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Original Research Article

PUVAsol therapy in the management of vitiligo: outcome in a dermatology clinic in South-West Nigeria

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ABSTRACT

Background: Vitiligo is a depigmentary dermatosis which currently has no cure but there are different treatment options available to treat affected patients with varying results. The aim of the study was to ascertain the effect of PUVAsol therapy offered to adult patients with vitiligo at a tertiary hospital in Nigeria.
Methods: All consecutive adult patients with vitiligo who presented at the Obafemi Awolowo University Teaching Hospitals complex, Ile-lfe and gave consent were recruited for the study which was for a period of 6 months. Patients with limited disease were treated with topical PUVAsol and those with extensive disease were treated with oral PUVAsol. The outcome of therapy for the patients was classified into three categories as follows: progressed (P), stable (S) and repigmented (R). The repigmented group was further sub-classified into fair (R1) and good (R2) representing ≤50% and >50% repigmentation of areas affected respectively.
Results: After 6 months of therapy, most of the patients (92%) had repigmentation of their lesions while the remaining had either stable lesions (6%) or progression of their lesions (2%). Amongst the patients whose lesions got repigmented, 96% of them had repigmentation in less than half of the areas affected by vitiligo which was a fair outcome.
Conclusions: PUVAsol is a treatment modality for vitiligo with some repigmentation of lesions following therapy for 6 months. PUVAsol therapy could be recommended in resource poor settings because of its low cost and availability in most parts of Nigeria.

Keywords: Nigeria, PUVAsol, Vitiligo

INTRODUCTION

Vitiligo is an acquired pigmentary disorder of the skin and mucous membranes which is characterized clinically by well-defined depigmented lesions (macules and/or patches) following melancyte dysfunction and loss.[1] It is the most common depigmentary disorder, affecting approximately 0.5 to 2.0 percent of the population and has no predilection for gender or race.[1,2] Vitiligo lesions are characteristically non-pruritic, non-scaly with preservation of skin texture and sensation. [3]

The disease could be simply classified into two broad types: non-segmental vitiligo (NSV) and segmental vitiligo (SV), with the former being commoner amongst affected patients. [4] NSV could be further sub-divided into generalized, acrofacial and mixed. Focal vitiligo could be either NSV or SV. The disorder causes a lot of cosmetic

concern especially in dark-skinned individuals due to the contrast created between normal and affected skin. Most of the patients affected have a reduction in their Quality of Life to various extents due to the embarrassment and psychological stress from the disease. [5,6]

Till date, the exact cause of vitiligo is unknown. However, there are various theories namely biochemical/cytotoxic, neural and auto-immune which are believed to be responsible for the destruction of melanocytes.[7-9]

The biochemical/cytotoxic theory emphasizes that vitiligo occurs when the melanocyte is killed by cytotoxic precursors to melanin synthesis.[7] The neural theory is based dysregulation of the nervous system either at a local or systemic level. In support of this, the melanocytes and neurons are all derived from the neural crest cells and some vitiligo are segmental following the distribution of nerves.[8] The autoimmune hypothesis is based on genetic data which are more associated to autoimmune diseases.[9] Futhermore, skin biopsies in vitiligo patients have shown the presence of epidermotropic T lymphocytes in perilesional skin which have an increased CD8/CD4 ratio and these lymphocytes frequently juxtapose the remaining melanocytes. Although these three theories are sufficient to explain the mechanisms of vitiligo, the convergence theory postulates that stress, accumulation of toxic compounds, infection, autoimmunity, mutations, altered cellular environment and impaired melanocyte migration and proliferation can all contribute in varying proportions to the aetiopathogenesis of vitiligo.[10]

There are various treatment options for vitiligo and the therapeutic option(s) chosen are dependent on various factors.[11,12] These factors include the following: age of the patient, stability of the disease, duration of treatment, side effects, cost and accessibility of medication.

