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Mini Pulse Oral Therapy, Phototherapy versus Topical Therapy for Treatment of Subclinical Vitiligo

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Abstract

Background: Subclinical vitiligo represents early or invisible depigmented lesions detectable only under Wood's light. This study compares the efficacy of oral mini-pulse corticosteroid therapy, narrowband UVB phototherapy, and topical therapy in patients with subclinical vitiligo.

Methods: Sixty patients were randomized into three equal groups. Group a received oral dexamethasone mini-pulse therapy (children: 8 mg/day; adults: 16 mg/day, administered on two consecutive days weekly for 3 months). Group B received NB-UVB phototherapy (starting 0.5 J/cm², incrementally increased to a maximum of 5 J/cm²) three times weekly for 3 months. Group C received topical mometasone furoate once daily (5 days/week) plus tacrolimus 0.1% ointment twice daily for 3 months. Outcomes included Vitiligo Area Severity Index (VASI), Vitiligo Disease Activity (VIDA), ΔVASI, and ΔVIDA.

Results: At 12-month follow-up, VASI and VIDA improvements were significantly greater in Group A, followed by Group B, with the least improvement in Group C ($P < 0.05$). ΔVASI and ΔVIDA were defined as the difference between baseline and follow-up scores. Mini-pulse therapy demonstrated earlier disease stabilization and superior repigmentation rates.

Conclusion: Oral mini-pulse therapy and NB-UVB are more effective than topical therapy in subclinical vitiligo.

INTRODUCTION

Vitiligo is an autoimmune disease that causes progressive loss of melanocytes which can lead to skin depigmentation. The diagnosis is made clinically by hypopigmented macules and patches to well-defined depigmentation [1]. The prevalence of vitiligo is estimated to be 0.4- 2% in the world population. The psychosocial burden remains one of the problems of this disease which has an impact on decreasing the patient's quality of life [2]. The same patients may have stable, progressive, and regressive spots in separate locations at the same time. It is desirable to use a non-invasive diagnostic method to evaluate all macules [3]. Wood's light examination as an integral part of vitiligo patient assessment, not only for confirmation of the diagnosis, but also for a better definition of the lesion margins and extent [4].

Wood's lamp is a source of long wave ultraviolet light (wavelength, 320-400 nm) and can be used to diagnose many skin disease including vitiligo [5]. Its use enhances the contrast between affected and unaffected skin helping in sharply defining the margins of vitiligo lesions. It actually constitutes one of the characteristic diagnostic features of vitiligo [6]. It can also detect the unseen areas of vitiligo that is called (subclinical vitiligo). Subclinical lesions represent a new addition to the current spectrum of vitiligo presentations. Their detection in the current study depended solely on Wood's light examination; hence, its use should be integral to vitiligo patient assessment. Pointing out these lesions to the patient is essential for proper application of local medications [7].

Vitiligo therapy is determined based on the disease activity and the extent of the lesions. Numerous treatment modalities have been used to treat vitiligo, such as corticosteroids, topical immunomodulatory drugs, phototherapy, skin grafts, and camouflage [8].

Phototherapy with narrowband ultraviolet B (NB-UVB) is the first line of treatment for vitiligo patients with a lesion area >5% of body surface area (BSA) [9]. The study of Liu et al. [10], stated that NB-UVB phototherapy can still be given as early as possible, even in limited lesions, with effective

results. NB-UVB phototherapy will cause repigmentation and stabilization of skin lesions with minimal side effects.

Corticosteroids in vitiligo act by reducing complement-mediated cytotoxicity. Levels of antibodies against the melanocyte surface have been found in patients with unstable vitiligo who showed good results using systemic corticosteroids [11]. When administering systemic corticosteroid therapy, the most commonly used method is to administer supra- pharmacological doses and intermittently administer betamethasone or dexamethasone via oral mini pulses. This is done in order to minimize the side effects that occurs when steroids are used in daily basis [12].

Topical treatment as Tacrolimus was found to inhibit expression of IL-17 or TNFα by reducing the proportion of Th17, suggesting the therapeutic effect on Th17-associated diseases such as inflammatory bowel disease, psoriasis, or vitiligo [13].

