A Cross-sectional Study of the Catalase Genetic Polymorphism (-262 cytosine/thymine) and Blood Catalase Activity among Jordanian Vitiligo Patients

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ABSTRACT

Vitiligo is brought on by functional melanocyte loss and manifests as white maculae that may cover the whole body's skin. There is a genetic background in the pathogenesis of vitiligo. Polymorphisms in different parts of catalase gene may affect the disease activity and result in less functional catalase, thus, accumulation of hydrogen peroxide, one of the oxidative factors that damage melanocytes. We evaluated the CAT 262 genetic polymorphism of vitiligo patients using the polymerase chain reaction (PCR) technique with at least one C and at least one T model. The study included 48 vitiligo patient and 51 control individuals. Family history of vitiligo was present in 27.1% of patients and autoimmune disease were diagnosed in 16.7% of patients. Three quarters of vitiligo patients (75.0%) reported that emotional stress was the major triggering factor for their disease. The CC genotype was predominant (56.2% in vitiligo patients and 62.7% in control) with no significant difference between the study groups (p=0.7). Catalase activity in blood was comparable between the study arms (159.1±21.6 MU/L in vitiligo patients and 151.3±25.4 MU/L in controls (p=0.15). We conclude that neither genetic polymorphism in CAT 262 C/T nor blood catalase activity is associated with vitiligo.

Keywords: Genetic Polymorphism, Blood Catalase, Vitiligo, CAT Gene (-262 cytosine/thymine), Jordanians.

INTRODUCTION

The most prevalent pigmenitary condition, vitiligo, is brought on by functional melanocyte loss and manifests as white maculae that may cover the whole body's skin[1].

The etiology of vitiligo appears to be multifactorial, including mainly environmental, genetic, and immunologic factors which may synergistically cause melanocyte destruction[2]. The two main types of vitiligo are non-segmental vitiligo, which is the most prevalent form and often manifests as bilateral white patches, and segmental vitiligo, which has a unilateral distribution[3] and contributes to 5–16% of cases[4]. In 87% of cases the disease started before the age of 30 years and in 41.3% it started before the age of 10[3].

In Jordan, the prevalence of vitiligo in children was found to rise with age (0.45% 1 years, 1% 1-5 years, 2.1% 5-12 years). The prevalence of vitiligo was found to range from 0.06% to 2.28% in the general population and from 0% to 2.16% in juvenile populations. In 92.9% of vitiligo patients, non-segmental form was present[5].

Studies using histology and immunohistochemistry demonstrated that the afflicted skin region is devoid of melanocytes, despite their occasional presence[6]. High level of both organ- and non-organ-specific autoantibodies have been reported in the serum of vitiligo patients [7]. The antimelanocyte and antinuclear antibodies were shown to be higher in vitiligo patients as opposed to controls, furthermore, these antibodies levels were shown to
correspond with disease activity[8]. These autoantibodies, which belong to the class G immunoglobulins, were also discovered in the lesional vitiligo epidermis' basal layer, together with deposits of complement component 3 (C3)[9]. Tyrosinase, tyrosinase-related protein-1 (TRP-1), TRP-2, Pmel17 (also known as gp100), the transcriptional factors SOX 9 and SOX 10, and the type 1 membrane receptor for melanin-concentrating hormone are the major melanocytic antigens (MCH-R1)[10,53]. Additionally, people with vitiligo have high frequencies of cytotoxic T lymphocytes that are reactive to melanocytes in their peripheral blood[11], capable of releasing type B granzyme, perforin, and IFN, while vitiligo epidermis exhibits perilesional T-cell infiltration[12]. It was shown that CD8+ T lymphocytes that have the characteristics of skin-homing, are oriented toward type-1 effector function, and are significantly cytotoxic being clustered around disappearing melanocytes[13].

Recent studies of the pathophysiology of vitiligo highlighted the significance of reactive oxygen species (ROS) and their function in melanocyte-intrinsic abnormalities as potential major triggers of the whole inflammatory cascade[14]. In oxidative stress, superoxide is a primary oxygen radical produced when an oxygen molecule receives one electron. Superoxide dismutase (SOD) converts the superoxide to hydrogen peroxide (H2O2) that, in the presence of free ferrous iron, may produce hydroxyl radicals and exacerbate diseases[15]. Normally, the antioxidant system defends the cell from ROS, however, during oxidative stress, this system becomes unbalanced[16,54], in addition to H2O2 buildup brought on by environmental trauma like UVB exposure (290-320 nm)[17]. Together, autoimmunity and oxidative stress have a synergistic impact on melanocytes, causing cell death and depigmentation[18]. The immune system creates a long-lasting inflammatory environment where ROS build and damage nearby cells[19].