Medical therapy of vitiligo has been achieved historically with agents such as;

- Topical agents (corticosteroids, calcineurin inhibitors): These agents dampen the cellular immune response and have similar efficacy. Long term application of topical corticosteroid is prone to numerous side effects and topical calcineurin inhibitors though more expensive are preferred.[11,13]
- Systemic agents (corticosteroids, minocycline): These agents are best used in patients with progressive disease to help with disease stabilization.[14,15]
- Phototherapy/ ultraviolet (UV) light therapy: Phototherapy represents another staple treatment of vitiligo which has both innate and cellular immunosuppressive as well as mitogenic and melanogenic properties that promote melanocyte proliferation and melanin synthesis. UV light occurs in two bands namely UVA and UVB. UVA phototherapy is almost always given in conjuction with with photosensitizers such as Psoralens (8-MethoxyPsoralen), referred to as PUVA therapy. The sun could be the source of the UVA light in PUVA—a technique termed PUVAsol.[16]
- Laser therapy (monochromatic excimer laser): The mechanism of action of lasers is thought to be similar to conventional phototherapy but lasers allow targeted treatment, have less total body irradiation and less impact on healthy skin.[17]

Amongst all the various treatment options mentioned above, PUVAsol has the advantage of requiring less health care resources and could be used for patients with stable or unstable disease. Futhermore, the psoralens are readily available and inexpensive and PUVAsol could be used especially in resource poor settings which have an abundance of natural sunlight as was the case in this study.

The objective of this study was to determine the treatment response to PUVAsol among patients with vitiligo in Nigeria.

METHODS

This was a prospective observational study that was carried out for a period of 6 months for each patient that was enrolled. The patients were recruited in the clinics over a period of 2 months which commenced in January/February 2015 and they were observed till June/July 2015. All consecutive adult patients with vitiligo who presented at the Dermatology clinics of the Obafemi Awolowo University Teaching Hospitals complex, Ile-lfe and gave consent were recruited for the study. Ethical Clearance was obtained from the Ethics and Research Committee of the institution.

All patients with vitiligo who were 16 years and above and gave consent were included while patients with vitiligo who had ocular abnormalities such as uveitis were excluded.

Patient's biodata were collected using a proforma that included socio-demographic information such as age, sex, occupation, marital status and educational qualification. Other relevant history such as duration of lesions, family history of vitiligo and prior history of any form of treatment for vitiligo were obtained. All the treatment experienced patients had applied some topical steroids to the lesions but with poor response.

Physical examination was done to document which of the spectrum (subtype) of the disease namely generalized, acrofacial, mixed, segmental and focal that each patient presented with. The presence or absence of leukotrichia was also noted and documented. Thereafter, severity of disease was determined using the extent of the total body surface area (BSA) of the patient that was involved. The BSA was calculated using the rule of nines and each

patient was classified as either having limited disease (BSA ≤20%) or extensive disease (BSA >20%).

The treatment modality used for therapy was the 8-MethOxyPsoralen also known as meladinine plus UltraViolet A radiation from early morning sunlight referred to as PUVAsol. Patients with limited disease were given topical PUVAsol (0.1% meladinine solution). The solution was diluted in 60% alcohol to a concentration of 0.01%. This was to prevent patients from having severe erythema (pinkish discoloration of depigmented skin which persists for more than 24 hours) after topical application.

The dilution was done and demonstrated for each patient in the clinic. The patients went home with the diluted solutions for application. The solution was applied 30 minutes before sun exposure to early morning sunlight (between 6am – 7am) for a period of 10 to 15 minutes.

For the patients with extensive disease, they were given oral PUVAsol (meladinine tablets). Each tablet has a strength of 10mg and the dose for each patient was calculated based on body weight at 0.2 to 0.4mg per Kg body weight. The tablets were taken one to two hours prior to sun exposure (early morning sunlight between 6am – 7am) for a period of 10 to 15 minutes. The patients were asked to wear dark goggles during sun exposure for ocular protection.

The duration of exposure to sunlight for all the patients was graded (reduced or increased) depending on the severity of erythema as the aim was to achieve erythema for a period not more than 24 hours. Patients were asked to reduce the time of sun exposure by five minutes if they developed severe erythema. Therapy was repeated on alternate days and outcome was checked at each return visit (every month) to the Dermatology clinic for a period of 6 months.

The global outcome of therapy for the patients was classified into three categories as follows: progressed (P), stable (S) and repigmented (R) after 6 months of therapy. The repigmented group was further classified into fair (R1) and good (R2) representing ≤50% and >50% repigmentation of the affected body surface areas.

Data was analyzed using the Statistical Package for the Social Sciences (SPSS) version 22.0.

RESULTS

Demographics

The age of the patients ranged from 18 years to 72 years and their mean age was 35 (±17) years. There were more females with vitiligo who were enrolled in the study as twenty patients (40%) were males while thirty (60%) were females. The occupational status of the patients revealed that most of the patients were farmers/ traders/ artisans (40%) and students (40%).For their marital status; twenty-six (52%) were married, twenty-one (42%) were single and three (6%) widowed. The distribution of the highest level of education attained by the patients revealed that eight patients (16%) had no formal education, seven patients (14%) had primary education, seventeen patients (34%) had secondary education and eighteen patients (36%) had tertiary education.