Several previous studies compared two of the three therapeutic modalities evaluated in this trial. Comparisons between oral mini-pulse corticosteroids and NB-UVB have shown enhanced disease stabilization with corticosteroids and superior repigmentation with combination approaches. Studies comparing NB-UVB with topical tacrolimus or corticosteroids demonstrated significant VASI reduction with both regimens. However, to our knowledge, no prior randomized clinical trial has compared all three strategies—oral mini-pulse therapy, NB-UVB phototherapy, and topical therapy—in patients specifically diagnosed with subclinical vitiligo.

The aim of the study is to compare the effectiveness of mini pulse oral therapy versus phototherapy versus topical therapy in the treatment of subclinical vitiligo.

PATIENTS AND METHODS

This prospective randomized clinical trial was carried out on 60 patients diagnosed with subclinical vitiligo had any sex and any age, during the period from July 2022 to July 2023. An informed consent for enrolment was obtained from all participants after discussing the protocol with patients or guardians of those younger than 18 years.

Exclusion criteria were pregnant or breast-feeding women, diabetics, hypertensives, immunosuppressed patients (e.g., organ transplantation, HIV), patients with other autoimmune diseases, mentally disabled or cognitively impaired subjects and malignancy.

All patients have been examined by naked eye and usual hand lens followed by examination using wood's light.

Face and neck lesions were excluded from VASI calculation because these areas demonstrate higher spontaneous and treatment-induced repigmentation rates due to greater follicular melanocyte reservoir density and superior vascular supply. Including these regions could have artificially inflated treatment response rates and confounded intergroup comparison. However, facial and neck lesions were documented separately for clinical monitoring.

Randomizations

The participants were randomized into three equal groups using a computer-generated list of random numbers sealed in an opaque envelope and were randomly allocated into three groups on a scale of 1:1:1.

Group A: Oral dexamethasone mini-pulse therapy. Children received 8 mg/day (approximately 0.15–0.2 mg/kg/day depending on weight) and adults received 16 mg/day, administered on two consecutive days weekly for 3 months.

Group B: NB-UVB phototherapy three times weekly starting at 0.5 J/cm^2 with increments of 0.5 J/cm^2 every two sessions until a maximum of 5 J/cm^2 depending on tolerance.

Group C: Topical mometasone furoate once daily (5 days/week) combined with tacrolimus 0.1% ointment twice daily for 3 months.

This regimen was adopted for all cases whatever the site of vitiligo whether face, body or acral areas for 3 months.

Patient assessment: by naked eye, hand lens and Wood's light after twelve weeks. The examination, follow up and reassessment were done by the two investigators. All patients underwent history taking including (sex, age,), disease characters such as (age of onset, duration, positive family history, positive psychological stress, site and type of vitiligo), history of prior therapy as (photo-(chemo) therapy, topical treatment as; topical calcineurin inhibitors or topical steroids, or others, oral medications as; systemic antioxidants, systemic steroids, or others).

Assessment of pre-existent lesions to determine Vitiligo area severity index (VASI) score and vitiligo disease activity (VIDA) score [14,15].

Vitiligo Area Severity Index (VASI) Score Evaluation

A hand unit rule was utilized for assessment of VASI score. There are 5 different sections of the body: the upper extremities (except hands), lower extremities

(except feet), hands, feet and trunk. The inguinal regions and buttocks are related to the lower extremities, while the axilla is related to the upper extremities. Face and neck are excluded from the examination but may be examined separately. The baseline proportion of vitiligo involvement was determined using hand units. One hand unit (composed of the palm and volar surfaces of all digits) corresponds to about 1 % of the total body surface area. Multiplying the vitiligo area in hand units (1 % per unit) by the degree of depigmentation in each hand-unit-measured patch yields the VASI for each body segment. The VASI of the entire body is calculated using the following formula: VASI = Σ (hand units) × (depigmentation) (range 0–100) [13,14].

The pigmentation degree is evaluated to the nearest of the following percentages: 10% - just specks of depigmentation present, 25% - pigmented area surpasses depigmented area, 50% - equal pigmented and depigmented regions, 75% - depigmented area exceeds pigmented area, 90% - specks of pigment present, and 100% - total depigmentation, no pigment is present [16].

Evaluation of the Disease Activity by Vitiligo Disease Activity Score (VIDA)

VIDA is a 6-point scale used to determine the activity of vitiligo based on the development of existent lesions or the formation of new lesions during a period of less than 6 wks. to 1 year. Patients with VIDA scores of -1 or 0 are the only ones eligible for vitiligo surgery [17].

