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Evaluation of serum levels of vitamin e and vitamin a in childhood vitiligo patients

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Abstract
Background: Vitiligo is a common acquired hypopigmentation disorder of the skin. Oxidative stress plays a key role in its pathogenesis by damaging melanocytes. Vitamins A and E known antioxidantsوص may be involved, but studies on their levels in vitiligo patients show conflicting results. So, the aim of this work was to evaluate the serum levels of vitamin E and vitamin A in childhood vitiligo to determine their possible roles in disease pathogenesis.
Methods: This case control study was carried out on 40 individuals: 20 patients aged less than 18 years old, both sexes, with non-segmental Vitiligo who didn't receive any kind of treatment of vitiligo for at least 3 months before the study and 20 healthy individuals with matching age and sex.
Results: There was a negative correlation between the patients' ages and their serum levels of vitamin E and A, as well as a non-significant correlation between the Vitiligo Disease Activity Index (VIDA score) and serum levels of vitamin E and A and the Vitiligo Area Scoring Index (VASI score).
Conclusions: Serum levels of vitamin A and vitamin E did not significantly differ between children with vitiligo and healthy controls, indicating that these vitamins may play a small part in the pathophysiology of the condition in children. Age and vitamin levels were found to be negatively correlated, which may be because of variations in metabolism and oxidative stress. Higher vitamin levels were observed in patients with less severe vitiligo, albeit these differences were not statistically significant. This suggests that antioxidants may have a part in lessening the severity of the condition.

Keywords: Vitamin E, Vitamin A, Childhood, Vitiligo

Introduction
The skin, mucous membranes, and retina are all affected by vitiligo, a common acquired hypopigmentation condition [1]. A well-defined, amelanotic macule that is round, oval, or irregular and ranges in diameter from a few millimeters to several centimeters is the usual appearance of vitiligo patches. Anywhere on the body, they can show up, although they tend to show up on traumatized areas including the face, hands, feet, fingers, elbows, knees, and anogenital area [2]. The head and neck area, particularly the eyelids, is where 31–59% of children with vitiligo initially experience the disorder [3-6]. Nonetheless, the most commonly impacted parts in maturity are the hands and fingers, perhaps due to koebnerization [5]. Because it affects the melanocytic reservoir in the hair follicles, leukotrichia, which has been seen in 3.7% to 32.5% of children with vitiligo, may be connected to childhood vitiligo [3, 4, 6].

Vitiligo begins in childhood in about one-third to half of cases and affects up to 2.16% of the global population [7, 8]. Early-onset vitiligo (Before age 12) is often linked to halo nevi, Koebner phenomenon, family history, segmental patterns, or atopy, while later onset (after age 12) is more associated with acrofacial lesions and thyroid disorders [9].

All age groups, races, and sexes are affected by vitiligo, although adult African females are more likely to have it [10]. Patients' quality of life is significantly impacted by this condition [11].

It is yet unknown what causes vitiligo [12]. It has been determined that oxidative stress plays a significant role in the pathogenesis of vitiligo [13]. According to certain research, vitiligo patients' melanocytes have compromised antioxidant function [14], which leads to free radical-induced melanocyte destruction [15]. In individuals with vitiligo, oxidative stress is commonly observed at both the systemic and local levels. It can provoke an autoimmune

response, which in turn leads to the loss of melanocytes [16, 17]. Because of this close connection, vitiligo patients who are remitting may be permanently at risk of recurrence due to an imbalance in their antioxidant system and the oxidative stress that results [18].

Serum levels of antioxidant vitamins, such as A and E, have been examined between vitiligo sufferers and healthy controls in a number of studies, although the findings are still mixed [12].

The lipid-soluble vitamins A and E are vital components of the diet [19, 20]. Retinol and retinyl esters are the most prevalent forms of vitamin A found in the human diet [19]. Through its metabolite, retinoic acid [21], vitamin A regulates the immunological response. Retinoic acid receptors affect the expression of target genes [21, 22]. Increased vulnerability to inflammation and skin infections has been associated with vitamin A deficiency [23, 24]. This vulnerability has raised the possibility that vitamin A plays a crucial part in supporting skin immunological activity [25]. It has been demonstrated that vitamin E is dispersed throughout the body and in plasma [26] making it a necessary and advantageous nutrient in many areas of health [27]. Vitamin E is one of the primary components of non-enzymatic antioxidant system. Chronic inflammatory skin illnesses have been linked to abnormal variations in vitamin E levels in the serum [28]. This raised awareness of the need of vitamin E for people with vitiligo [18].

