medications were continued. The initial treatment plan was to administer acitretin for 3–6 months, depending on the clinical response. After 12 weeks of once-daily acitretin at 10 mg, complete resolution of the skin lesions on the bilateral

knees, hands, and feet was observed (**Figure 3**). The acitretin dose was subsequently reduced to 10 mg every other day. However, the patient took the medication for only 2 weeks before being lost to follow-up. After discontinuing treatment for 2 months, the patient returned with recurrence of skin-coloured to yellowish, waxy plaques on the palms and soles.

Following the relapse, acitretin was reintroduced at 10 mg once daily, together with topical clobetasol propionate ointment. The lesions again responded favourably following treatment reintroduction. Appropriate laboratory monitoring was performed throughout acitretin therapy, and no treatment-related adverse effects were observed. The total duration of treatment was 16 weeks, comprising 12 weeks of once-daily acitretin, 2 weeks of alternate-day dosing and 2 weeks of once-daily treatment following reintroduction. Treatment was interrupted by a period of approximately 2 months, during which the patient was lost to follow-up. The patient attended six follow-up visits in total.

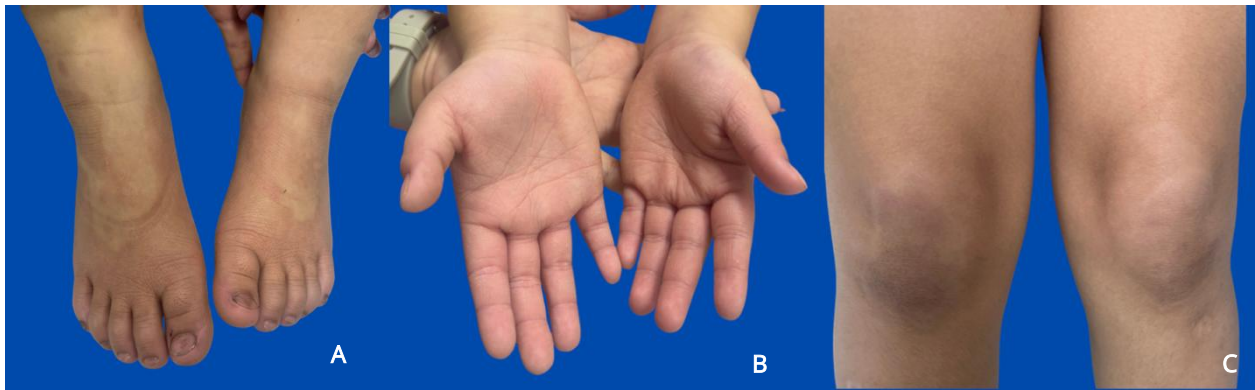


Figure 3. After 12 weeks of acitretin treatment showing complete flattening of the lesions on the feet (A), palms (B), and knees (C).

DISCUSSION

PRP is a rare inflammatory papulosquamous dermatosis. Griffiths originally classified it into five types. Later, a sixth category (HIV-associated) was added [1]. The exact aetiology and pathogenesis remain unclear. Several factors may be associated, such as heredity, endocrine dysfunction, vitamin A deficiency, autoimmune diseases, infections, trauma, and vaccination [3]. Most PRP types have generalised skin lesions, except Type IV, where the lesions are typically localised to the elbows, knees, palms, and soles [4]. This is the most common PRP type in children, comprising about 25% of paediatric cases. It is characterised by well-demarcated, hyperkeratotic erythematous plaques on the elbows and knees, resembling localised psoriasis. Palmoplantar keratoderma is characteristic of Type IV PRP but may also be absent [1].

PRP can be differentiated from psoriasis by the absence of characteristic silvery scales and the relative lack of scalp and nail involvement, which are common features of psoriasis. Dermoscopy further aids in distinguishing the two conditions. PRP typically demonstrates focal yellow-orange structureless areas with fine whitish scales, whereas psoriasis characteristically shows regularly distributed red dots and diffuse white scales on a pink to light red background. In the case presented, the presence of palmoplantar keratoderma along with erythematous plaques on the body initially led us to consider a diagnosis of paediatric psoriasis. A biopsy showing alternating parakeratosis and orthokeratosis in a checkerboard pattern supported the diagnosis of PRP. Other histopathologic features include hyperkeratosis and focal acantholytic dyskeratosis [1]. This underscores the diagnostic challenge of PRP, which can mimic other benign inflammatory dermatoses.