Table 1: Demographics (n=50).

Variables	Total N (%)
Age (years)	
<20	15 (30)
21-40	17 (34)
41-60	12 (24)
61-80	6 (12)
Sex	
Male	20 (40)
Female	30 (60)
Occupation	
Students	20 (40)
Civil servants	10 (20)
Traders	16 (32)
Farmers	2 (4)
Artisans	2 (4)
Marital status	
Single	21 (42)
Married	26 (52)
Widowed	3 (6)
Educational level	
None	8 (16)
Primary	7 (14)
Secondary	17 (34)
Tertiary	18 (36)
Duration of lesions	
≤2 years	23 (46)
>2 years	27 (54)
Family history of vitiligo	
Present	6 (12)
Absent	44 (88)
History of previous treatment	
Treatment experienced	18 (36)
Treatment naïve	32 (64)

Most of the patients who presented have had the disorder for more than 2 years (54%) and a few (36%) had applied topical steroids prior to presentation. A family history of vitiligo was also present in only 12% of the patients. These are depicted in Table 1.

Clinical characteristics

The distribution of disease spectrum in decreasing order of frequency is as follows: generalized vitiligo (56%), segmental vitiligo (24%), acrofacial vitiligo (14%),

mixed vitiligo (4%), focal vitiligo (2%). Most of the patients (60%) had limited disease with ≤20% of BSA affected and a few (14%) had leukotrichia. These are illustrated in Table 2.

Table 2: Clinical characteristics (disease spectrum, disease severity and presence of leukotrichia)

Variables	N (%)
Clinical spectrum	
Generalized	28 (56)
Acrofacial	7 (14)
Mixed	2 (4)
Segmental	12 (24)
Focal	1 (2)
%BSA affected	
≤20	30 (60)
>20	20 (40)
Leukotrichia	
Present	7 (14)
Absent	43 (86)

Outcome of treatment

Out of the fifty patients who participated in the study, forty six (92%) had repigmentation of their lesions and four (8%) had no repigmentation of their lesions. Among the patients whose lesions repigmented, forty-four patients(88%) had less than fifty percent repigmentation of the affected areas (R1) while two patients (4%) had more than fifty percent repigmentation of the areas affected (R2). Among the patients whose lesions did not repigment, one patient (2%) had progression of his lesions while three patients (6%) had stable lesions. These are shown in Tables 3 and 4.

Table 3: The outcome of treatment offered to the patients.

Treatment offered	Outcome	
	Lesions did not repigment	**Lesions partially or completely repigmented**
	N (%)	**N (%)**
Topical PUVAsol	3 (6)	27 (54)
Oral PUVAsol	1 (2)	19 (38)
Total	4 (8)	46 (92)

X^2=0.41, p=0.641.

Furthermore, with reference to the treatment groups, 27 (90%) out of the 30 patients with limited disease had some repigmentation of their lesions while 19 (95%) out of the 20 patients with extensive disease had some repigmentation of their lesions.

Table 4: Breakdown of the outcome of treatment offered to the patients.

Treatment options	P	S	R_1	R_2
	N (%)	N (%)	N (%)	N (%)
Topical PUVAsol	1 (2)	2 (4)	25 (50)	2 (4)
Oral PUVAsol	0 (0)	1 (2)	19 (38)	0 (0)
Total	1 (2)	3 (6)	44 (88)	2 (4)

P=Progressed, S=stable, R1=Repigmented≤50%, R2= Repigmented >50%; Fisher's exact test =1.93, p=0.869.

Table 5: Relationship between clinical spectrum of vitiligo and treatment outcome.

Spectrum of vitiligo	Treatment outcome	
	No repigmentation	**Partial/ complete repigmentation**
	N (%)	N (%)
Generalized	0 (0)	28 (56)
Acrofacial	0 (0)	7 (14)
Mixed	0 (0)	2 (4)
Segmental	3 (6)	9 (18)
Focal	1 (2)	0 (0)
Total	4 (8)	46 (92)

Fisher's exact test = 12.45, p=0.005.

Table 6: Effect of leukotrichia on treatment outcome.

