



Evaluation of Efficacy and Safety of Topical 88% Phenol in the Treatment of Alopecia Areata at a Tertiary Care Hospital: A Prospective Clinical Study.

Dr. Suhail Khan M K¹, Dr. M Shreesha Bairy², Dr. Mohan Rao^{3*}, Dr. Khuteja Tul Kubra⁴, Dr. G S Asha⁵.

¹Consultant Dermatologist, Department of Dermatology, Chinmaya Mission Hospital, Bengaluru, Karnataka, India.

²Senior resident, Department of Dermatology, The Oxford Medical College, Hospital and Research Centre, Bengaluru, Karnataka, India.

³Consultant Dermatologist, Dermaqure Skin and Cosmetology Clinic, Bengaluru, Karnataka, India.

⁴Senior Lecturer, Department of Prosthodontics and Crown and Bridge, Bangalore institute of dental sciences, Bengaluru, Karnataka, India.

⁵Professor, Department of Dermatology, BGS Medical College & Hospital, Nagarur, Bengaluru, Karnataka, India.



Correspondence:

Dr. Mohan Rao.

Consultant Dermatologist,
Dermaqure Skin and
Cosmetology Clinic, Bengaluru,
Karnataka, India.

Email:

mohannaraharirao@gmail.com

Received: 15-06-2026

Revised: 03-07-2026

Accepted: 13-07-2026

Published: 29-07-2026

Abstract

Background: Alopecia areata (AA) is a frequent non-scarring autoimmune hair loss disorder. Many therapies are available but access to affordable, effective and easily administered topical modalities remains a clinical challenge, particularly in resource-limited tertiary care settings. Topical 88% phenol is a chemical irritant and immunomodulator, providing a potential alternative to conventional contact sensitization. **Methods:** This was a prospective observational clinical study carried out on 60 patients of limited AA attending the outpatient department of dermatology of a tertiary care hospital over a defined period of time. Patches were treated with topical 88% phenol at intervals of 3 weeks for 9 weeks. Serial assessments of hair texture (vellus, intermediate, terminal), pigmentation, regrowth density, global clinical response and adverse effects were documented at baseline, week 3, week 6 and week 9. Repeated measures tests were used to assess statistical significance across time points. **Results:** Sixty patients (39 females, 21 males; mean age group 41-50 years) showed significant longitudinal improvement for all the regrowth parameters ($p < 0.001$). By week 9, 81.7% ($n = 49$) of patients had conversion to terminal hair, 76.7% ($n = 46$) had normal hair pigmentation, and 89.9% ($n = 54$) had moderate-to-excellent hair density. At Week 9, overall clinical assessment showed a good to excellent response in 80.0% of patients ($n = 48$). Adverse events were mild and self-limiting in 23.3% of the patients ($n=14$), with erythema (8.3%) and transient hypopigmentation (8.3%) being most common. **Conclusion:** Topical 88% phenol is an effective, rapid, well tolerated and economical therapeutic modality for localized alopecia areata with significant terminal hair regrowth with minimal side effects over 9 weeks of treatment.

Keywords: Alopecia areata, phenol, contact immunotherapy, hair regrowth, tertiary care hospital.



This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (<http://creativecommons.org/licenses/by/4.0/>).

INTRODUCTION

Alopecia areata (AA) is an immune-mediated, non-scarring form of hair loss characterized by the sudden appearance of well-demarcated areas of hair loss on the scalp and body [1]. It affects about 2% of the general population worldwide occurring in all ethnic, age and gender groups [2]. Pathophysiology The pathophysiology is based on the breakdown of immune privilege in the anagen hair follicle via autoreactive CD8⁺ T-lymphocytes and pro-inflammatory cytokines including interferon-gamma (IFN- γ) and interleukin-15 (IL-15) which induce premature catagen and telogen arrest of cycling hair follicles without destruction of the follicular stem cell pool [3, 4].

