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The Role of Vitamin D3 in Skin Pigments

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ABSTRACT---*The relationship of Vitamin D3 deficiency and increased melanin production that leads to appearances of facial pigments such as melasma, unevenness and hyper pigmented patches. Sample size was 28 individuals, the study was comparative study. Group A was 9 individuals with real Vitamin D3 deficiency < 12 ng/ml according to WHO definition, group B was 13 individuals which normal Vitamin D3 level, 6 individuals didn't complete the study. The study lasts for 5 months from May to October 2019. Follow up visits on regular baseline, one after 3 weeks, one after another 3 weeks and the last visits after completion the 12 weeks. The results of a response to the current study showed that the response rate to Vitamin D therapy was very good compared to the rest of the subjects and for all groups. Where the non-response rate was zero, and the poor response rate was from 10 %, then above, the average response rate was from 50 % to 60 %, while the good rate was from 60 % to 70 %, while the highest response rate was from 80 % and above and in the figure 1 explains the difference between before and after treatment. Investigation had been done. It concluded that Vitamin D3 has an effect on improving and treating skin pigments.*

Keywords---*pigments, melasma, Vitamin D3 level, hydroquinone elements, azelic acid cream, sunblock and glycolic acid.*

I. INTRODUCTION

Pigmentation means coloration. Disorders of skin coloration affect skin color. Your skin gets its color from a pigment called melanin, which is produced by special skin cells. When these cells are damaged or diseased, melanin production is affected. Some pigmentation disorders affect only certain regions of the skin. Others affect the entire body. Melasma is a common skin problem. The condition causes dark, discolored patches on your skin. It's also called chloasma, or the "mask of pregnancy," when it occurs in pregnant women. The condition is much more common in women than men, though men can get it too. According to the American Academy of Dermatology, 90% of people who develop melasma are women. Melasma causes patches of discoloration. The patches are darker than your usual skin color. It typically occurs on the face and is symmetrical, with matching marks on both sides of the face. Other areas of your body that are often exposed to sun can also develop melasma. Vitaminamin D insufficiency (25-hydroxyVitaminamin D3 (25(OH)D) o50 nmol l 1) is common and is related to rachitis and osteoporosis, and may increase the risk of some internal malignancies and autoimmune diseases (Chapuy et al., 1992; Ahonen et al., 2000; Munger et al., 2004; Ponsonby et al., 2005; Kemp et al., 2007). UV radiation of the skin

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provides 90–95% of the total Vitaminamin D requirements for the majority of the population (Holick, 1998, 2004). Vitaminamin D insufficiency (Holick, 2008; Moan et al., 2008); and this has led to scientific debate (Samanek et al., 2006; Gilchrest, 2008). Studies suggest a faster Vitaminamin D production among individuals with a very low Vitaminamin D level (Viljakainen et al., 2006; Binkley et al., 2007; Brustad et al., 2007; Edvardsen et al., 2007), and skin pigmentation is generally con- sidered to diminish Vitaminamin D production from sun exposure (Holick et al., 1981; Clemens et al., 1982; Chen et al., 2007). However, these theories arenot well documented (Young, 2006).

II. MATERIALS & METHODS

Methodology

Duration 5 months from May to October 2019, while duration of treated was 12 weeks.

Samples size

Twenty eights participations divided to three groups

- 9 with Vitamin D3 deficient
- 13 with normal Vitamin D3 level
- 6 skipped

Inclusion criteria

Inclusion criteria for all participants age groups from 15 – 50 years divided between females and males, all materials statue single, married and widow.

Exclusive criteria

The participants were not admitted to the study if they had age < 15 or >50, Recent pigment < 1 month, Skin type I, II, and VII, pregnancy and nursing women.

Treatment

Group A : 12 weeks by foamer G.A (glycolic acid) 15 %, Hydroquinone cream 4 %, Azelic acid 15 %, Sunblock, Vitamin D3 50.000 l/week for 6 weeks then 1000 mg/d for a last 6 weeks and encourage taking food rich with Vitamin D3 such as salmon, tuna, fish, dairy products, liver, egg yolk and mushroom.

