



Measurement Serum Levels of Vitamin B12 in vitiligo Patients

Ali Mohammed Abd AL-Ameer^{1*}, Adil Mohammed Hashim¹, Asmaa Adnan Najm².

¹DNA Research Center, University of Babylon, Al-Hilla, Iraq.

²Babylon Technical Institute, Al-Furat Al-Awsat Technical University, Babylon, Iraq.

*Corresponding Author: Ali Mohammed Abd AL-Ameer, DNA Research Center, University of Babylon, Al-Hilla, Iraq, Email: ali.2000mo@yahoo.com.

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ABSTRACT

Vitiligo is a skin disease caused by a disorder in the work of the immune system that attacks melanocytes causing "to kill them and the loss of melanin pigment in the skin, several theories have been proposed to understand the causes of the occurrence of the disease, including the theory of autoimmune diseases, oxidative stress, neurological and genetic factors, including those related to exposure to some chemicals and the appearance of the disease may be associated with a decrease or increase in some vitamins, including vitamin B12, the aim of this research was to learn more about this vitamin because of its important effects on the system Blood and nervous system and its complex relationship to the skin and its association with the onset of the disease by measurement level's of concentration of vitamin B12 .

Methods: The current study included 120 participants as the control group (who appeared to be in good health) and 100 participants with vitiligo. These samples were obtained between 1 October to 15 December 2022, from the Dermatological Consultation Unit of the Imam Sadiq Teaching Hospital in the Babylon Governorate. ELISA test kits were used to measure the amount of vitamin B12 concentration in the serum of all patients and the control group.

Results: Serum for vitamins B12 levels significant decrease at the probability level ($P \leq 0.05$) of patient's with vitiligo, while evaluating the same-criteria rates for the control group.

Conclusions: In the current study, it was revealed Patients with vitiligo reported reduced serum for vitamins D levels compared to control group had.

Keywords: *Melanin, Autoimmune diseases, Vitamin B12.*

INTRODUCTION

Vitiligo is one of the important and common skin diseases, which is characterized by the loss of pigmentation of the skin and its appearance in the form of chalky white spots and defined or reddish-pink color that covers the surface of the skin[1]. Vitiligo is spread all over the world, with a global prevalence of 0.5-4 %. Studies indicate that the pathological pathways that occur due to

melanocyte apoptosis and lead to the causes of vitiligo [2], root causes of vitiligo are still not known precisely, and studies confirm the existence of many causes, including autoimmune factors, genetic causes, neurological causes, the hypothesis of cytotoxicity, and stress factors that will result of imbalance in rates of free radicals and their rise inside pigment cells, causing an increase in oxidative stress for melanocytes [3].

All contribute to the emergence of vitiligo in humans. Vitamin B12 is one of the most important vitamins of the B group of vitamins. It is characterized by an effective vital role in the functions of the nervous system and blood. A deficiency in vitamin B12 causes vitiligo by raising the level of homocysteine concentration in serum, which in turn inhibits tyrosinase (the enzyme responsible for melanin synthesis) [4]. It increases oxidative stress. Vitamin B12, cobalamin, is a water-soluble vitamin. It is also an important coenzyme in the metabolism of DNA, proteins, fats, and carbohydrates. Microorganisms such as bacteria are an essential source of vitamin B12 in nature [5,6]. Vitamin B12 is available in animal-source foods such as meat, liver, fish, and eggs. Because the human body is unable to produce vitamin B12, the body needs (2-3 micrograms) of to provide the body with enough vitamin B12. The properties of vitamin B12 include co-factor for the enzyme homocysteine and methyltransferase and affects the formation of methionine from homocysteine [7]. According to studies, a lack of vitamin B12 causes serum levels for homocysteine to rise. Hyperhomocysteinemia contributes to stimulating some diseases, and many studies have shown a high concentration of homocysteine in the blood in patients with vitiligo, Where homocysteine inhibits and disrupts melanin formation through various mechanisms, such as inhibiting tyrosinase activity [8], activating various cytokines, and increasing oxidative stress within melanocytes as a result of imbalance and rising of free radicals. Numerous Research has indicated that vitiligo is an autoimmune disease that targets melanocytes and there is a link between vitiligo and many other diseases such as thyroiditis[9] and pernicious anemia and diabetes. The fact that those infected with these diseases have developed autoantibodies that can attack melanocytes and destroy them with immune killing pathways [10,11], as patients with vitiligo have antibodies against the thyroid gland, the adrenal gland, and the parietal cells of the stomach responsible for the secretion of the Intrinsic Factor for the absorption of vitamin B12 [12].