Although follicular arrest is reversible, the clinical management of AA poses a challenge. Treatment options include topical, intralesional and systemic corticosteroids, topical minoxidil, calcineurin inhibitors, contact sensitizers such as diphenylcyclopropenone (DPCP) and squaric acid dibutylester (SADBE) and new systemic Janus kinase (JAK) inhibitors [5,6]. Intralesional triamcinolone acetonide is considered the first-line therapy for localized patchy AA, but pain, systemic absorption, and the risk of localized cutaneous atrophy and telangiectasia limit its use in extensive or multiple patches [7]. Similarly, classical contact immunotherapy (DPCP and SADBE) requires extensive pharmaceutical compounding, multiple sensitization visits and careful titration to prevent severe eczematous reactions and cervical lymphadenopathy, making it difficult to maintain consistently in busy tertiary care centers in resource-limited settings [8].

Topical phenol (carbolic acid) at high concentrations (88%) has been used as an immunomodulatory agent and counter-irritant agent in dermatological practice [9]. When applied topically to patches of alopecia areata, 88% phenol produces a controlled superficial irritant contact dermatitis. It is hypothesised that this localised chemical irritation acts via “antigenic competition”, where the recruitment of a generalised inflammatory infiltrate distracts or downregulates the follicle-specific autoreactive T-cell attack, allowing hair follicles to re-enter the normal anagen growth phase [10,11]. Also, phenol has antiseptic and anesthetic effects, thus its application is relatively painless after a transient burning sensation [12].

Despite its clinical potential, there is limited robust prospective data evaluating standardized schedules of topical phenol in AA. Therefore, this study was undertaken to assess the clinical efficacy, longitudinal regrowth pattern and safety profile of topical 88% phenol in 3 weekly intervals, for 9 weeks of the course of treatment, in patients presenting with patchy alopecia areata in a tertiary care hospital.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital based, prospective, observational clinical study conducted in the Department of Dermatology, Venereology and Leprosy in a tertiary care medical college and hospital during a specified period. The study protocol was approved by the institutional medical ethics committee and written informed consent was obtained from all patients (or their legal guardians in the case of minors) prior to enrollment and initiation of therapy.

Study Population and Selection Criteria

A total of 60 consecutive patients diagnosed with clinically localized patchy alopecia areata of the scalp or beard and meeting the inclusion criteria were enrolled. Inclusion criteria were (1) patchy AA involving less than 40% of the scalp surface area, (2) age older than 20 years, and (3) willingness to adhere to the 9-week treatment schedule and follow-up protocols. Exclusion criteria included: (1) alopecia totalis, alopecia universalis, or ophiasis pattern AA; (2) patients who had received topical, intralesional, or systemic immunosuppressive treatment for AA within the last 4 weeks; (3) active scalp infection, dermatoses, or known hypersensitivity to phenol; and (4) pregnant or lactating women.

Treatment Protocol and Application Technique

At baseline, a detailed clinical history was recorded, including disease duration, age of onset, family history, and associated dermatological or systemic comorbidities (such as vitiligo, atopic dermatitis, psoriasis, lichen planus, and tuberculosis). Affected areas were cleansed with normal saline and allowed to dry. Topical 88% phenol solution (USP grade carbolic acid crystals dissolved in distilled water to achieve an 88% w/v concentration) was carefully applied to the alopecic patches using a cotton-tipped applicator. The solution was applied in a single, even layer until a uniform, mild greyish-white frosting appeared on the treated skin, typically within 30 to 60 seconds. Care was taken to avoid spilling onto surrounding normal skin or eyes. No post-application neutralization or dressings were applied. Patients were advised to avoid washing the treated area for at least 6 hours post-procedure. Treatment sessions were repeated at 3-week intervals (Baseline, Week 3, and Week 6) for a total study duration of 9 weeks.