Group B: same Rx without Vitamin D3 for 12 weeks

Design:

P : patients with Vitamin D3 deficient and pigments

I : Vitamin D3 replacement

C : patients with normal Vitamin D3 level and pigment

O : improving the pigments

Regular visits

Visit 1: after 3 weeks from the starting of R_X

Visit 2: after 6 weeks from the starting of R_X

Visit 3: after 12 weeks (completion of R_X duration)

With clinical assessment, photo in each visits and check Vitamin D3 level at the end of R_X

Statistical analysis

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The data were statistically handled using SPSS 17.0 for Windows (SPSS, Chicago, IL).

III. RESULTS

The results of a response to the current study showed that the response rate to Vitaminamin D therapy was very good compared to other subjects and for all groups. Where the non-response rate was zero, the poor response rate was from 10% and above, and the average response rate was from 50% to 60%. The good response was from 60% to 70%. While the Excellent response was above 80 % .

Table 1: explained ratio of types response of treated with Vitamin D3

No.	Type of response	Number and %responses
1	No response	-----
2	Poor response	10 - <50
3	Fair response	50-60
4	Good response	70-80
5	Excellent response	< 80

Group A : 12 weeks by foamer G.A (glycolic acid) 15 %, Hydroquinone cream 4 %, Azelic acid 15 %, Sunblock, Vitamin D3 50.000 l/week for 6 weeks then 1000 mg/d for a last 6 weeks and encourage taking food rich with Vitamin D3 such as salmon, tuna, fish, dairy products, liver, egg yolk and mushroom.

Group B: same Rx without Vitamin D3 for 12 weeks

Distribution of 25(OH)D levels for the 28 participants at the initial screening. 67% Of participants wereVitamin D insufficient (25(OH)Do50 nmol/l), and 18% were Vitaminamin D deficient

(25(OH)Dp25 nmol/l). The dark and light grey lines show cutoffs at 25 nmol/l and 50 nmol/l, respectively. participants with a wide range in baseline 25(OH)D levels When the influence of baseline 25(OH)D level on Vitaminamin D production after exposure to UV was examined, participants with all levels of Vitamin D3due to previous different sun exposures were included: with an insufficient Vitamin D3 level (evenly distributed 25–50 nmol l 1), with a deficient Vitamin D3 level (p25 nmol l 1), and with a highly sufficient 25(OH)D level (470 nmo l 1). As the level ofbaseline Vitamin D3is expected to be of importance to the baseline parathyroid hormone (PTH) level, this was also examined in this group.

However, we found a positive linear relation between baseline Vitamin D3levels and the number of fish meals per week (P ¼ 0.009; R2 ¼ 0.132), with the baseline VitaminD3 level being 12 nmol l 1 higher in the group eating fish more than once a week.

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Vitamin D3level (P ¼ 0.018) between the 28 non-sun worshippers and the 22 sun worshippers due to different amounts of previous sun exposure. In the selected 28 non-sun worshippers (group 2), Vitamin D3 levels increased by (mean) (SD) 25.3 nmol l1 (10.5) in response to the UVB treatments. No significant correlations were found between VitaminD3 and constitutive (PPF buttock) (P ¼ 0.5) or facultative (P ¼ 0.4) skin pigmentation. There were no significant relations between D25(OH)D and age, height, weight, numbers of fish meals per week, body mass index, PTH, alkaline phosphatase, ionized calcium, or skin redness percent after exposure. The facultative skin pigmentation increased, and the skin redness percent increased during the course of UVB treatments.The results of the images in Figure 1 showed that there is a clear difference before and after treatment.

Fig 1: explain before and after treatment

IV. DISCUSSION

In total, 80% of the 28 participants had Vitamin D3levels below 50 nmol l 1, and 18% were below 25 nmol l 1. The recommended level of Vitamin D3 is estimated to be 50 nmol l 1 or higher (Brot et al., 2001;Lips, 2004; Mosekilde et al., (2005). However, it is debated whether higher Vitamin D3levels of 80–100 nmol l 1 are preferable (Holick, 1998; Heaney, 2005; Viet, 2006). To strengthen our findings, we included detailed information on lifestyle factors, collected information on intakes of calcium and Vitamin D, included two serum samples from each subject, measured pigmentation, and conducted the study when the ambient UV radiation was negligible and the blood concentration of Vitamin D was considered as the year's lowest in Denmark 561N. Furthermore, we selected a homogeneous population of 28 non-sun worshippers (group 2) with limited past sun exposure, because differences in sun habits is a confounding factor when analyzing the influence of skin pigmentation, UVB radiation was used as it fits the Vitamin D action spectrum and uniform irradiation of large body surface areas can be obtained. A

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limitation of this study was the relatively small sample size, especially in the dark-skinned group. Furthermore, we found a high variance in the liquid chromatography-tandem mass spectrometry analysis of Vitamin D3 of B8.5%, despite including two serum samples from each subject and performing each Vitamin D3analysis twice.