MATERIALS AND METHODS

Samples were collected from the Dermatology Consultation Unit at the Imam Al-Sadiq Teaching Hospital in the Babil Governorate

between the dates of 1/10/2022 to 15/12/2022. As part of the current study, (5 ml) venous blood was drawn from (120) participants with vitiligo and (100) participants who were clinically diagnosed as healthy, they had no symptoms and were used as a control group for the study, The two groups ranged in age from 21 to 55 years. The blood was put in a gel tube and allowed to curdle at room temperature (20–25) C⁰ and being centrifuged for 10 minutes at 3000 rpm [13]. The level of vitamin B12 concentration in the sera of vitiligo patients and the control group was investigated. Evaluating with enzyme-linked immunosorbent assay (ELISA) according to manual procedures of (J. Mitra & Co. Pvt. Ltd ,INDIA), Used statistical analysis (using SAS version 9.2) to examine data results (SAS institute inc, Cary,NC,USA). Vitamin B12 levels in the serum of the patient and control groups were compared using the t test.

RESULTS

The findings of the present investigation revealed a substantial ($P < 0.05$) decline in vitamin B12 levels in the patient group when compared to a healthy group (70.64 ± 611.58), (379.18 ± 199.15) (pg/ml), respectively, Patients with vitiligo range in age was (21.01 ± 50.54) years, and the range age of a healthy group was (28.08 ± 46.29) years, there was no statistically in Sig ($P = 0.74$) between patient group and a healthy group, as shown in table 1.

TABLE 1: Show averages of B-12 and age for patients and control group.

Sample	SD±Mean	Sig
B12(Pg/ml)	patients	0.44
	Control	
Age (years)	Patients	0.74
	Control	

DISCUSSION

The causes of vitiligo are still unclear and unknown, and some of these reasons may be due to the disorder that occurs in the immune system, which works to attack pigment cells and cause them programmed cell death [14,15] Vitamin B12 deficiency in vitiligo patients may also be attributed to the condition of vitiligo an autoimmune disease and has developed special antibodies against parietal cells (which secrete

the essential factor for vitamin B12 binding so that the body can absorb it) of the stomach [16]. Some studies have shown a section of these mechanisms that may participate in making pigment cells a target for cells of the immune system, including the occurrence of oxidative stress within pigment cells due to the stress that occurs within the endoplasmic reticulum as a result of elevated homocysteine [17]. This study supports what was indicated, which is that the increase in the concentration of homocysteine is matched by a decrease in the concentration of vitamin B12 levels, as symptoms of vitamin B12 deficiency cause serum levels of homocysteine to rise [18], which works to disrupt the tyrosinase enzyme and thus causes a deficiency in the formation of melanin pigment, as well as vitamin deficiency. B12 leads to an increase in homocysteine and damage to pigment cells in several ways, such as activating inflammatory cytokines through the rise of free radicals within pigment cells, which makes these cells stressed and weak, stimulating the release of protein substances of a special kind [19]. Characterized as antigens by the immune system to make them a “target” for attack by its cells and causing them to be killed by the process of programmed cell death. Other studies have shown that the level for vitamins B12 concentration between patient's group and a healthy group is not significantly different, and this may be due to the difference in Dietary habits, geographical area, and different calibration methods in some studies.

CONCLUSION

According to the current study, lower levels for vitamin B12 Patients with vitiligo had than the study's control group.