In order to ascertain their potential roles in the pathophysiology of the disease, the objective of this study was to assess the serum levels of vitamin E and vitamin A in children with vitiligo.

Patients and Methods

20 vitiligo patients under the age of 18, both sexes, with non-segmental vitiligo who had not had any form of treatment for at least three months before to the trial, and 20 healthy participants of the same age and sex participated in this case control study. The study met all ethical requirements for human research and was approved by Tanta University's Faculty of Medicine's Research Ethics Committee (approval code: 35290/2/22). Family members of the patients gave their signed, informed consent.

Patients with a history of other skin conditions as well as those with systemic illnesses or undergoing systemic treatment were excluded.

General examination, regular investigations, dermatological examination (clinical, Wood's light, and dermoscopy), clinical scoring of vitiligo patients (Vitiligo Disease Activity Index (VIDA) and Vitiligo Area Scoring Index (VASI scores), and a thorough history taking were all performed on each patient.

Vitiligo Disease Activity Index (VIDA) [29]

Based on the patient's assessment of how recently the disease has advanced, the VIDA score is a six-point rating system that gauges vitiligo activity. While lower values (0 to −1) show long-term stability or repigmentation, higher scores (+4 to +1) show more recent activity.

Vitiligo Area Scoring Index (VASI) [30]

The Vitiligo Area Scoring Index (VASI) is a quantitative parametric score that is theoretically developed from the PASI score, which is commonly used in the assessment of psoriasis. A method that incorporates contributions from everybody region (possible range: 0–100) is used to compute the total body VASI.

VASI $=\sum_{\text{All Body Sites}}$ [Hand Units] x [Residual Depigmentation].

Each finger's palm and volar surface together make up 1% of the body's surface area and are utilized as a single unit to determine the degree of vitiligo.

The trunk, lower extremities (including the buttocks and groin regions), hands, upper extremities (including the axillae), and feet are the five distinct sections of the body that are evaluated**.** These percentages indicate the degree of residual depigmentation: 0, 10%, 25%, 50%, 75%, 90%, or 100%. There is no pigment at 100% depigmentation; there are pigment specks at 90%; the depigmented area is larger than the pigmented area at 75%; the depigmented and pigmented areas are equal at 50%; the pigmented area is larger than the depigmented area at 25%; and there are only depigmentation specks at 10%.

Digital colored photographs restricted to sites of lesions were taken. Photographic evaluation was taken at the same equipment, lighting and location using a 48 mega pixels AI mobile camera (Samsung Note 9) made in China.

ELISA Assay

A volume of 3 ml venous blood was collected from all participants in sterile tubes and was subjected to ELISA Assays

(VE) ELISA Kit Instruction

Catalogue No: 201-12-1548(48T)

Test principle

The kit uses an enzyme-linked immunosorbent assay with a double-antibody sandwich to determine the level of human vitamin E (VE) in samples. [31] Vitamin E is first added to wells coated with monoclonal anti-VE antibodies in order to detect it using this technique. Streptavidin-HRP and biotin-labeled anti-VE antibodies are added to create an immunological complex following incubation and washing. Following the application of chromogen solutions, the addition of acid causes the color to shift from blue to yellow. The concentration of vitamin E in the sample is directly correlated with the yellow color's intensity.

Standard dilution

This test kit was supplying one original Standard reagent. Table 1

Table 1: Standard dilution

Concentration (µg/ml)	Standard No.	Preparation
4.8	5	120 µl original standard + 120 µl diluent
2.4	4	120 µl of Standard No.5 + 120 µl diluent
1.2	3	120 µl of Standard No.4 + 120 µl diluent
0.6	2	120 µl of Standard No.3 + 120 µl diluent
0.3	1	120 µl of Standard No.2 + 120 µl diluent

The number of plates is determined by the standards and the amounts of samples that will be analyzed. Duplicating each standard and blank well is advised. Each sample will be created in the number you specify, and every effort will be made to make the best use of the duplicates.