Clinical Monitoring and Outcome Measures

Patients were evaluated at baseline and serially at Week 3, Week 6, and Week 9 using standardized clinical examination and digital trichoscopy/dermoscopy. Therapeutic efficacy was quantified across four standardized clinical parameters:

- Regrown Hair Texture: Classified categorically as Vellus hair (fine, non-pigmented, short hairs), Intermediate hair (transitional caliber and length), or Terminal hair (coarse, long, fully mature hair shafts).
- Regrown Hair Pigmentation: Scored as Lightly pigmented, Moderately pigmented, or Normally pigmented (matching baseline natural hair color).
- Density of Hair Regrowth: Graded on a 4-point ordinal scale as Minimal (< 25% area coverage), Moderate (25–49% area coverage), Good (50–74% area coverage), or Excellent (≥ 75% area coverage).
- Overall Assessment Score: Assigned at the end of Week 9 as Poor (no clinical benefit or < 25% regrowth), Moderate (25–49% regrowth), Good (50–74% regrowth), or Excellent (≥ 75% complete terminal regrowth). A score of Good or Excellent was defined as a clinically successful therapeutic outcome.

Safety and tolerability were monitored at each visit by documenting adverse effects, including erythema, blistering/erosion/wounds, hypopigmentation, and hyperpigmentation.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using appropriate statistical software. Categorical variables and frequency distribution are expressed as number of cases (n) and percentages (%). Longitudinal changes in ordinal and categorical regrowth scores across time points (Weeks 3, 6 and 9) were assessed using non-parametric repeated measures tests (Friedman test for ordinal density scores and Cochran's Q test / Chi-square test of independence for categorical texture and pigmentation changes). Two-sided p values of <0.05 were considered statistically significant.

RESULTS

Table 1: Baseline Demographic Profile, Disease Duration, and Associated Medical Comorbidities of the Study Cohort (n = 60)

Parameter	Male (n = 21)	Female (n = 39)	Total (N = 60), n (%)
Age Group (Years)			
21–30	2	4	6 (10.0%)
31–40	7	11	18 (30.0%)
41–50	9	18	27 (45.0%)
51–60	1	4	5 (8.3%)
> 60	2	2	4 (6.7%)
Duration of Disease			
< 6 Months	4	7	11 (18.3%)
6–12 Months	14	21	35 (58.3%)
> 12 Months	3	11	14 (23.3%)
Associated Comorbidities			
Vitiligo	2	1	3 (5.0%)
Tuberculosis	1	1	2 (3.3%)
Atopy	1	1	2 (3.3%)
Lichen Planus	1	0	1 (1.7%)
Psoriasis	1	0	1 (1.7%)
Other Conditions	1	2	3 (5.0%)

The treatment course was completed by the 60 patients (n = 60) diagnosed with patchy alopecia areata. The baseline demographic profiles, duration of disease, and accompanying medical co-morbidities of the subjects in the study are given in Table 1. 39 females (65.0%) and 21 males (35.0%) comprising the entire cohort showing the overall male-female ratio of 1:1.86. The patients' age ranged from 21 years to more than 60 years. The maximum number of cases was in the 41–50 year age group (n = 27, 45.0%). The second maximum cases were noted in the 31–40 year age group (n = 18, 30.0%). Of the present cohort, more than half (n = 35, 58.3%) reported hair loss for a period that was between 6 and 12 months. Eleven patients (18.3%) presented within 6 months of onset and 14 patients (23.3%) had chronic patches of more than 12 months. Twelve patients had associated systemic or dermatological comorbidities (20.0%) with vitiligo (n = 3, 5.0%) tuberculosis (n = 2, 3.3%) and atopy (n = 2, 3.3%) being the most common associated diseases.

Table 2: Serial Evaluation of Hair Texture, Pigmentation, and Density Across the 9-Week Treatment Regimen

Parameter	Week 3 (n)	Week 6 (n)	Week 9 (n)	p-value
Hair Texture				< 0.001
Vellus Hair	37	31	9	
Intermediate Hair	6	1	1	
Terminal Hair	17	28	49	
Hair Pigmentation				< 0.001
Lightly Pigmented	34	19	8*	
Moderately Pigmented	25	27	16*	
Normally Pigmented	1	14	46*	
Hair Density				< 0.001
Minimal	27	11	6	
Moderate	23	21	8	
Good	6	19	26	
Excellent	4	9	20	

Table 2 shows that serial assessments over the 9-week treatment course resulted in significant (and analysing) improvements in hair texture, colour and mainly density of hair growth. At Week 3 the major regrowth texture was fine vellus hair (n = 37, 61.7%) and only 17 (28.3%) had terminal hair shaft. By Week 6, terminal hair regrowth extended to 28 patients (46.7%) and by Week 9, 49 patients (81.7% of the studied population) had displayed mature terminal hair regrowth (p < 0.001 over time).

