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ORIGINAL CONTRIBUTION

Clinical and trichoscopic evaluation of trichloroacetic acid 35% vs phenol 88% peels in treatment of alopecia areata

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Abstract

Background: Among alopecia areata (AA) treatments, contact irritants (anthralin) and topical immunotherapies (diphenylcyclopropenone) have been successfully used. Chemoexfoliation can potentially be utilized, acting as irritants and consecutively immunomodulators. Peels via therapeutic wounding provoke growth factors and cytokines that may induce hair regrowth.

Aim: To evaluate and compare trichloroacetic acid (TCA) 35% and phenol 88% peels effectiveness and tolerability in patchy AA.

Patients/Methods: This comparative, randomized, double-blind study included 20 patients with multifocal patchy AA. In each patient, 2 patches were selected and randomized into group I (20 patches: TCA 35%) and group II (20 patches: phenol 88%). A session was performed every 3 weeks for 9 weeks. Response was assessed by two blinded observers as regards percentage of clinical improvement, severity of alopecia tool (SALT), and trichoscopic scaled scores for dystrophic and terminal hairs, respectively. Patients were scheduled for follow-up visits over 6 months past treatment cessation.

Results: A total of 19 patients completed the study and showed significant reduction in SALT score. TCA- and phenol-treated patches demonstrated significant improvement in the percentage of clinical improvement, trichoscopic scale of dystrophic and terminal hairs. However, TCA was superior to phenol as it showed significant more reduction in trichoscopic score of dystrophic hairs and significant higher increase in terminal hairs. Phenol yielded significant higher discomfort than TCA. No relapse was detected.

Conclusions: Trichloroacetic acid 35% and phenol 88% peels can be considered effective therapeutic modalities for patchy AA. TCA 35% represents a treatment of choice in terms of the efficacy and tolerability.

KEYWORDS

alopecia areata, chemical peel, phenol, trichloroacetic acid, trichoscopy

1 | INTRODUCTION

Alopecia areata (AA) is a common autoimmune disorder afflicting hair follicles in the anagen phase.[1] Despite its benign nature, its esthetic repercussions have a profound psychological impact.[2] Treatment primarily relies on intralesional and topical corticosteroids. Other topicals include minoxidil, contact irritants "anthralin," and immunotherapy "diphenylcyclopropenone (DPCP)." Photochemotherapy and systemic immunosuppressive therapy can also be used. Biologics like etanercept and infliximab have been investigated.[3] Up till now, there is no universally proven therapy that induces and maintains remission of AA in all patients.[4]

Growing evidence supports the role of cytotoxic CD8+ NKG2D+ T cells in AA. They maintain IFN gamma and IL-15-mediated inflammatory response around hair follicles through pathways mediated by JAK receptors.[5]

Novel Janus kinase (JAK) inhibitors such as ruxolitinib, tofacitinib, and baricitinib were reported to be effective in moderate-to-severe AA, alopecia totalis and universalis.[5-7]

Among topical immunotherapeutic agents, 88% phenol peel is a contact irritant[8] that induces epidermal coagulation "therapeutic wounding" and can halt the immune attack on anagen hairs through provoking antigenic competition and immunomodulation.[9] It is an affordable office procedure, yet it can lead to cardiotoxicity and other systemic toxicities within hours when applied over large areas.[10,11] QTc prolongation may get further prolonged with phenol.[12] Therefore, expertise and proper patient monitoring are a must. Likewise, trichloroacetic acid (TCA) peel causes epidermal and dermal coagulation as well as collagen necrosis[13] with subsequent release of various inflammatory cytokines and growth factors such as platelet-derived growth factor (PDGF).[14]

Although phenol was successfully used in previous studies,[8,9,15,16] TCA 35% was not formally studied before in the treatment of AA. This study was conducted to evaluate TCA 35% peel as a potential therapeutic modality of AA in comparison with phenol 88% as regards the efficacy, safety, tolerability, and patients' satisfaction.

2 | MATERIALS AND METHODS

This study was designed as a 9-week prospective therapeutic trial. Fifty-one patients with multifocal patchy AA (≥2 patches) of both sexes and from all age groups were assessed. Twenty eligible patients were enrolled in this study. Informed consents were obtained from all patients or guardians.