Inject specimens

In the blank well, add only Chromogen A, B, and stop

solution; skip sample, biotin antibody, and streptavidin-HRP.
Sample 40P 1 should be added to test wells, followed by VE antibody 10 µ 1 and streptavidin-HRP 50P 1. After that, incubate for 60 minutes at 37 °C while gently shaking the sealing memberance.
Distilled water should be used to dilute the 20X wash buffer 1:20. Tap off any remaining drips and discard the liquid after incubation. Each well should contain 50 µL of Chromogen Solutions A and B. Gently stir, then incubate for 10 minutes at 37°C in the dark. To turn each well from blue to yellow, add 50 µL of Stop Solution. After 15 minutes, use the blank as a reference to measure the OD at 450 nm. To generate a standard curve and a linear regression equation for analysis, plot OD against known concentrations.
Lastly, use this equation to calculate the vitamin E concentrations of unknown samples by using their OD values. Software or manual labor can be used for this.
Sensitivity: 0.031 mg/dl. Assay range: 0.05 mg/dl—>9mg/dl.

(VA) ELISA Kit Instruction
Catalogue No: 201-12-1549(48T).
Test principle: The kit determines the amount of human vitamin A (VA) in samples using a double-antibody sandwich enzyme-linked immunosorbent assay [31]. In order to detect human vitamin A (VA), this ELISA method first coats wells with anti-VA monoclonal antibodies and then incubates them. After that, streptavidin-HRP and biotin-labeled anti-VA antibodies are added to create an immunological complex. Chromogen Solutions A and B are added after washing, turning the solution blue. When acid is added, the solution turns yellow. The amount of vitamin A present in the sample is closely correlated with the intensity of the yellow color.

Table 2: Materials supplied in the Test Kit

1	**Standard(480 pg/ml)**	0.5 ml
2	Standard diluent	3 ml
3	Microelisa Strip plate	12 wellX4 strips
4	Str- HRP-Conjugate Reagent	3 ml
5	20Xwash solution	20 ml
6	Biotin- VA Ab	1 ml
7	Chromogen Solution A	3 ml
8	Chromogen Solution B	3 ml
9	Stop Solution	3 ml
10	Instruction	1
11	Closure plate membrane	2
12	Sealed bags	1

Assay procedure
Standard dilution: This test kit was supplying one original Standard reagent

Table 3: Standard dilution

Concentration (µg/ml)	Standard No.	Preparation
240	5	120 µl original standard + 120 µl diluent
120	4	120 µl of Standard No.5 + 120 µl diluent
60	3	120 µl of Standard No.4 + 120 µl diluent
30	2	120 µl of Standard No.3 + 120 µl diluent
15	1	120 µl of Standard No.2 + 120 µl diluent

The quantity of test samples and standards determines how many plates are required. Running each standard and blank in triplicate is advised. To guarantee accuracy and dependability, samples should also be prepared in the necessary quantity, ideally using duplicate wells.

Inject samples
Blank wells in this ELISA procedure for vitamin A (VA) only get the stop solution and Chromogen A/B; no sample, antibody, or enzyme is added. Streptavidin-HRP and 50 µL of standard are added to standard wells (biotin antibody is pre-included). Following incubation, test wells are filled with 40 µL of sample, 10 µL of biotin-labeled VA antibody, and 50 µL of streptavidin-HRP. Add Chromogen A and B after washing, let it sit in the dark, and then use acid to terminate the reaction (the color changes from blue to yellow). OD at 450 nm should be measured in 15 minutes. Calculate sample concentrations using the regression equation or curve after plotting a standard curve (concentration vs. OD). Analysis can be done with software. Sensitivity: 1.332 µg/ml, Assay range: 2 µg/ml—»400 µ/ml

Statistical analysis
The statistical analysis was conducted using SPSS v26 (IBM Inc., Chicago, IL, USA). Mean± SD was used to express quantitative data, and the unpaired Student's t-test was used for comparison. Chi-square or Fisher's exact test was used to examine the qualitative data, which were displayed as frequency (%) and were considered significant if $p < 0.05$.