Leukotrichia Treatment outcome	P	S	R1	R2
	N (%)	**N (%)**	**N (%)**	**N (%)**
Present	1 (2)	2 (4)	4 (8)	0 (0)
Absent	0 (0)	1 (2)	40 (80)	2 (4)
Total	1 (2)	3 (6)	44 (88)	2 (4)

Fisher's test =10.18, p=0.013.

Table 7: Effect of family history of vitiligo on treatment outcome.

Family history treatment outcome of vitiligo	P	S	R_1	R_2
	N (%)	**N (%)**	**N (%)**	**N (%)**
Present	0 (0)	0 (0)	6 (12)	0 (0)
Absent	1 (2)	3 (6)	38 (76)	2 (4)
Total	1 (2)	3 (6)	44 (88)	2 (4)

Fisher's test = 1.19, p=1.00.

Relationships between clinical variables and treatment outcome

The relationship between the spectrum of vitiligo and treatment outcome is highlighted in Table 5. It showed that repigmentation of the lesions occurred in all the patients with generalized, acrofacial and mixed vitiligo. Of the twelve patients with segmental vitiligo, nine (75%) of them had repigmentation of their lesions and

among the remaining three patients, two patients had stable lesions and one patient had progression of his lesions. The only patient with focal vitiligo had no repigmentation of lesions. There was a statistically significant correlation between the clinical spectrum of vitiligo and treatment outcome. Furthermore, it was discovered that 4 (57%) out of the 7 patients who had leukotrichia had repigmentation of their lesions while 42 (98%) out of the 43 patients who did not have leukotrichia had repigmentation of their lesions which was a statistically significant finding (Table 6). It is worthy of mention that all the patients who had leukotrichia had segmental vitiligo.

Table 8: Effect of duration of lesions on treatment outcome.

Duration of lesions treatment outcome	P	S	R_1	R_2
	N (%)	N (%)	N (%)	N (%)
Less than 2 yrs	1 (2)	1 (2)	19 (38)	2 (4)
More than 2 yrs	0 (0)	2 (4)	25 (50)	0 (0)
Total	1 (2)	3 (6.0)	44 (88)	2 (4)

Fisher's exact test = 3.48, p = 0.346

Table 9: Comparison of outcome of PUVAsol therapy between treatment naive and treatment experienced patients.

	Treatment outcome			
Treatment exposure groups	P	S	R1	R2
	N (%)	N (%)	N (%)	N (%)
Treatment naïve	1 (2)	3 (6)	26 (52)	2 (4)
Treatment experienced	0 (0)	0 (0)	18 (36)	0 (0)
Total	1 (2)	3 (6)	44 (88)	2 (4)

Fisher's test =2.96, P=0.38.

The effects of family history of vitiligo, duration of disease and history of previous treatment on treatment outcome were not statistically significant as shown in Tables 7-9 respectively.

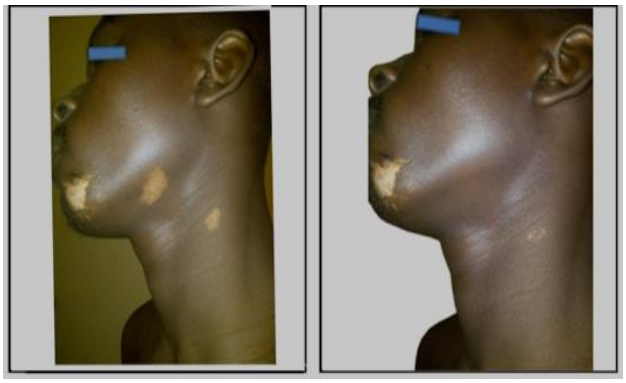

Figure 1: Partial repigmentation of the facial lesions after treatment for 6 months in a patient with NSV.

DISCUSSION

Vitiligo is a chronic depigmentary cutaneous disorder due to the loss of melanocytes in the skin which currently has no cure. The mainstay of therapy is to stop disease progression and induce repigmentation with the aim of achieving an acceptable cosmetic result.[12] The disorder could be socially embarrassing and cause psychological distress to those affected.[5] Therefore, the use of cosmetic camouflage and psychotherapy as adjunctive therapy is encouraged.

The choice of agents used for therapy should be based on various factors such as age of the patient, stability of the disease, duration of treatment, side effects, cost and accessibility of medication. However, cost and accessibility should be of priority especially in limited resource settings as in this study. It is worthy of mention that most of the patients in this study were small scale farmers, traders, artisans and students who could barely afford the cost of transportation to the hospital. As such, the burden of any treatment modality that would incur much more finances would not be acceptable to the patients. In the light of these, PUVAsol was chosen as the treatment modality because it was readily available and inexpensive.