Due to the heterogeneity of the disease, there is no single standardised treatment approach, and various therapies may need to be considered to achieve adequate disease control. Currently, paediatric patients with PRP may be managed with topical treatment alone, particularly in less severe disease, while oral retinoids are widely accepted as the first-line option for systemic therapy [5]. Topical treatment includes medium- to high-potency corticosteroids, keratolytics, emollients, and

vitamin D analogues (e.g., calcipotriol) [1,2]. Combining topical and systemic therapies can be effective in managing hyperkeratosis and palmoplantar keratoderma [2].

In our patient, a combination of systemic acitretin and topical therapies was selected to target both the underlying disorder of keratinisation and the localised hyperkeratotic inflammation. Marked clinical improvement was observed during treatment. A relatively low dose was used to achieve disease control while limiting potential adverse effects associated with long-term retinoid therapy in children. Topical clobetasol propionate was initially prescribed to rapidly suppress inflammation and improve symptomatic hyperkeratotic plaques. Because prolonged use of superpotent topical corticosteroids is associated with cutaneous adverse effects, clobetasol was limited to 2 weeks and subsequently replaced with calcipotriol for maintenance therapy. Emollients, mild soap, and 30% urea lotion were included to restore the skin barrier, reduce hyperkeratosis, and improve scale removal.

Clinical improvement in PRP is generally reported within 3–6 months of oral retinoid therapy [6]. A 2024 scoping review reported that, among 32 patients treated with acitretin, 59.4% achieved a significant response, 25.0% achieved a partial response, and 3.1% showed no response. These findings support the potential role of oral retinoids in the management of PRP [7]. The recommended oral acitretin dosage is 0.5 to 0.75 mg/kg/day [2]. In our patient, a relatively low dose of acitretin was used, which was associated with complete clinical remission when combined with adjunctive topical therapy, without treatment-related adverse effects. High doses of retinoids are often poorly tolerated because of frequent mucocutaneous adverse effects. The most common side effects of retinoids include skin dryness and chapped lips. Elevated lipid levels, elevated liver enzymes, and visual or skeletal changes can also occur. In children, retinoids may cause premature epiphyseal closure, especially in prepubertal patients receiving high doses [2]. These safety considerations may be particularly relevant when treating paediatric patients with PRP, and lower doses may potentially improve treatment tolerability and adherence.

A notable aspect of this case is the documented disease recurrence following treatment interruption due

to financial constraints rather than documented therapeutic failure. The recurrence of lesions approximately 1 week after treatment discontinuation, followed by a favourable response after reintroduction of acitretin at an increased dosing frequency alongside topical therapy, highlights the potential importance of treatment adherence and continuity in maintaining disease control. This case adds to the clinical experience with acitretin in PRP and illustrates the possibility of disease recurrence following treatment discontinuation. Unlike previously reported cases of refractory or extensive PRP requiring escalation to immunosuppressive therapies or biologic agents, our patient achieved complete clinical remission with low-dose acitretin combined with topical therapies. Nevertheless, the long-term prognosis remains uncertain given the variable natural history of juvenile type IV PRP. Although some cases resolve within 1–3 years of disease onset, others achieve complete remission by late adolescence [8].

CONCLUSION

PRP can significantly affect patients' quality of life, particularly in its chronic and treatment-resistant forms. This case highlights the clinical response to low-dose acitretin combined with topical therapy in achieving complete clinical remission of circumscribed juvenile PRP (Type IV) without treatment-related adverse effects. The recurrence following treatment interruption highlights the importance of treatment adherence and continuity in maintaining disease control. In the absence of standardised treatment guidelines, this case adds to the limited clinical evidence regarding the use of low-dose acitretin in paediatric Type IV PRP.

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None.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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