In parallel, hair pigmentation improved significantly over time. At Week 3, lightly pigmented regrowth was observed in 34 patients (56.7%); meanwhile, 1 patient (1.7%) developed normally pigmented hair matching the baseline natural color. By Week 9, normal pigmentation was observed in 46 patients (76.7%), which marked a statistically significant change toward the functional restoration of melanocytes ($p < 0.001$). The density of regrowth also grew considerably: the patients that presented with little regrowth density ($< 25\%$) fell significantly from 27 (45.0%) at Week 3 to just 6 (10.0%) at Week 9. In contrast, Good (50–74%) or Excellent ($\geq 75\%$) density increased from 10 patients (16.7%) at Week 3 to 46 patients (76.7%) at Week 9 ($p < 0.001$).

Table 3: Global Clinical Efficacy at Week 9 Based on Standardized 4-Point Overall Assessment Score

Clinical Response Category	Number of Patients (n)	Percentage (%)
Excellent ($\geq 75\%$ Regrowth)	20	33.3%
Good (50–74% Regrowth)	28	46.7%
Moderate (25–49% Regrowth)	5	8.3%
Poor ($< 25\%$ Regrowth / No Response)	7	11.7%
Total Cohort Accounted	60	100.0%

At the end of the 9-week treatment programme, the overall clinical efficacy was assessed and graded in accordance with the standardized 4-point overall assessment score (Table 3). One-third of the study population ($n = 20$, 33.3%) achieved an Excellent clinical response which was more than 75% complete terminal hair regrowth. A total of 28 patients (46.7%) attained Good clinical response (50–74% regrowth). A combined total of 48 patients achieved a successful therapeutic outcome (Good to Excellent response) with overall clinical success of 80.0%. Out of the total, 5 patients (8.3%) achieved moderate improvement (25–49% regrowth) while 7 patients (11.7%) were classed as non-responders or Poor responders ($< 25\%$ regrowth or no clinical benefit).

Table 4: Incidence of Documented Adverse Cutaneous Effects During the 9-Week Topical 88% Phenol Regimen

Reported Adverse Effect	Number of Patients (n)	Percentage (%)
Erythema	5	8.3%
Hypopigmentation	5	8.3%
Wound / Localized Erosion	3	5.0%
Hyperpigmentation	1	1.7%
No Reported Side Effects	46	76.7%
Total Cohort Accounted	60	100.0%

Table 4 shows that topical 88% phenol was well tolerated through the various sessions. Most of the study patients ($n = 46$, 76.7%) completed the 9-week course without reporting and showing cutaneous or systemic adverse side effects apart from a mild burning sensation at the site of application. Of the 14 patients (23.3%) who experienced document side effects, the most common were localized erythema ($n = 5$, 8.3%) and post-inflammatory hypopigmentation ($n = 5$, 8.3%). There was a 5.0% incidence of superficial localized wound or erosion of the epidermis at the site of application in three patients which was resolved with conservative topical antibiotic ointment within 7 to 10 days without leaving any scars. A patient (1.7%) developed mild PIH. On the treated patches, all adverse effects were local. None of the patients in this study developed systemic phenol toxicity, severe allergic contact dermatitis, regional lymphadenopathy, or a secondary bacterial infection.

DISCUSSION

The baseline demographics displayed a predominance of females (65.0% females vs. 35.0% males, female to male ratio 1.86:1). The epidemiological reviews of Strazzulla et al. [1] and Villasante Fricke et al. [2] in the general population usually find an equal sex distribution for AA. Conversely, most hospital- or clinic-based cohorts are chiefly female biased. The findings of Sharma et al. [14] complemented the current ones. Women attending the targeted tertiary centres seeking treatment options indicate rising cosmetic awareness and health-seeking behaviour. In our study, we found that the most affected age group were those in the 41–50 range (45.0%), followed by the 31–40 range (30.0%). Patchy AA in this outpatient setting predominantly affects those in their productive years. Moreover, 58.3% of our patients presented after 6 and 12 months' disease onset.