Repigmentation was graded as no improvement; minimal, less than 25%; mild, 26-50%; moderate, 51-75% and marked to complete improvement, more than 75%.

Statistical Analysis

Statistical analysis was done by SPSS v28 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing ANOVA (F) test. Qualitative variables were presented as frequency and percentage (%) and were analysed utilizing the Chi-square test. A two tailed P value < 0.05 was considered statistically significant.

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RESULTS

In this study, 87 patients were assessed for eligibility, 14 patients did not meet the criteria and 13 patients refused to participate in the study. The remaining 60 patients were randomly allocated into three groups (20 patients in each). All allocated patients were followed-up and analysed statistically (Figure 1).

The baseline characteristics (age, gender, residence and family history) were insignificantly different among the studied groups (Table 1).

Table 2 shows that there was an insignificant difference among the studied groups regarding the clinical type, disease activity and the duration of the disease.

VASI was insignificantly different among the studied groups before and. While after treatment and at follow-up at 12 months, VASI was significantly lower in group A

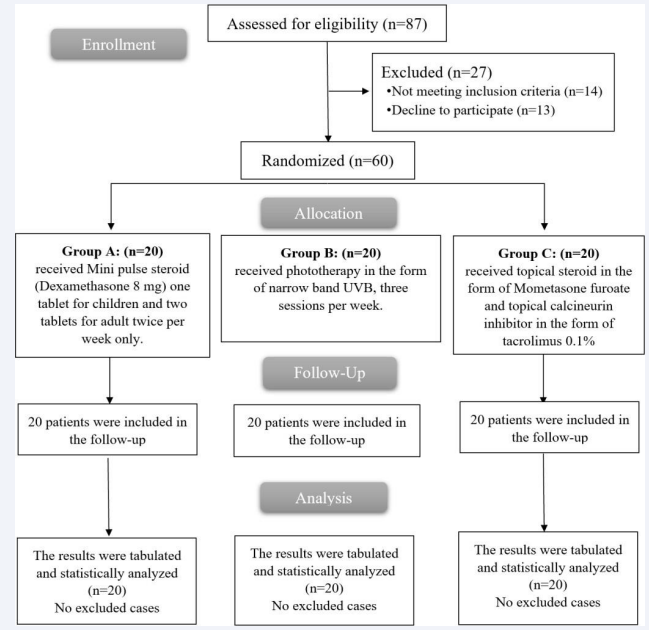

Figure 1 CONSORT flowchart of the enrolled patients

Table 1: Baseline characteristics of the studied groups

		Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Age (years)		32.6 ± 11.69	31.2 ± 11.5	31.6 ± 10.64	0.927
Gender	Male	10 (50%)	8 (40%)	13 (65%)	0.281
	Female	10 (50%)	12 (60%)	7 (35%)	
Family history		6 (30%)	7 (35%)	4 (20%)	0.563

Data presented as mean ± SD or frequency (%).

Table 2: Clinical data of the studied groups

		Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Clinical type	Generalized	10 (50%)	7 (35%)	6 (30%)	0.527
	Segmental	4 (20%)	4 (20%)	4 (20%)	
	Mixed	2 (10%)	5 (25%)	2 (10%)	
	Focal	4 (20%)	4 (20%)	8 (40%)	
Duration of the disease (years)		19.0 ± 13.75	18.3 ± 12.14	21.3 ± 10.93	0.733

Data presented as mean ± SD or frequency (%).

compared to group B and group C (P=0.031, <0.001) and was significantly lower in group B compared to group C (P=0.047). Δ VASI was significantly higher in group A compared to group B and group C (P<0.001, <0.001) and was significantly higher in group B compared to group C (P<0.001) (Table 3).

VIDA was insignificantly different among the studied groups before treatment. While after treatment, VIDA was significantly lower in group A compared to group C (P=0.014), with no significant difference between group A & B and between group B & C. At follow-up at 12 months, VIDA was significantly lower in group A compared to group B and group C (P<0.001, <0.001), with no significant difference between group B & C. Δ VIDA was significantly higher in group A compared to group B and group C (P<0.001, <0.001) and was significantly higher in group B compared to group C (P=0.027) (Table 4).