2.1 | Exclusion criteria

- Patients with AA other than the multifocal patchy variant.
- Patients experiencing significant spontaneous regrowth of terminal hair.
- Patients who received intralesional or systemic corticosteroids or any AA treatment 2 months prior to their enrollment in the study.
- Patients having another dermatological condition affecting the scalp, for example, eczema.
- Patients with psychiatric disorders influencing their compliance or expectation.
- Patients with cardiac problems or prolonged QTc interval.
- Patients with hepatic or renal disorders or autoimmune diseases.
- Pregnant and lactating females.

At initial presentation, 20 patients were subjected to thorough history taking. Scalp was examined, and severity of alopecia tool (SALT) was used to determine the percentage hair loss of the scalp.[17]

Two patches of AA/patient (total surface area to be treated: <30% of the scalp) were chosen and randomly allocated into 2 groups: group I (20 patches treated with TCA 35% in w/v) and group II (20 patches treated with phenol 88% in water). The area to be treated was degreased by alcohol 70%. In a small container, 0.5 mL of the peeling agent was placed, wherein thin cotton-tipped applicator was dipped and gently applied in uniform smooth 1-2 layers till frosting. Feathering of the borders was done by painting the chemoexfoliant from the periphery into the surrounding normal skin. Patients were asked to drink plenty of water before, during, and after phenol application. All patients were manually monitored for one hour through 1-minute assessments for radial pulse rate and rhythm with 9-minute intervals. Patients were instructed to apply a topical antibiotic cream (garamycin cream) twice daily for a week and to take the prescribed systemic antihistamines (chlorpheniramine) in case of itching. Patients received 3 regular sessions of each modality with 3-week intervals.

Response evaluation was performed by two independent dermatologists who were blinded to treatment modalities. Response was assessed as follows:

- Standardized scalp photographs by Canon PowerShot digital camera (SX700 HS, Full HD, 16.1 Megapixels, made in Japan) were taken. Four views (top, both sides, and back) were obtained at baseline, on 3-weekly basis, and at the study end point (3 months). Objective clinical evaluation for the percentage of grossly perceived terminal hairs of each individual patch and SALT was scored before and after therapy.
- Trichoscopic assessment was carried out at each visit for early detection of adverse effects and thorough evaluation of dystrophic forms (yellow and black dots, broken hairs, exclamation marks, and coudability, angulated, and tulip hairs) and terminal hairs. Baseline and post-therapy digital microphotographs of the representative trichoscopic fields standardized by skull bony landmarks were obtained using DermLite HÜD™ handheld dermoscope attached to a smart phone. Trichoscopic parameters were quantified in the center of each index patch and at four representative fields in the periphery designated as 3, 6, 9, and 12 o'clock positions. A

percentage per patch was calculated as summation of readings at the center and the periphery.
  - A semi-quantitative scaled score was then deployed and graded according to the percentage of dystrophic forms in the representative fields per patch: 4 = 75%-100%, 3 = 50%- 74%, 2 = 25%- 49%, 1 = 1%- 24%, and 0 = 0%.
  - The percentage of terminal hairs in the patch was reported.
- Patients were properly examined to detect any cutaneous side effects such as pain, erythema, pigment alteration, superficial scarring, and/or infections. Patients were asked to scale the pain as follows: 1 = mild; 2 = moderate; and 3 = severe pain.
- Patients' global satisfaction was assessed at the end of the study via using the five-point grading scale: 3 = excellent, cannot be more satisfied; 2 = moderately satisfied; 1 = poorly satisfied; 0 = not satisfied; and −1 = condition worsened.
- By the end of sessions, monthly follow-up was performed for 6 consecutive months to detect AA relapse at the treated sites or elsewhere as well as the development of delayed-onset adverse effects.

2.2 | Statistical analysis

Patients' data were tabulated and analyzed using IBM SPSS (Statistical Package for the Social Science; IBM Corp) release 22 for Microsoft Windows. *P* values <.05 were considered statistically significant.