Discussion
In average, between 0.5 and 2% of persons have vitiligo, the most common depigmentation skin disorder. Clinical symptoms are caused by the decomposition and death of pigment cells. These processes result in various types of depigmented skin patches. Their most common locations are the face, the backs of the hands, and the breast, axillary, sacral, inguinal, and anus areas.
The clinical course is the primary basis for diagnosing vitiligo. Only to rule out the associated conditions are laboratory tests and expert consultations required [32]. In order to determine the amount of skin lesions, Wood's lamp proved useful in diagnostics, particularly for individuals with light complexions [33].
Vitamin E is a vital and advantageous component that is becoming more and more well-known in the skin care industry. Alpha, beta, gamma, and delta are the four isomers of vitamin E, which are separated into two groups: tocopherols and tocotrienols. Vitamin E was found to be more uniformly distributed throughout the body, particularly in the plasma, than the body's exogenous lipophilic vitamins [34]. Chronic inflammatory skin illnesses have been linked to abnormal alterations in serum vitamin levels [35].
Since vitamin A is regarded as a family of necessary, fat-soluble dietary chemicals that share structural similarities with the lipid alcohol retinol, the current study assessed vitamin A blood levels in vitiligo cases. Additionally, vitamin A is necessary to preserve the integrity of the body's epithelial cell barriers and immune system. [36].
Furthermore, a prior study that combined oral vitamin A and E administration found that this combination was linked to improved vitiligo spot re-pigmentation outcomes [37]. Further research is need to validate these results, though. In

order to ascertain their potential roles in the pathophysiology of the disease, the purpose of this study was to assess the serum levels of vitamin E and vitamin A in children with vitiligo.

Twenty children with non-segmental vitiligo, under the age of eighteen, and twenty healthy people who were the same age and sex as the patients were included in this study.

Regarding Family history in the current study, 15.0% of vitiligo patients had a positive family history.

This was supported by previous studies [38 39, 40].

In the present study, regarding the present history of illness, the duration of illness was 1 month in 15.0% of vitiligo patients, > 1 – 12 months in 35.0% of patients, and >12 months in 50.0% of patients. The duration of illness ranged from 1.0 – 84.0 months with a mean value (±SD) of 25.85 (±23.25) months in the patients group.

Praharsini *et al*. [38] reported that 34.38% of cases had early onset vitiligo (Onset >30 years old), which is consistent with our findings [38]. Furthermore, a research by Lazzeri *et al*. [41] found that 35.60 percent of 191 individuals with early-onset vitiligo had similar outcomes [41].

The present study showed that regarding stability, 85.0% of vitiligo patients were unstable, and 15.0% of patients were stable.

According to stability, Kassab *et al*. [42] showed that 50% of the patients were unstable and 50% were in the stable. Moreover, Liu *et al*. [26] reported that 20.83% of vitiligo cases were stable and 79.17% of cases were unstable.

According to the location and severity of the vitiligo lesion, 10 patients (50.0%) had it on their face, 7 patients (35.0%) had it on their neck, 6 patients (30.0%) had it on their trunk, 13 patients (65.0%) had it on their upper leg, and 14 patients (70.0%) had it on their lower leg. With a mean value (±SD) of 17.25 (±10.57) percent, the VASI score varied from 10.0 to 50.0%.

Nagaty *et al*. [43] conducted a study on 30 vitiligo patients, which is consistent with our findings. 16 patients (53.3%) had facial lesions. In 18 cases, the hands and feet were afflicted (60.0%). 15 patients (50.0%) had biopsies taken from their back, 7 patients (23.3%) from their leg, 2 patients (6.7%) from their foot, and 3 patients (10.0%) from their arm.

Additionally, Kassab *et al*. [42] demonstrated that the VASI score had a mean value (±SD) of 9.40±2.4% (180) and varied from 2 to 12.5%. According to a different study by Li *et al*., the median VASI score for vitiligo patients was 0.7 (0.3-2)%, whereas the mean score was 4.07.

