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Brief communication

Ultraviolet A in vitiligo

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Both types of Ultraviolet (UV), UVB (290–320 nm) and UVA (320–400 nm), produce increased pigmentation or tanning. However, no evaluation of UVA alone in the treatment of vitiligo has been reported. Therefore, it was the purpose of this work to study the pigmentogenic effect of UVA (5 and 15 J/cm^2) in vitiligo. The study included 20 randomly selected patients with vitiligo involving more than 30% of the body surface area with a bilateral/symmetrical distribution. They were equally divided into two groups each of 10 patients. All patients received three weekly sessions of UVA, 15 J/cm^2 in group I and 5 J/cm^2 in group II, a total of 48 sessions over 16 weeks. Overall pigmentation of 60% and above was recorded in 50% and 10% of patients in groups I and II, respectively. We conclude that broadband UVA alone, without psoralens, and in appropriate doses may be of important therapeutic value in vitiligo.

Key words: **vitiligo; UVA; treatment.**

Among the many lines of treatment used in the management of vitiligo, psoralen and ultraviolet A and narrow-band 311 UVB (NB-UVB) are well documented.

When solar exposure of the skin occurs, a biphasic process takes place. First, is the immediate pigment darkening (IPD) caused by UVA, which starts seconds after exposure and fades within minutes. If large or continuous doses are given, pigmentation will persist for several days and will blend with the second pigmentary stage, namely delayed tanning (DT). This is usually produced by UVB, appearing 72 h after exposure and fading away gradually. IPD is caused by oxidation and redistribution of already present melanin, whereas DT is caused by the formation of new melanin (1). So, it is clear that UVA may produce both types of pigmentary responses if sufficiently large or continuous doses are given. These large doses were suggested to range from 8 to 25 J/cm^2 (2), leading to persistent pigment darkening.

Among the other differences that exist between the pigmentary skin response to UVR, UVB was found to produce more erythema than tanning in fair-skinned individuals (skin types I and II), while UVA will produce more tanning than erythema in dark-skinned individuals (skin types III and IV) (1).

Both types of UV will produce increased number and activity of melanocytes, increased melanin density, elongation and branching of dendrites, ending by increased transfer of more heavily melanized melanosomes to keratinocytes, which is seen by the naked eye as increased pigmentation or tanning (1, 3).

The aim of this work is to study the pigmentogenic effect of UVA in vitiligo using two different doses, namely, 5 and 15 J/cm^2.

Patients and methods

Our study included 20 patients with generalized vitiligo (17 females and three males), who had not received local or systemic treatment for at least 1 month, with a mean age of 28 years. Patients were randomly divided into two groups each of 10 patients. Group I received UVA (15 J/cm^2) and group II received UVA (5 J/cm^2) in each session throughout the study. They were subjected to a total body broadband UVA (BB-UVA) phototherapy unit (UV1000 Waldmann lighting, Villingen Schwenningen, Germany) equipped with 26 UVA lamps emitting a radiation spectrum of 315–400 nm with a peak of 365 nm. Patients received therapy three times weekly for 16 weeks (a total of 48 sessions). Patients were

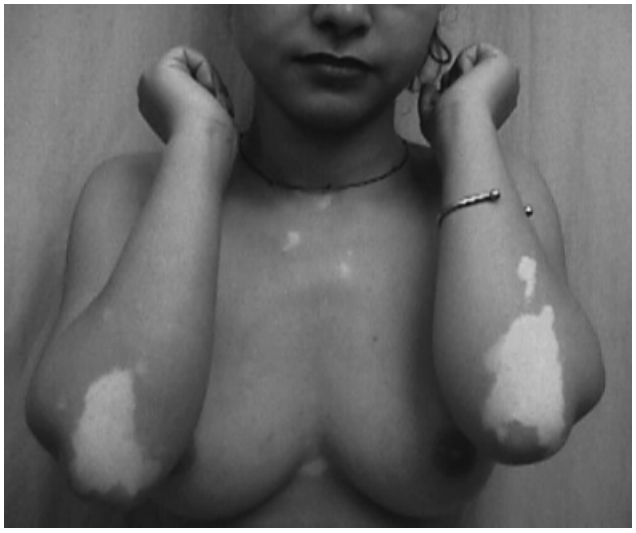

Fig. 1. Lesions on elbows before treatment using UVA 15 J/cm^2.

evaluated every 2 weeks and photographs were taken at sessions 0, 24 and 48 (Fig. 1).