One of the antioxidant enzymes is catalase which is responsible of the conversion of hydrogen peroxide, one of ROS, to water and oxygen. A number of studies investigated the catalase activity in both plasma and epidermis of vitiligo patients with contradictory results[7,17,20].

It has been suggested that a relation exists between genetic factors and susceptibility of vitiligo.

Genetic polymorphisms in different regions in CAT gene (a gene that encodes for catalase) have been studied in relation to vitiligo[21]. The gene for human catalase has been mapped to chromosome 11, band p13, and is split into 13 exons by 12 introns and spans[22]. Numerous polymorphisms have been described in the promoter, 5’ and 3’- untranslated regions (UTRs), exons and introns[23,24]. This study focuses mainly on the most common CAT gene polymorphism -262 cytosine/thymine (-262 C/T) in the promoter region in which C to T substitution occur at position -262 (db SNP ID: rs1001179) and that has been found to be associated with alteration in the CAT activities. The 262 C/T polymorphism may affect CAT transcription by modulation of the transcriptional factor binding position and increased basal CAT expression in various cell including erythrocytes[24]. The latter study investigated the relation between 262 C/T genetic polymorphism and the level of catalase in general, not in relation to any specific disease, and found that individuals carrying T allele have significant higher level of catalase in comparison with individuals carrying homozygote C allele[24]. On the contrary, another group of investigators showed that CC homozygotes had higher activity of CAT compared to those with CT or TT genotypes[25]. A later study found no association between the CAT262C/T polymorphism and CAT activity[26].

With regards to the association of CAT262C/T polymorphism and vitiligo in particular we found two studies, and both demonstrated lack of such association[17,27].

The aim of the study was dual: to assess the relation between the CAT 262 C/T polymorphism of and the susceptibility to vitiligo in Jordanian patients; in addition to the study of blood catalase activity in Jordanian vitiligo patients.
METHODOLOGY

Study design

This is a cross-sectional study that involved patients diagnosed with vitiligo and apparently healthy individual matched by age and gender who served as control.

Sample size

a) Using the online software https://clincalc.com/stats/samplesize.aspx, we have calculated the required sample size for detecting differences in blood catalase activity between vitiligo patients and found the size to be 24 per group based on the data from the previous publication[28]. However, we included 48 patients and 51 controls.

Clinical Setting and Patients

The vitiligo patients (cases) were recruited from the Dermatology Clinics at Al-Basheer Hospital related to Jordanian Ministry of Health and Kind Hussein Medical City related to the Royal Medical Services, Amman, Jordan over the period of May 1, 2015 to June 17, 2015. Vitiligo diagnosis was established by dermatologists using standard diagnostic criteria[3]. The active vitiligo was defined as the progression or appearance of new lesions in the last 3 months and the stable vitiligo was defined as the absence of new lesions or progression in the last 6 months, respectively[29]. Healthy volunteers (control) were recruited from the outpatient clinics.

Inclusion criteria for vitiligo cases

1. Jordanian nationality.
2. Age between 18 and 60 years.

Exclusion criteria for cases

1. Any acute or chronic disease
2. Patient with other skin diseases

Inclusion criteria for controls:

1. Jordanian nationality
2. Age-matched (by decades) and gender-matched to control apparently healthy individuals.

Exclusion criteria for control

1. Any acute or chronic disease
2. History, either personal, or family, of any autoimmune diseases
3. No personal or family history of vitiligo.

Ethical consideration

The privacy and rights of the human participants were upheld throughout the investigation. The Institutional Review Boards’ research ethical permissions were obtained from Jordanian Ministry of Health (approval number 4822 on March 10, 2015) and Royal Medical Services (approval number 3542 on February 25, 2015). Written informed consent form was provided to each patient, who was then requested to read and sign the form. The use of a code number for each participant rather than his name or file number were used to protect the anonymity and confidentiality of the information, and the potential study recruits were notified that participation in the study is voluntary and that they have the choice to decline without consequence.

Assessment of the CAT 262 polymorphism

The CAT 262 genetic polymorphism was evaluated using the polymerase chain reaction (PCR) with at least one C and at least one T model.