The repigmentation rate was 26-50% was 70% in group A, 55% in group B and 25% in group C. The repigmentation rate at 12 months was 51-75% was 60% in group A, 20% in group B and 10% in group C. The repigmentation rate and repigmentation rate at 12 months were significantly different among the studied groups being better in group A followed by group B (P=0.015, 0.003) (Table 5).

Table 6 shows that the improvement duration was significantly earlier in group A compared to group B and group C (P<0.001, <0.001) and was significantly earlier in group B compared to group C (P<0.001).

Table 3: Vitiligo area severity index (VASI) of the studied groups

	Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Before treatment	12.30 ± 2.03	11.60 ± 1.88	11.35 ± 2.13	0.310
After treatment	8.50 ± 1.91	8.90 ± 2.1	10.25 ± 2.34	<0.001*
Follow-up at 12 months	6.00 ± 2.22	7.40 ± 1.7	8.60 ± 1.98	0.001*
	P1=0.031*, P2<0.001*, P3=0.047*			
Δ VASI	6.30 ± 1.92	4.20 ± 1.11	2.75 ± 0.72	<0.001*
	P1<0.001*, P2<0.001*, P3<0.001*			

Data presented as mean ± SD. VASI: vitiligo area severity index, *: statistically significant as p value <0.05, P1: p value between groups A&B, P2: p value between groups A&C, P3: p value between groups B&C.

Table 4: VIDA score of the studied groups

	Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Before treatment	3.3 ± 0.47	3.4 ± 0.5	3.45 ± 0.51	0.623
After treatment	2.05 ± 0.94	2.45 ± 0.76	2.8 ± 0.89	0.030*
	P1=0.148, P2=0.014*, P3=0.190			
Follow-up at 12 months	0.15 ± 0.88	1.45 ± 0.94	2.00 ± 0.92	<0.001*
	P1<0.001*, P2<0.001*, P3=0.070			
Δ VIDA	3.15 ± 0.88	1.95 ± 0.83	1.45 ± 0.51	<0.001*
	P1<0.001*, P2<0.001*, P3=0.027*			

Data presented as mean ± SD. *: statistically significant as p value <0.05, P1: p value between groups A&B, P2: p value between groups A&C, P3: p value between groups B&C.

Table 5: Repigmentation of the studied groups

		Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Repigmentation	Less than 25%	6 (30%)	9 (45%)	15 (75%)	0.015*
	Mild, 26-50%	14 (70%)	11 (55%)	5 (25%)	
	Moderate, 51-75%	0 (0%)	0 (0%)	0 (0%)	
Repigmentation at 12 months	Less than 25%	0 (0%)	5 (25%)	7 (35%)	0.003*
	Mild, 26-50%	8 (40%)	11 (55%)	11 (55%)	
	Moderate, 51-75%	12 (60%)	4 (20%)	2 (10%)	

Data presented as frequency (%). *: statistically significant as p value <0.05.

Table 6: Improvement duration and repigmentation of the studied groups

	Group A (n=20)	Group B (n=20)	Group C (n=20)	P value
Improvement duration	1.55 ± 0.51	2.45 ± 0.51	4.1 ± 0.85	<0.001*
	P1<0.001*, P2<0.001*, P3<0.001*			

Data presented as mean ± SD. *: statistically significant as p value <0.05, P1: p value between groups A&B, P2: p value between groups A&C, P3: p value between groups B&C.

NB: ΔVASI was defined as baseline VASI minus post-treatment or follow-up VASI. ΔVIDA was defined as baseline VIDA minus post-treatment or follow-up VIDA. Higher Δ values indicate greater clinical improvement.

DISCUSSION

Loss of pigmentation in vitiligo lesions occurs in a gradual manner that apparently starts by a stage of hypopigmentation and passes through different shades of colour reduction ending in total depigmentation [18]. This colour reduction may not be detected either by naked eye or hand lens until the distinction between the affected areas and the normal skin reaches a certain threshold [19]. Pigmentary disorders are a standard skyline for effective role of Wood's light in disease diagnosis. Skin devoid of melanin fluoresces brightly. The depigmentation of vitiligo appears strikingly white and sharply delineated under a Wood's light. Wood's light effectively magnifies depigmentation that the naked eye can miss otherwise" unseen or invisible vitiligo lesions" [20].

In the current study, we detected the" invisible or unseen vitiligo lesions" that couldn't be detected by the naked eye or with hand lens, only detected under wood's light.