3 | RESULTS

This study comprised 19 patients with multifocal patchy AA, as there was 1 dropout. Patients' demographic and clinical characteristics are summarized in Table 1.

Trichoscopy assessment of the patients before instituting the treatments revealed yellow dots in 6 patches (31.6%) and 4 patches (21.1%); black dots in 12 patches (63.2%) and 18 patches (94.7%); broken hairs in 18 patches (94.7%) and 18 patches (94.7%); exclamation mark hairs in 18 patches (94.7%) and 18 patches (94.7%); coudability hairs in 10 patches (52.6%) and 8 patches (42.1%); angulated hairs in 14 patches (73.7%) and 19 patches (100%); and tulip hairs in 6 patches (31.6%) and 8 patches (42.1%).

3.1 | Clinical assessment

After 3 months therapy, the percentage of clinical improvement in total number of terminal hairs ranged from 0% to 95% with a median of 90% (mean 54.74 ± 37.28) in group I (TCA 35%), and ranged from 0% to 95% with median 85% (mean 42.89 ± 41.00)

TABLE 1 Baseline demographic and clinical data of the 19 patients

Age (Years) Mean ± SD	6-43 20.8 ± 12.73	
Sex No. (%)	Males: 5 (26.31) Females: 14 (73.68)	
Age at onset (Years) Mean ± SD	4-43 18.925 ± 13.4	
AA duration (Months) Mean ± SD	1-120 23.6 ± 30.3	
Duration of last episode (Months) Mean ± SD	1-18 5 ± 4.7	
DLQI score Mean ± SD	3-19 10.95 ± 5.42	
SALT score Mean ± SD	2-31.5 13.325 ± 8.49	
Nail affection +ve/−ve, No. (%)	2 (10.52) /17(89.47)	
Family history of AA +ve/−ve, No. (%)	2 (10.52) /17(89.47)	
Prior AA episodes +ve/−ve, No. (%)	8 (42.10) /11 (57.89)	
Previous treatments No. (%)	Topical corticosteroids	2 (10.52)
	IL corticosteroids	6 (31.57)
	Systemic corticosteroids	2 (10.52)
	No treatment	9 (47.36)

Abbreviations: AA, alopecia areata; DLQI, dermatology life quality index; SALT, severity of alopecia tool; SD, standard deviation.

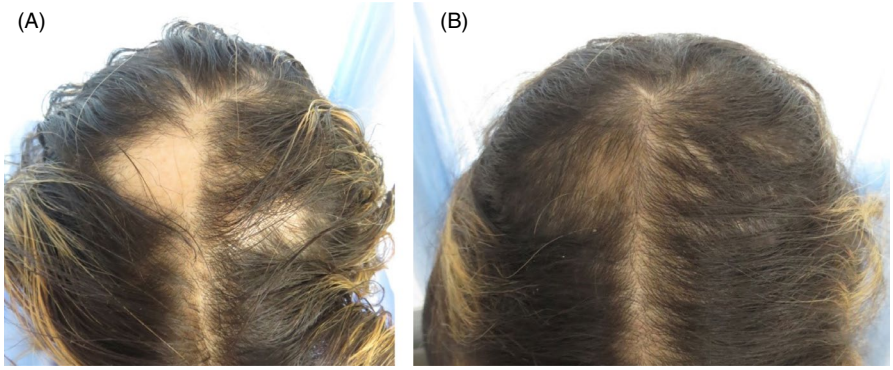

FIGURE 1 Trichloroacetic acid -treated patches (A) before treatment (B) after 9 wk