This was consistent with a research that found that 55.1% of vitiligo cases included the face, and 41.1% involved the hands or feet [44].

The result of the present study revealed that the mean serum level of vitamin E was 1.90±1.05 in vitiligo patients compared to 1.70±0.62 in the healthy control with non-significant difference between both.

The level of vitamin E in vitiligo patients was examined in a study by Korobko [18] which indicates that vitamin E supplementation in vitiligo therapy would probably not have any effect on re-pigmentation. This is further supported by the idea that oxidative stress is a trigger rather than a driver of melanocyte destruction carried out by immune system cells. As a matter of fact, in the studies that used vitamin E monotherapy as a control arm, there was no discernible impact on re-pigmentation.

Furthermore, Ines *et al*. [14] demonstrated that serum vitamin E levels in vitiligo patients (both stable and unstable) and healthy controls did not differ statistically significantly.

Additionally, Oğuz *et al*. [45] revealed that the mean level of vitamin E was 11.8±3.4 µg/L in the vitiligo patients compared to 12.6±4.1 µg/L in the healthy control with non-significant difference between both. A 3-month study at Ankara City Hospital included 83 vitiligo patients and 72 age- and gender-matched controls.by Oğuz *et al*. [45] revealed that the mean level of vitamin E was 11.8±3.4 µg/L in the vitiligo patients compared to 12.6±4.1 µg/L in the healthy control with non-significant difference between both.

According to the research, vitiligo patients had substantially lower vitamin E concentrations than the healthy control group [14, 46]. The age and race differences between the patients in the current study and those in these studies may be the cause of this discrepancy.

The result of the present study revealed that the mean level of vitamin A was 87.52±42.73 µg/mL in the vitiligo patients compared to 72.61±29.65 µg/mL in the healthy control with non-significant difference between both.

Similarly, Huo *et al*. [46] showed that Vitamin A was not significantly different between.

Also. The result of Oğuz *et al*. [45] study revealed that the mean level of vitamin A was (517.3±263.1 µg/L) in the vitiligo patients compared to (624.2±217.6 µg/L) in the healthy control with non-significant difference between both.

In accordance with our results, Ines *et al*. [14] showed that there was no statistically significant difference in vitamin A level between vitiligo patients (9.18±1.56 µg/mL) and control groups (9.61±1.39 µg/mL).

The current findings demonstrated that the serum level of vitamin E and vitamin A showed an increase in unstable vitiligo patients but with a non-significant difference between stable and unstable vitiligo patients.

There is no clear evidence linking vitamin A levels to the stability of the vitiligo condition. While active vitiligo may involve increased oxidative stress, and maintaining adequate antioxidant levels might be beneficial, vitamin A alone has not been proven to affect the progression or stability of vitiligo significantly.

The present study showed that there was a non-significant correlation between VASI score and (Level of vitamin E and level of vitamin A).

The possible explanation may be due to that while vitamin E is known for its antioxidant properties and potential role in reducing oxidative stress, its levels might not directly influence the extent or severity of vitiligo in a measurable way. Consequently, while maintaining adequate vitamin E intake is beneficial for overall health, it may not be a significant factor in managing vitiligo severity based on the VASI score alone [47].

Oxidative stress plays a role in the development and progression of vitiligo. Some studies have indicated that patients with vitiligo may have lower levels of vitamin E. Higher levels of vitamin E might theoretically help reduce oxidative stress and improve skin condition, potentially affecting the VASI score positively. However, direct clinical evidence linking vitamin E levels specifically to changes in the VASI score is limited. While antioxidant therapy, including vitamin E, might offer some benefit, it should be considered as part of a broader treatment strategy [48].

The role of vitamin A in vitiligo is less clear-cut, with

limited research directly linking its levels to the VASI score. Some studies suggested that vitamin A might help with skin repair and pigmentation, but there was insufficient evidence to definitively state how vitamin A levels correlate with VASI scores. Ensuring adequate vitamin A intake was important for overall skinhealth, but its direct impact on vitiligo severity as measured by the VASI score is not well-established [49].

There was a non-significant correlation between duration of illness and VIDA score and (serum level of vitamin E) (p= 0.233 and 0.513, r= -0.279 and 0.155, respectively) and a negative correlation between age and serum level of vitamin E (p= 0.025, r= -0.5).