REFERENCES

- Ozturk, I. C. *Et al.* Comparison of plasma malondialdehyde, glutathione, glutathione peroxidase, hydroxyproline and selenium levels in patients with vitiligo and healthy controls. *Indian J. Dermatol.* **53**, 106–110 (2008).
- Dell'Anna, M. L. *Et al.* Membrane lipid alterations as a possible basis for melanocyte degeneration in vitiligo. *J. Invest. Dermatol.* **5**, 1226–1233 (2007).
- Jimbow, K., Chen, H., Park, J. S. & Thomas, P. D. Increased sensitivity of melanocytes to oxidative stress and abnormal expression of tyrosinase-related protein in vitiligo. *Br. J. Dermatol.* **1**, 55–65 (2001).
- Boissy, R. E. & Manga, P. On the etiology of contact/occupational vitiligo. *Pigment Cell Res.* **3**, 208–214 (2004).
- Hasse, S., Gibbons, N. C., Rokos, H., Marles, L. K. & Schallreuter, K. U. Perturbed 6-tetrahydrobiopterin recycling via decreased dihydropteridine reductase in vitiligo: more evidence for H₂O₂ stress. *J. Invest. Dermatol.* **2**, 307–313 (2004).
- Schallreuter, K. U., Elwary, S. M., Gibbons, N. C., Rokos, H. & Wood, J. M. Activation/deactivation of acetylcholinesterase by H₂O₂: more evidence for oxidative stress in vitiligo. *Biochem. Biophys. Res. Commun.* **2**, 502–508 (2004).
- Dell'Anna, M. L. *Et al.* Membrane lipid defects are responsible for the generation of reactive oxygen species in peripheral blood mononuclear cells from vitiligo patients. *J. Cell. Physiol.* **1**, 187–193 (2010).
- Le Poole, I. C., van den Wijngaard, R. M., Westerhof, W. & Das, P. K. Tenascin is overexpressed in vitiligo lesional skin and inhibits melanocyte adhesion. *Br. J. Dermatol.* **2**, 171–178 (1997).
- Gauthier, Y., Cario-Andrè, M., Lepreux, S., Pain, C. & Taieb, A. Melanocyte detachment after skin friction in non lesional skin of patients with generalized vitiligo. *Br. J. Dermatol.* **148**, 95–101 (2003).
- Rokos, H., Beazley, W. D. & Schallreuter, K. U. Oxidative stress in vitiligo: photo-oxidation of pterins produces H₂O₂ and pterin-6-carboxylic acid. *Biochem. Biophys. Res. Commun.* **4**, 805–811 (2002).
- Moore, J., Wood, J. M. & Schallreuter, K. U. Evidence for specific complex formation between alpha-melanocyte stimulating hormone and 6(R)-L-erythro-5,6,7,8-tetrahydrobiopterin using near infrared Fourier transform Raman spectroscopy. *Biochemistry* **46**, 15317–15324 (1999).
- Schallreuter, K. U. *Et al.* Epidermal H₂O₂ accumulation alters tetrahydrobiopterin (6BH₄) recycling in vitiligo: identification of a general mechanism in regulation of all 6BH₄-dependent processes? *J. Invest. Dermatol.* **1**, 167–174 (2001).
- Bellei, B. *Et al.* Vitiligo: a possible model of degenerative diseases. *Plos ONE* **3**, e59782 (2013).
- Salem, M. M. A. E. L. *Et al.* Enhanced DNA binding capacity on up-regulated epidermal wild-type p53 in vitiligo by H₂O₂-mediated oxidation: a possible repair mechanism for DNA damage. *FASEB J.* **23**, 3790–3807 (2009).
- Xavier, J. M., Morgado, A. L., Solá, S. & Rodrigues, C. M. Mitochondrial translocation of p53 modulates neuronal fate by preventing differentiation-induced mitochondrial stress. *Antioxid. Redox Signal.* **21**, 1009–1024 (2014).
- Paradisi, A. *Et al.* Markedly reduced incidence of melanoma and nonmelanoma skin cancer in a

- nonconcurrent cohort of 10,040 patients with vitiligo. *J. Am. Acad. Dermatol.* **71**, 1110–1116 (2014).
17. Teulings, H. E. *Et al.* Decreased risk of melanoma and nonmelanoma skin cancer in patients with vitiligo: a survey among 1307 patients and their partners. *Br. J. Dermatol.* **168**, 162–171 (2013).
 18. Dell'Anna, M. L. *Et al.* Alterations of mitochondria in peripheral blood mononuclear cells of vitiligo patients. *Pigment Cell Res.* **16**, 553–559 (2003).
 19. Dell'Anna, M. L. *Et al.* Mitochondrial impairment in peripheral blood mononuclear cells during the active phase of vitiligo. *J. Invest. Dermatol.* **117**, 908–913 (2001).