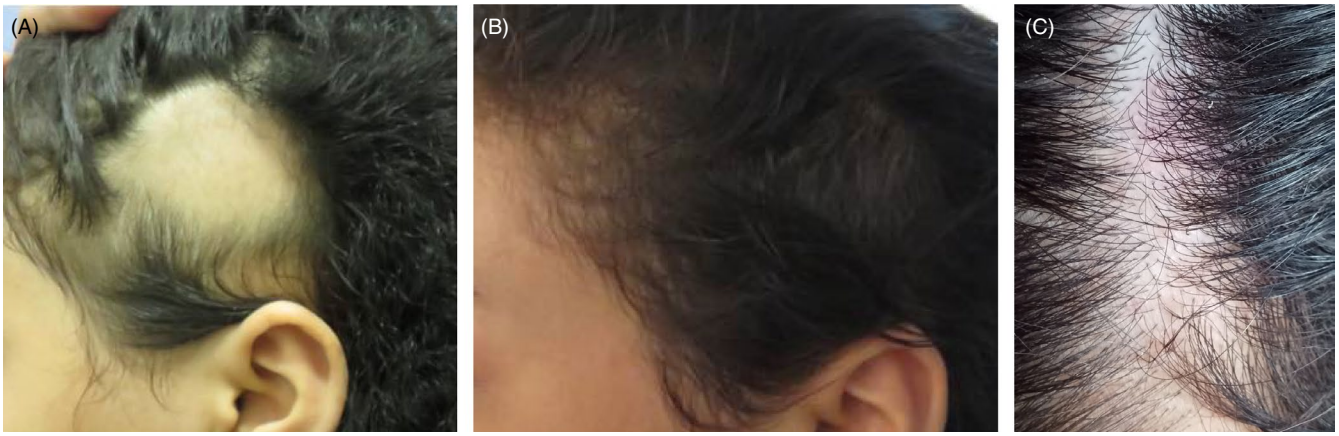

FIGURE 2 Phenol-treated patch (A) before treatment (B) after 9 wk. C, Note the persistent background erythema

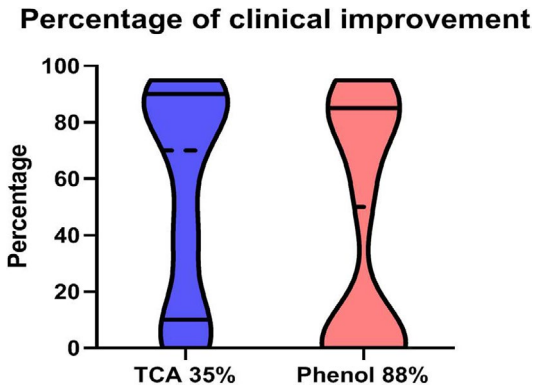

FIGURE 3 Violin plot showing the percentage of clinical improvement in trichloroacetic acid - and phenol-treated patches

in group II (phenol 88%) (Figures 1A-B, 2A-C). Both groups I and II showed significant improvement at the end of therapy, but the difference between the groups was not of statistical significance (P = .063) (Figure 3). However, 2 patients displayed excellent response in the patches treated with TCA, while those treated with phenol showed no response with further increase in the size of one patch. In addition, final SALT scores had shown significant reduction from 2-31.5 (mean 14.68 ± 9.3) to 0-27 (mean 7.13 ± 10.2) (P=<0.001).

3.2 | Trichoscopic assessment of dystrophic and terminal hairs

Group I (TCA 35%) showed significant reduction in the trichoscopic score of dystrophic hair from (1-4, mean 3.25 ± 1.11) at baseline assessment to (1-4, mean 1.05 ± 1.31) at the end point of the study (P < .001). The trichoscopic percentage of terminal hairs followed a statistically significant upward trajectory, leapfrogging from (1%-15%, mean 5.95 ± 4.33) to (1%-95%, mean 56.11 ± 40.18), (P = .001) (Figure 4A-B). Likewise, phenol-treated patches showed significant reduction in the trichoscopic score of dystrophic hair from (1-4, mean 2.90 ± 1.16) to (1-4, mean 1.68 ± 1.49), (P = .004), and a statistically significant increase in the percentage of terminal hair from (1%-20%, mean 7.85 ± 6.73) to (1%-95%, mean 42.42 ± 39.38), (P = .004) (Figure 5A-B).

When the two groups are compared, TCA was superior to phenol as it showed significant more reduction in trichoscopic score of dystrophic hairs (Figure 6) and significant more increase in terminal hairs (Figure 7) (P = .026 and .041, respectively).