There was non-significant correlation between duration of illness and VIDA score and serum level of vitamin A (p= 0.262 and 0.49, r= -0.327 and 0.229, respectively) and negative correlation between age and serum level of vitamin A (p= 0.037, r= -0.468).

Supporting our results, a previous study by Praharsini *et al.* [38] who found a significant positive correlation between VIDA score and oxidative state suggesting that anti-oxidant administration might decrease the VIDA score.

Additionally, Oğuz *et al.* [45]. showed that the levels of vitamin A (P = 0.350, r = -0.11), vitamin E (P = 0.18, r = 0.17), and the activity of vitiligo illness (VIDA score) did not correlate. Additionally, Vitamin E (P = 0.590, r = -0.07) and vitamin A (P = 0.050, r = -0.22) levels did not correlate with the disease's spread (VASI score).

According to Agrawal *et al*., people with vitiligo typically have reduced vitamin E levels. Vitamin E levels were 0.67±0.22 µg/mL in vitiligo patients and 0.67±0.15 µg/mL in the healthy population, with no significant difference between the two groups. Patients with higher levels of oxidative stress probably consumed more vitamins, which resulted in lower levels [50].

However, there were different results reported by Oğuz *et al.* [45] who found that there was no correlation between vitamin A (P = 0.650, r = 0.05) and vitamin E (P = 0.940, r = -0.03) levels and age. The different results may be accounted by the different sample size [45].

Recommendations: Further prospective multicenter studies with larger sample size would be more informative, further studies on additional inflammatory and oxidative stress markers are required and more studies are required to clarify the causal relationship between vitamins A, and vitamin E on vitiligo.

Table 1: Sex and age were non-significant difference between both patients and controls.

Table 1: Comparison between the patients and the controls according to age and sex

Variable		Patients (n = 20)	Controls (n = 20)	Test of Sig.	p
Sex	Male	14 (70.0%)	10 (50.0%)	$\chi^2 = 1.667$	0.197
	Female	6 (30.0%)	10 (50.0%)		
Age (years)		10.48±4.77	8.35±4.45	t = 1.456	0.154

Data are presented as mean± SD or frequency (%). X^2: Chi square test, t: Student t-t-test. p: p value for comparing between patients and controls. IQR: Inter quartile range SD: Standard deviation

Table 2: Distribution of the studied patients according to duration of illness in patients, VIDA score, site of lesions and VASI Score Range were enumerated.

Table 2: Distribution of the studied patients according to duration of illness in patients, VIDA score, site of lesions and VASI Score Range

Duration		Patients
1 month		3 (15.0%)
>1 – 12 months		7 (35.0%)
>12 months		10 (50.0%)
Mean±SD		25.85±23.25
VIDA score	Stable	3 (15.0%)
	Unstable	17 (85.0%)
	–1	0 (0%)
	+1	0 (0%)
	+2	1 (5.0%)
	+3	4 (20.0%)
	+4	12 (60.0%)
site of lesions	Face	10 (50.0%)
	Neck	7 (35.0%)
	Trunk	6 (30.0%)
	UL	13 (65.0%)
	LL	14 (70.0%)
VASI Score Range	10–20%	12 (60.0%)
	>20–30%	7 (35.0%)
	>30–40%	0 (0%)
	>40%	1 (5.0%)
	Mean± SD	17.25±10.57

Data are presented as mean± SD or frequency (%). VIDA: Vitiligo Disease Activity Index, VASI: Vitiligo Area Scoring Index, UL: Upper limbs, LL: Lower limbs.

Table 3: Level of vitamin E and A were higher in patients than controls but with non-significant difference between both (p value>0.05).