This falls within the ideal therapeutic window specified by Tosti et al. [15], which indicates that longstanding alopecia (> 2 years) is frequently associated with the presence of fibrosis in the follicles as well as resistance to topical immunotherapy. The principles behind chemical counter-irritation and immunomodulation have contributed to the effectiveness of high-concentration topical phenol in alopecia areata. The application of 88% phenol to alopecic skin causes denaturation of superficially located epidermal proteins to occur immediately, leading to clinical presentation of greyish-

white frosting on the skin [9, 10]. This localized chemical injury causes a non-specific inflammatory reaction, which consists of an increase in blood flow (vasodilation), macrophages, and neutrophils infiltration. This theory of antigenic competition postulated by Happle et al. [11] and similarly used in contact immunotherapy by Horio et al. [12], states that this major influx of non-specific inflammatory mediators transforms the local cytokine microenvironment, inhibiting or redirecting the autoimmune T-cell assault on follicular melanocytes and keratinocytes. As a result of autoimmune lymphocytic arrest, dormant telogen follicles are released to re-enter the active anagen phase of the hair cycle. Our longitudinal clinical data are very consistent with this theory.

Throughout the 9-week course of treatment, we noted a striking and statistically significant change from fine vellus hair (61.7% at Week 3) to coarse mature terminal hair (81.7% at Week 9; $p < 0.001$). Meanwhile, melanocytic function in the regrowing hair matrix normalized, with 76.7% of patients having normal hair pigmentation at Week 9 compared to merely 1.7% ($p < 0.001$) at Week 3. The overall clinical success rate of 80.0% achieved in our study matches, or is better than, the response rates of the international guidelines and trials for conventional therapies. For example, Messenger et al. [5] and Meah et al. [6] observed a response rate of 50-78% for DPCP and SADBE, while Kumari et al. [7] reported efficacy of 60-80% for intralesional triamcinolone acetonide in limited patchy AA. Likewise, our results are comparable to those of Singh et al. [8] 80% regrowth in responders DPCP immunotherapy, without the allergic complications. When viewed from a clinical and operational perspective, topical 88% phenol offers several distinct advantages over contact sensitizers when used in tertiary care centers in developing countries/resource poor set ups. According to Singh et al. [8], classical sensitizers DPCP and SADBE display ant mutagenicity in vitro, require careful titration to prevent the severe eczematous spread, photolabile, expensive to compound. In contrast, phenol is cheap, easily available as an antiseptic chemical in hospital formulation, very stable, and does not require a preliminary sensitization visit or complex titration schedule, as indicated by Savin et al. [10].

Furthermore, it has a narcotic effect which numbs the skin instantly on application. This guarantees good compliance from the patient with no chronic pruritus and cervical lymphadenopathy that was observed frequently with DPCP therapy shown by Horio et al. [12] in their study. The safety profile of our cohort enhances its clinical viability. Of the study population, 76.7% did not experience any side effects. Complications reported were limited to mild localized erythema (8.3%), transient hypopigmentation (8.3%), localized superficial erosion (5.0%), and minor hyperpigmentation (1.7%). Crucially, we did not observe any patients with scarring alopecia, persistent dyschromia, allergic contact dermatitis or systemic phenol toxicity which, according to Deprez et al. [9], occurs infrequently. They warned that this systems complication may occur when the body surface area undergoing a chemical peel exceeds 50%. Application of localized area with cotton tipped swabs in case of limited patchy AA (< 40% scalp area) remains systemically safe. Although our future findings have exciting promise, there are several limitations.