3.3 | Adverse effects

All the patients had experienced initial burning pain during application of the chemoexfoliants in all areas treated regardless

the peel type as well as the lesion site and size (P = 1). However, on a pain scale ranging from 0 to 3, the discomfort induced by 88% phenol application was significantly higher than 35% TCA (P < .001). Pruritus in between sessions was approximately twice as frequently reported with phenol (83%) as TCA (38.9%) (P = .21). Itching sensation resolved with chlorpheniramine within 1-2 days. Dyschromia (hyper- or hypo-pigmentation) and erythema were frequently detected in phenol group (72.2%, 22.2%) (Figure 2B) rather than TCA group (44.4%, 5.6%), but these findings were not significant (P = .063, P = .25, respectively). There was no cutaneous scarring, abnormal pulse rate, or rhythm during treatment period.

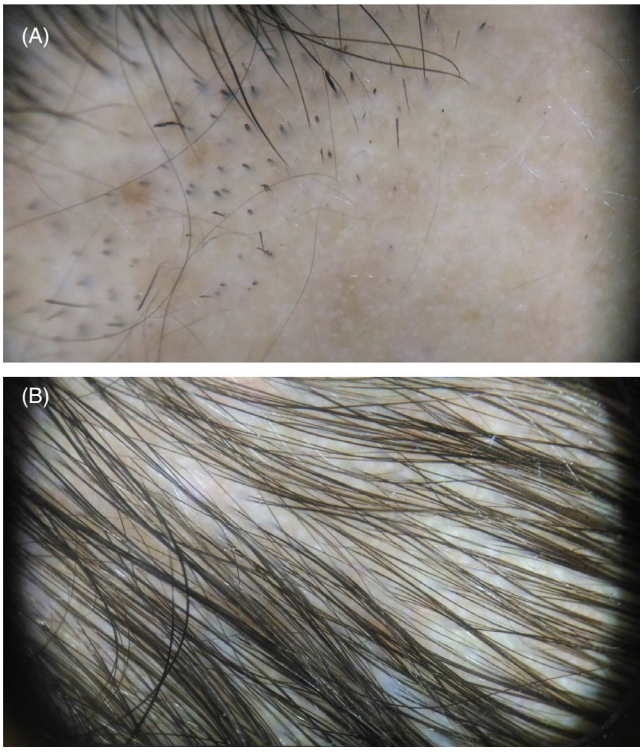

FIGURE 4 Trichoscopic images: A, baseline alopecia areata patch featuring broken hairs and black dots and B, the same patch after 9 wk of treatment with trichloroacetic acid, depicting regrowth of terminal hairs covering the entire patch

3.4 | Patients' satisfaction

By the end of sessions, patients' global satisfaction for TCA- and phenol-treated patches ranged from −1 to 3, with a median of 3 "excellent." The difference was not statistically significant (P = .065).

3.5 | Follow-up

Over 6-month follow-up, no relapse was detected in responder patches subjected to chemical peels in both groups. In addition, none of the patients showed persistent dyschromia, erythema, or scarring or systemic complications.

4 | DISCUSSION

The exact mechanism of action of various irritants (anthralin, phenol, etc) and topical sensitizers in AA is yet to be elucidated; however, immunomodulation can be considered the key role. With contact sensitizers, a counteracting cytokine milieu is induced with subsequent hair growth stimulation.[18]

The efficacy of phenol peel 88% has been documented in literature, but it has not secured a place in the priority list.[8] Concerns about systemic phenol toxicity especially when applied to large areas[10,11] warranted testing other chemoexfoliant agents to obtain equivalent therapeutic responses with better safety profile.

This study is the first to be conducted in quest of evaluating the efficacy, safety, and tolerability of TCA 35% in comparison with phenol 88% peels in patchy AA. A total of 19 patients with multifocal patchy AA completed the study. In each patient, 2 patches were managed by applying TCA 35% to one patch and phenol 88% to the other patch. This has the merit of mitigating the confounding effect of different genetic sets, comorbidities, and susceptibilities to spontaneous remission for different individuals. Those confounders when present render interpretation of results rather complicated.