Subclinical lesions might be areas of failed attack upon the melanocytes that did not progress to the full pathology, or an early part of the spectrum of the disease before reaching the stage of visible hypopigmentation. They might also be areas of incomplete pigment recovery or even subclinical recurrence since few patients reported the previous involvement of these areas by vitiligo. Longitudinal follow-up for such lesions with and without treatment is further needed to determine their significance, and if there is a value for their early detection.

Pointing out these lesions to the patient so as to be included while applying topical medications is of importance, it might halt the disease process in these areas before becoming clinically visible.

We aimed to compare the effectiveness of mini pulse oral therapy versus phototherapy versus topical therapy in the treatment of subclinical vitiligo. To the best of our knowledge there is lack of studied that evaluate these regimens in treatment of subclinical vitiligo.

We found that improvement was significantly higher in minipulse oral therapy compared to phototherapy and topical treatment as the repigmentation rate was 26-50% was 70% in group A, 55% in group B and 25% in group C. The repigmentation rate at 12 months was 51-75% was 60% in group A, 20% in group B and 10% in group C. The repigmentation rate and repigmentation rate at 12 months were significantly different among the studied groups being better in group A followed by group B (P=0.015, 0.003). Moreover, the improvement duration was significantly earlier in group A compared to group B and group C (P<0.001, <0.001) and was significantly earlier in group B compared to group C (P<0.001).

Fijan et al. [21], findings indicate that oral corticosteroid pulse therapy for vitiligo is an effective means to arrest active disease; however, it only has low potential to induce substantial repigmentation when given for a period of up to 24 weeks. In Pasricha and Khaitan. [22], the probability of good or excellent repigmentation was found to correlate with increasing duration of treatment.

Two Indian trials on patients with extensive or fast-spreading vitiligo used two weekly doses of 5 mg of betamethasone/dexamethasone each or 5 mg dexamethasone with good and moderate results, respectively [22,23].

In research comparing phototherapy and oral mini pulse corticosteroid, 37 percent of patients showed a repigmentation rate of more than 75 percent after six months of treatment with the OMP + NB-UVB. These findings of Patra et al. [24], showed that there was a greater repigmentation rate of > 75 percent than the 18 percent and 8 percent repigmentation rates observed in the OMP + PUVA and OMP + BB-UVB therapy groups.

Marchioro et al. [25], found that the dexamethasone mini-pulse treatment was effective in producing stabilization and repigmentation in progressing vitiligo. At present, phototherapy has been clinically proven to be a routine option for repigmentation by promoting the recovery of melanocyte function [26].

A previous study was done by El Taieb et al. [12], where they evaluated the efficacy of tacrolimus versus NB-UVB treatment in vitiligo according to VASI score and serum IL-17 levels. VSAI has been significantly decreased after treatment with both NB-UVB and tacrolimus. Their results agree with the pervious evidences about the efficacy of NB-UVB in vitiligo [27,28]. They concluded that both tacrolimus and NB-UVB are effective treatments in patients with vitiligo for the same duration of treatment

Li et al. [29], in their meta-analysis, found that adding topical calcineurin inhibitors and vitamin D3 analogues to NB-UVB did not increase treatment outcome except in face and neck lesions.

In Pradipta [7] study, there was little difference in VASI reduction between patients with vitiligo receiving combination therapy or monotherapy with UVB-NB phototherapy after 36 and 48 phototherapy sessions.

This study provides the first three-arm comparison among oral mini-pulse corticosteroid therapy, NB-UVB phototherapy, and topical therapy specifically in subclinical vitiligo. Previous literature evaluated dual comparisons only. The superior performance of oral mini-pulse therapy may be attributed to early immunomodulatory suppression of melanocyte destruction. Exclusion of face and neck lesions strengthened internal validity by preventing anatomical response bias.

CONCLUSION

Mini-pulse oral corticosteroid therapy demonstrated the most rapid and sustained improvement in subclinical vitiligo, followed by NB-UVB phototherapy. Topical therapy showed comparatively lower efficacy.

New technologies and combinations can improve the therapeutic effect of vitiligo. Our findings would be a useful guideline for clinicians to establish the treatment strategy for patients with subclinical vitiligo and provide reference for high quality studies in the future. In the future, different combination therapies should be separately analysed to determine whether they can improve the efficacy and safety of the treatment of vitiligo.

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