Among the 50 participants in group1, we found a significantly higher 25(OH)D production after UVB exposure in participants with a low baseline Vitamin D3level. Although not specifically exploring the mechanism, several studies have reported that individuals with low baseline Vitamin D are the most effective at increasing serum levels of Vitamin D after UVB exposure (Viljakainen et al., 2006; Binkley et al., 2007; Brustad et al., 2007; Edvardsen et al., 2007; Stephenson et al., 2007; Carbone et al., 2008). The reasons for this are unknown, but it may be because 25(OH)D inhibits 25-hydroxylase in the liver; the liver prompts hydroxylation of Vitamin D3 to Vitamin D3(Champe et al., (2005). Among the 28 non-sun worshippers in group 2, we found no significant relation between DVitamin D3and constitutive or facultative skin pigmentation.

no significant differences in D25(OH)D between the dark- skinned persons and the 25(OH)D baseline-matched fair- skinned persons (group 3) despite their highly significant difference in constitutive and facultative skin pigmentation. This shows that D25(OH)D is unrelated to skin pigmentation. A number of very small studies in Vitro (Holick et al., 1981) and in vivo (Clemens et al., 1982; Chen et al., 2007) indicate that melanin pigmentation diminishes Vitamin D production in the skin.

Conversely, Brazerol et al. (1988) found a similar Vitamin D increase in a fair-skinned group (n ¼ 13) compared with a dark-skinned group (n ¼ 7) exposed to suberythemal whole- body UVR twice a week for 6 weeks. Furthermore, a study of Rockell et al. (2008) found that only facultative skin color is a determinant of Vitamin D production and claims that constitutive skin type isof no significant importance. In addition, Marks et al. (1995) reported no significant relations between skin type and Vitamin D production. There is an obvious lack of agreement about the role of skin pigmentation in Vitamin D production after UVB exposure, and skin pigmentation was not measured in any of the studies. However, several studies have reported a lower level of Vitamin D among dark-skinned than among fair-skinned individuals (Harris and Dawson-Hughes, 1998; Nesby- O'Dell et al., 2002), and it might be possible that Vitamin D insufficiency amongcertain ethnic groups results from other factors than skin pigmentation such as behavior or diet. We found a remarkable difference in sun habits between the dark-skinned group, who were non-sun worshippers with limited levels of sun exposure, and the fair-skinned group, in which the main part was sun worshippers with excessive levels of sun exposure. These differences in sun habits may explain why dark-skinned persons are reported to have lower Vitamin D levels than fair-skinned persons (Harris and Dawson-Hughes, 1998; Nesby-O'Dell et al., 2002, Young, 2006). Although we found no differences in VitaminD formation between dark- and fair-skinned persons during wintertime, when melanin is located in the basal layers of epidermis, a relation could exist in the summertime, when melanin moves further up in epidermis due to sun exposure. Another explanation could be that 3 standard erythema doses (SEDs) administered four times to 24% ofthe body surface area is sufficient to reach a state of saturation. Notably, wherein it is isomerized to pre-Vitamin D3 (cholecalciferol) by UVB radiation (Champe et al., 2005). However, there is no indication of a fall in Vitamin D status afterstatin therapy (Dobs et al., 1991; Pe´ rez-Castrillo´ n et al., (2007). Statin therapy inhibits the enzyme hydroxymethylglutaryl-CoA (HMG-CoA) reductase (Champe et al., 2005). Our study shows the ratio of response of

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treated of the pigments of skin its above 80 % its response with the Vitamin D 3. However, due to its skin-carcinogenic effect, we would not recommend UVB treatment to be used as a source of Vitamin D to the general population, because sufficient Vitamin D levels can be re-established with Vitamin D supplements (Gilchrest, 2007; Hathcock et al., 2007; Thieden et al., 2008).

In conclusion, we found that baseline Vitamin D level is an important determinant of Vitamin D production after UVB treatment. We also found that constitutive or facultative skin pigmentation in winter is of no importance for the Vitamin D production in the skin.

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