In this study, high concentration of phenol peel was used (88%). At this concentration, phenol acts as keratocoagulant that prevents

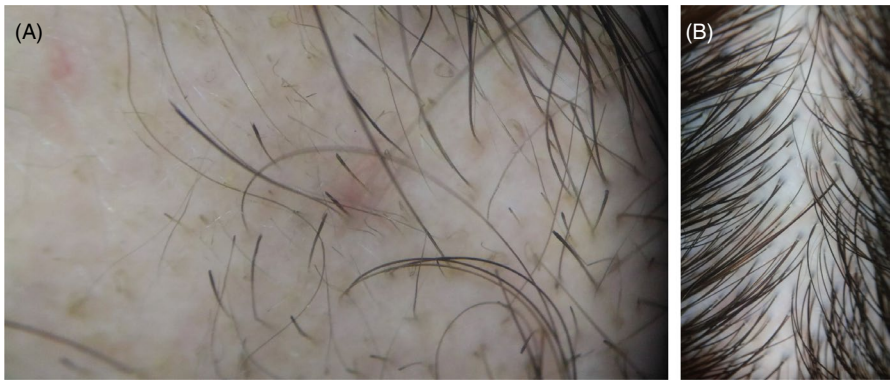

FIGURE 5 Trichoscopic images: A, baseline alopecia areata patch showing multiple tapering hairs, pigtail hairs, exclamation mark hairs, angulated hair, with yellow dots of the scalp and B, the same patch showing hair regrowth after 9 wk of treatment with phenol

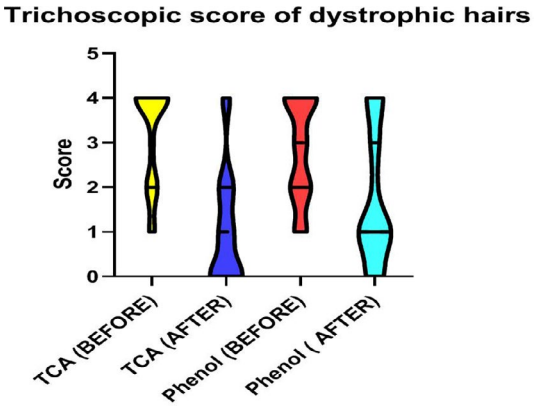

FIGURE 6 Violin plot showing the trichoscopic scores of dystrophic hairs in alopecia areata patches before and after trichloroacetic acid and phenol sessions

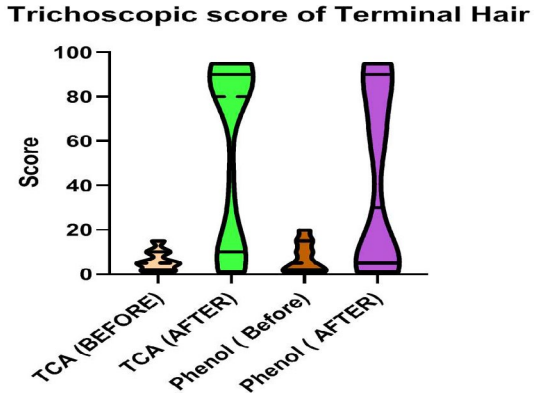

FIGURE 7 Violin plot showing the trichoscopic scores of terminal hairs in alopecia areata patches before and after trichloroacetic acid and phenol sessions

further penetration into deep dermis and can eliminate the risk of systemic absorption.[19] Phenol was chiefly employed for facial rejuvenation and was associated with cardiac arrhythmias if wide areas were treated with peel exceeding 3 mL.[20] For this sake, a small amount of phenol peel (0.5 mL)[21] was applied to a small patch of AA (total treated surface area with peels per patient was <30% of the scalp) and diuresis was enhanced by drinking plenty of water pre-, during, and postpeel.

By the end of sessions, TCA and phenol groups showed significant improvement in the percentage of grossly perceived terminal hairs, trichoscopic scaled scores for dystrophic forms and terminal hairs, and patients' global satisfaction. The outcome of phenol 88% was higher than in Saini and Shishak (85% vs 66.7%).[16]

Noteworthy to mention that TCA 35% was superior to phenol 88% as it displayed significant more reduction in trichoscopic score of dystrophic hairs and significant more increase in terminal hairs.