Although the exact mechanism of action of PUVA in melanogenesis is not yet known, it has been postulated that psoralens penetrate the melanocytes and intercalate between Deoxyribonucleic Acid (DNA) base pairs. On exposure to UVA, psoralens absorb photons, get activated and covalently bind to DNA base pairs forming crosslinks. The DNA crosslinks have antiangiogenic, antiproliferative, apoptotic, and immunosuppressive effects. The immunosuppressive effects include alteration in cytokines and antigen presenting cells with reduced expression of adhesion molecules and lymphocyte apoptosis.[12]

Most of the patients have had the disorder for more than 2 years before presenting to the teaching hospital. This was largely because they did not have knowledge of the disorder and sought help at either local patent medicine shops and unorthodox health centres. A few went to nearby district and general hospitals and were given topical corticosteroids by the general practitioners. This underscores the need for improved health education about pigmentary skin disorders both to the general population and General Practitioners so that patients could be referred promptly to a Dermatologist. There is also a need for more Dermatologists to be trained and employed so that they could offer their expertise services at rural communities.

In this study, which was for a period of 6 months, the results obtained with PUVAsol was quite encouraging as 46 out of the 50 patients who participated in the study had repigmentation of their lesions, though 44 patients had less than 50% of the areas affected repigmented

which was a fair outcome and 2 patients had more than 50% repigmentation of their lesions. Among the 44 patients who had less than 50% repigmentation of their lesions, 27 patients had limited disease and received topical PUVAsol while 19 patients had extensive disease and received oral PUVAsol. These findings are similar to the results in a previous study on treatment options for vitiligo by Handa et al.[18] There is a strong possibility that if therapy is continued for a longer period of time that the level of repigmentation that would be obtained in both groups would be much more. It is thus suggested that further studies with an extension in the treatment duration with PUVAsol should be done and also compare with other treatment modalities.

The patients with NSV (generalized, acrofacial and mixed) showed better response to therapy than those with SV. The loss of melanocytes in patients with NSV has been largely postulated to be due to the inflammatory response from cellular and humoral auto-immune process against melanocytes while the neural theory has been postulated to be responsible for melanocyte loss in SV.7 Interestingly, ultraviolet (UV) light therapy has been shown to possess both innate and cellular immunosuppressive as well as mitogenic and melanogenic properties that promote melanocyte proliferation and melanin synthesis which makes it a viable option for NSV.[19] Furthermore, a study by Anbar et al showed that UVA light therapy combined with psoralen (PUVA) reversed melanocyte and keratinocyte degeneration in and around lesions of NSV.[20] The lesions on the face also had the best response to treatment in comparison with other body sites (Figure 1)

The presence of leukotrichia, which was found only in patients with SV was associated with poor treatment outcome in this study. A previous study by Lee et al revealed a similar outcome.[21] Hence, it is essential to counsel patients with leukotrichia on the possible outcome prior to PUVAsol therapy or offer them other treatment options.

In this study, there was no relationship between family history of vitiligo and treatment outcome which was the case in a previous study. Furthermore, vitiligo patients with a family history have a tendency for disease progression if untreated and prompt referral to a Dermatologist is advised.

Patients with disease of recent onset have been shown to have the best treatment outcomes.[12] This was the case here as the only patients who had >50% repigmentation of their lesions following therapy presented within 2 years of disease onset. Although the finding was not statistically significant, a study with a larger sample size would be required to substantiate it.

All the 18 patients who were treatment experienced had repigmentation of their lesions while 28 (88%) out of 32 patients who were treatment naïve had repigmentation of their lesions. The treatment experienced patients received topical corticosteroids prior to commencement of PUVAsol. It has been documented that combination therapy achieves better results than monotherapy[12] and this could be responsible for the difference in the outcome between the two groups though the drugs were administered sequentially not simultaneously.

CONCLUSION

PUVAsol is a treatment modality for vitiligo with some repigmentation of lesions following therapy for 6 months. Outcomes are better in patients with NSV and could improve if duration of treatment is extended. PUVAsol therapy could be recommended in resource poor settings because of its low cost and availability in most parts of Nigeria.

Limitations

There was lack of standardization of therapy as the amount of UVA radiation could not be estimated.

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