Table 3: Comparison between the patients and controls according to serum levels of vitamin E and Vitamin A

	Patients (n = 20)	Controls (n = 20)	U	p
Serum Vitamin E (µg/ml)	1.90±1.05	1.70±0.62	187.5	0.738
Serum vitamin A(µg/mL)	87.52±42.73	72.61±29.65	132.50	0.068

Data are presented as mean± SD or frequency (%). U: Mann Whitney test. p: p value for comparing between patients and controls. IQR: Inter quartile range SD: Standard deviation

Table 4: Serum level of vitamin E was higher in unstable patients (+3 and +4) than stable patients and unstable patients (+2) but with non-significant difference between stable patients and unstable patients (+2, +3 and +4). Serum vitamin E was higher in unstable patients (+3) than unstable patients (+2) and showed significant difference between both (P=0.038). Serum level of vitamin A was higher in unstable patients (+3 and +4) than stable patients and unstable patients (+2) but with non-significant difference between stable patients and unstable patients (+2, +3 and +4). Serum vitamin A was higher in unstable patients (+4) than unstable patients (+3) and showed significant difference between both. (P=0.040).

Table 4: Relation between VIDA score and vitamin (E and A) serum levels in the studied patients (n = 20)

	VIDA score				H	p
Level of vitamin E (µg/mL)	Stable (n = 3)	Unstable				
		+2 (n = 1)	+3 (n = 4)	+4 (n = 12)		
	1.4±0.06	1.1	2.4±1.43	2.2±2.29		
P1		0.237	0.209	0.733	4.814	0.186
P2			0.038*	0.128		
P3				0.201		
Level of vitamin A (µg/mL)	69.5±8.03	62.7	97.6±36.77	110.6±124.93		
P1		0.770	0.060	0.694	5.58	0.134
P2			0.112	0.570		
P3				0.040*		

Data are presented as mean± SD or frequency (%). H: H for Kruskal Wallis test, p: p value for comparing between stable and unstable patients, P1: p value for comparing between stable and unstable (+2, +3 and +4) patients, P2: p value for comparing between unstable (+2) and unstable (+3 and +4) patients, P3: p value for comparing between unstable (+3) and unstable (+4) patients.

Table 5: Serum level of vitamin E and A was higher in patients with VASI score >20-30% than patients with VASI score >40% but with non-significant difference between different scores of severities.

Table 5: Relation between VASI score (%) and mean serum level of vitamin E and Vitamin A in the patients (n = 20)

	VASI score (%)			H	P
	10-20% (n = 12)	>20-30% (n = 7)	>40% (n = 1)		
Level of vitamin E (µg/mL)	1.9±0.94	2.5±2.99	1.1	3.040	0.219
	P1=0.598, P2=0.084, P3=0.148				
Level of vitamin A (µg/mL)	83.9±29.84	131.3±163.43	62.7	0.817	0.665
	P1=0.649, P2=0.397, P3=0.534				

H: H for Kruskal Wallis test, p: p value for comparing between the different categories, P1: p value for comparing between patients with VASI score 10-20% and >20-30%, P2: p value for comparing between patients with VASI score 10-20% and >40%, P3: p value for comparing between patients with VASI score >20-30% and >40%.

Table 6: There was non-significant correlation between VASI score and serum levels of vitamin E and vitamin A and between duration of illness and VIDA score and serum levels of vitamin E and A and negative correlation between age and serum levels of vitamin E and A.

Table 6: Correlation between VASI score (%) with vitamin E and A levels and between serum levels of vitamin E and A with (Age, duration of illness and VIDA score) in the studied patients

(n = 20)	VASI score (%)	
	r_s	P
Level of vitamin E (µg/mL)	–0.315	0.176
Level of vitamin A (µg/mL)	–0.164	0.489
Serum level of vitamin E (µg/mL)		
Age (Years)	-0.500	0.025*
Duration of illness	-0.279	0.233
VIDA score	0.155	0.513
Serum level of vitamin A (µg/mL)		
Age (years)	-0.468	0.037*
Duration of illness	-0.327	0.262
VIDA score	-0.229	0.49

r_s: Spearman coefficient

Conclusions

Due to differences in metabolic rate and oxidative stress between children and adults, vitamin A and vitamin E played a minor impact in the pathophysiology of vitiligo illness in children rather than adults. It is unknown how antioxidants affect vitiligo stability. Compared to patients with severe vitiligo, those with less severe vitiligo had higher serum levels of vitamin A and vitamin E. This may imply that antioxidants play a part in managing or lessening the severity of vitiligo in kids.

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