Results

Perifollicular and/or peripheral pigmentation were considered as an initial response. Treatment failure was considered when no initial response occurred up to the 24th session (Table 1).

Percentage of good and very good responders (>60% overall re-pigmentation) at 48 sessions was in 50% of patients in group I and 10% in group II (Table 1).

Discussion

To our knowledge, no studies evaluating UVA in vitiligo were reported in the literature. However, cell culture studies on normal human melanocytes showed that only a high dose of UVA (7.2 J/cm^2) induced slight up-regulation of the activation marker HMB-45 with an increase in melanin content and down-regulation of the proliferation marker Ki-67 (4). This could explain the better therapeutic efficacy of the higher dose of UVA (15 J/cm^2) than the lower dose (5 J/cm^2) obtained in this study. The 5 J/cm^2 dose was chosen to try to find the least possible dose that could be effective, whereas the 15 J/cm^2 dose was chosen because it is a moderately high dose.

One would expect that BB-UVA would have a sufficient pigmentogenic effect. This could be explained by the fact that the wave bands 320–340 nm (UVA2) induce pigmentation through DNA damage similar to the UVB mechanism, while the rest of the spectrum, 340–400 nm (UVA1), induces pigmentation through a different mechanism that is oxygen dependent (5). Therefore, BB-UVA would probably induce pigmentation by the two mechanisms.

Table 1. Comparison of different patients' responses after 48 sessions of phototherapy regimens in 20 vitiligo patients

	Treatment Failure (24 sessions)	Poor 0–40%	Moderate 40–60%	Good and very good >60%
Group 1	2 (20%)	2 (20%)	1 (10%)	5 (50%)
Group 2	3 (30%)	4 (40%)	2 (20%)	1 (10%)

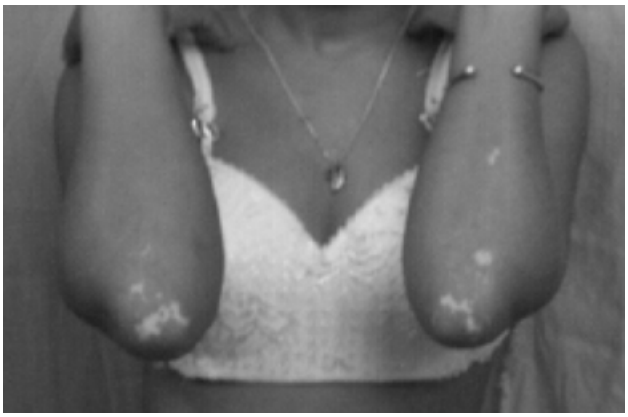

Fig. 2. Lesions on elbows after 48 sessions of UVA 15 J/cm^2.

The 60% success of BB-UVA therapy in re-pigmenting vitiligo patients by three weekly sessions at a dose of 15 J/cm^2 over 16 weeks is described here (Fig. 2).

We may conclude that the use of UVA 15 J/cm^2 may be of important therapeutic value in vitiligo, thus sparing the side effects of added psoralen and can be given for relatively shorter periods, thus decreasing any potential side effects.

Further studies could also follow to find out the least dose trying 8, 10, 12, 17 and 20 J/cm^2 that would give the best response. Two sessions weekly could also be tried and compared with the usual three-sessions weekly regimen. The duration of treatment could also be studied to compare the best results after 6, 8 and 12 months.

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