The present study displayed significant postpeel decline in SALT scores. SALT is a helpful objective tool for estimation of hair loss; therefore, a further depth classification was proposed wherein AA can be classified as "AA + SALT score + the presence or absence of body involvement (complete or incomplete loss of body hair)".[22]

Only five previous studies had evaluated phenol in the treatment of AA, and it was associated with excellent responses.[8,9,15,16,18] Saini and Shishak stated that intralesional corticosteroids and topical phenol were comparably effective.[16] Chikhalkar et al studied the efficacy of 88% phenol in patchy AA, and they further elaborated on the characters of regrown hair regarding hair density, texture, and pigmentation.[9] A more impressive response to phenol 88% was reported by Chikhalkar et al than the current study. This may be attributed to their inclusion criteria that allowed only patients with stable AA to be recruited. Moreover, 82% of the patients in their study had disease duration <6 months, which confers better prognosis. Moreover, in their clinical assessment of hair density, fine vellus nonpigmented hairs were allocated points in contrast with our clinical assessment scoring system that allocated points only for pigmented intermediate and terminal hairs and excluded fine vellus hairs, since it is still a matter of controversy whether vellus hairs indicate remission or herald hair loss in active lesions of AA.[9]

In 2013, Kar and Singh combined 88% phenol with IV dexamethasone pulse therapy in a case of extensive recalcitrant AA of the scalp, reporting an excellent therapeutic response.[8]

Insights gleaned from those studies highlighted the efficacy of phenol 88% in induction of hair regrowth in patchy AA. The common denominator between all of the aforementioned studies and our study is that a significant proportion of patches treated with phenol 88% attained statistically and cosmetically significant hair regrowth at regular intervals of 2-4 weeks for 2-5 applications.[8,9,15,16]

The present study has introduced TCA 35% peel as a novel feasible line of treatment in patchy AA. The success of phenol 88% and TCA 35% in treatment of AA could be explained by the release of several growth factors and cytokines during wound repair process, which stimulate the affected follicle and induce hair regrowth. TCA acts on epidermal keratinocytes and can induce the production PDGF, tissue growth factor-α1 and β1, and vascular endothelial growth factors as well as interleukins-1 and 10.[14] Moreover, cytokines released during TCA- and phenol-induced wound healing may neutralize the peribulbar infiltrates and provoke hair regrowth. Lastly, most of the follicles in AA are in the telogen phase and consequently lie high in the dermis; therefore, it is proposed that phenol can pass through the follicular opening and directly stimulate the germinal center.[8]

There was an initial burning pain following TCA and phenol application in all treated patches; however, phenol 88% application was accompanied by significantly higher discomfort than TCA 35% based upon the 0 to 3 pain scale. This was analogous to what was stated by Dalpizzol and colleagues who compared a higher concentration of TCA 90% to phenol 88% in acne scars.[23] Phenol had nonsignificant higher incidence of pruritus, dyschromia, and erythema than TCA-treated patches. The transient dyschromia was higher than Saini and Shishak (72.2% vs 56.6%).[16]

No cutaneous scarring or abnormal pulse rate or rhythm detected in the current study which was consistent with earlier reports.[21,24]

Furthermore, all treated patches showed remission and resolution of all side effects when examined 6 months past therapy, in agreement with Kar and Singh.[8]

Chemical peeling is a cost-effective office procedure as long as certain precautions are taken. It spares the patient the pain of intralesional corticosteroid injections, the systemic side effects of systemic immunosuppressive therapies, the severe inflammatory reactions that occur in the setting of DPCP and phototoxic PUVA, and the burden of daily application of topical corticosteroids and/or minoxidil. Furthermore, chemical peels have the virtue of being well tolerated in patients with needle phobia as well as those with steroid phobia.

In conclusion, chemical peeling with either TCA 35% or phenol 88% at 3 weekly intervals can significantly induce hair regrowth in patchy AA. However, TCA 35% is superior to phenol 88% in terms of efficacy and safety profile. In addition, TCA can be applied to large areas. Trichoscopic examination confers an additive value, both diagnostically and prognostically in the management of AA. Limitations of our study include relatively small sample size and the absence of control patches.

CONFLICT OF INTEREST

No conflict of interest.

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