First, the research did not have a randomised vehicle-controlled or positive-control comparator arm (e.g. intralesional triamcinolone or topical minoxidil). As such, despite the rapid timing of terminal regrowth with respect to phenol application, spontaneous remission as suggested by Ikeda et al. [13] cannot be discounted either. Moreover, the second limitation of this study is of small sample size ($n = 60$) and single tertiary care center. Third, the 9-week treatment window limited patient follow-up. To assess long-term relapse rates following the cessation of phenol treatment, extended post-treatment follow-up (i.e. 6 to 12 months) is required. Future studies with further follow-up will hopefully add to these data and confirm the present findings and the role of endovenous laser treatment in treatment algorithms.

CONCLUSION

Topical 88% phenol is a safe, economical, effective and rapid treatment modality for localised patchy alopecia areata. During a treatment course of 9 weeks at 3-week intervals, it successfully induced mature terminal hair regrowth and normal pigmentation in the vast majority of patients (80.0% favorable response) with minimal and reversible adverse effects. Topical 88% phenol is an affordable, stable and easily administered option without the compounding complexities or allergic risks of classical contact immunotherapies, and is a valuable, practical addition to the dermatological armamentarium in tertiary care medical centers.

REFERENCES

1. Strazzulla LC, Wang EHC, Avila L, Lo Sicco K, Brinster N, Christiano AM, et al. Alopecia areata: Disease characteristics, clinical evaluation, and new perspectives on pathogenesis. *J Am Acad Dermatol*. 2018;78(1):1-12.
2. Villasante Fricke AC, Miteva M. Epidemiology and burden of alopecia areata: a systematic review. *Clin Cosmet Invest Dermatol*. 2015;8:397-403.
3. Rajabi F, Drake LA, Senna MM, Rezaei N. Alopecia areata: a review of disease pathogenesis. *Br J Dermatol*. 2018;179(5):1033-1048.
4. Simakou T, Becerril-Villanueva E, Doherty CB, Posso-De Los Rios CJ, Miteva M, McElwee KJ. Alopecia areata: A multifactorial autoimmune condition. *J Autoimmun*. 2019;98:74-83.

5. Messenger AG, McKillop J, Farrant P, McDonagh AJ, Sladden M. British Association of Dermatologists' guidelines for the management of alopecia areata 2012. *Br J Dermatol.* 2012;166(5):916-926.
6. Meah N, Wall D, York K, Bhoyrul B, Boyers M, Jolliffe V, et al. The Alopecia Areata Consensus of Experts (ACE) study: Results of an international expert opinion voting process on recommendations for the diagnosis and management of alopecia areata. *J Am Acad Dermatol.* 2020;83(1):123-130.
7. Kumari P, Ramesh V, Singh A. Randomized controlled trial comparing intralesional triamcinolone acetonide and topical minoxidil versus intralesional triamcinolone acetonide alone in patchy alopecia areata. *Indian J Dermatol Venereol Leprol.* 2021;87(1):33-39.
8. Singh G, Desai SC, Pande S, Dorik AP. Diphenylcyclopropanone (DPCP) immunotherapy in alopecia areata: A prospective study of 40 cases from a tertiary care center. *Indian J Dermatol.* 2016;61(4):389-394.
9. Deprez P. Easy Guide to Chemical Peelings. 2nd ed. London: Informa Healthcare; 2008. p. 112-125.
10. Savin RC. Treatment of chronic alopecia areata with topical phenol. *Br J Dermatol.* 1993;128(2):191-193.
11. Happle R. Antigenic competition as a therapeutic concept for alopecia areata. *Arch Dermatol Res.* 1980;267(2):109-114.
12. Horio T, Arakawa K. Contact immunotherapy for alopecia areata. *Arch Dermatol.* 1981;117(8):473-476.
13. Ikeda T. A new classification of alopecia areata. *Dermatologica.* 1965;131(6):421-445.
14. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in Northern India. *Int J Dermatol.* 1996;35(1):22-27.
15. Tosti A, Guidetti MS, Bardazzi F, Misciali C. Long-term results of topical immunotherapy in children with alopecia totalis or alopecia universalis. *J Am Acad Dermatol.* 1996;35(2):199-201.