DNA extraction

Venous blood samples were collected from the patients and healthy subjects in K3 EDTA-coated tubes. The tubes were kept in icebox and DNA extraction was performed in the same day. Using the "Promega Wizard genomic DNA purification kit, Promega Corporation, USA" in accordance with the manufacturer's instructions, genomic DNA was isolated from the whole blood.

Assessment of DNA yield and purity

DNA was kept at -20° C until further investigation and the quality of the DNA was assessed using 0.5% agarose gel electrophoresis.

Genomic amplification

Using the forward and reverse primers, a 320 bp amplicon containing the targeted site, CAT 262 (rs1001179), was sequenced using PCR.

Measurement of blood catalase activity

Blood samples were collected by using EDTA tubes;
all samples were prepared after 4 hours of being collected to have consistency in our results. Erythrocyte hemolysis was induced by using saponin due to the high concentration of catalase in erythrocytes. Spectrophotometric assay was used to measure catalase activity in the blood based on the formation of stable complex between hydrogen peroxide and ammonium molybdate based on the method described by Goth (1992)\textsuperscript{30}. In more details, one hundred µL of blood was dissolved it in 2 ml of 10% saponin solution. After finishing sample preparation 180 µL of lysate was taken and incubated it at 37° C for 60 second with 1.5 ml of 65 µmol/ml hydrogen peroxide, then 1.5 ml of 32.4 mmol/l ammonium molybdate was added to stop the enzymatic reaction resulting in a yellow solution that was measured against blank 3 at 405 nm.

The following equation was used to measure catalase activity:

\[
\text{Blood catalase activity (A) (kunits/l) = } \frac{A \text{ (sample)} - A \text{ (blank 1)}}{A \text{ (blank 2)} - A \text{ (blank 3)}} \times 4.26 \times 10^5
\]

In which:

Blank 1: 1.5ml hydrogen peroxide + 1.5 ml ammonium molybdate + 1.5 PBS + 180 µL sample
Blank 2: 1.5ml hydrogen peroxide + 1.5 ml ammonium molybdate + 1.5 PBS
Blank 3: 1.5 ml ammonium molybdate + 1.5 PBS

**Data management and statistical analysis**

Continuous variables were reported as mean ± standard deviation (SD) and the comparison between the groups was conducted using independent sample t-test or one-way ANOVA test, as appropriate. Levene’s test was utilized to examine the homogeneity of variance, and Kolmogrov-Smirnov test was employed to evaluate the normality of distribution. Chi-square or Fisher exact test was used to compare frequency of alleles and genotypes between the vitiligo and the control groups, where allele frequency = number of copies of an allele in a population /total number of all alleles for that gene in a population, and genotype frequency = number of individuals with a particular genotype in a population /total number of all individuals in a population. Genotype and allele frequencies were matched to expectation by Hardy-Weinberg equilibrium. Hardy-Weinberg equilibrium was assessed utilizing the formula:

\[(a+b)^2 \text{ with degree of freedom of } 1,\]

where a is the frequency of one allele, and b is the frequency of the other allele. As a measure of the relationship between genotypes, odds ratios (OR) and their 95% confidence intervals (CI) were determined. The Mantel-Haenszel statistics were used to estimate the common odds ratios, their CI, and their p-values. For all comparisons, a p-value of 0.05 or less was regarded as statistically significant.

**RESULTS**

**Study subject’s description:**

The study flow chart is shown in Figure1. Among 90 vitiligo patients approached initially, 42 were excluded, while among 121 healthy individuals approached initially, 70 were excluded from the study. The ultimate number of study participants was 48 vitiligo patients 51 control subjects.
Demographic and clinical characteristics of vitiligo patients:

Demographic and clinical characteristics of vitiligo patients are shown in Table 1. The mean age of vitiligo patients was 33.7 (±13.2) years.

The major proportion of vitiligo patients were females (72.9%). Non-segmental vitiligo was diagnosed in majority of patients (93.7%), and the rest of patients had segmental vitiligo. Active disease was documented in more than half (53.9%) of cases. Three-quarters of patients (75.0%) reported that the emotional stress served as a triggering factor for their disease. Family history of vitiligo was present in 27.1% of vitiligo patients, while total of 16.7% of patients reported presence of autoimmune disease.

Blood catalase activity

The mean catalase activity in the blood (MU/L) did not differ significantly between the vitiligo patients (159.1±21.6) and the controls (151.3±25.4) (p=0.15). The catalase activity was also similar among the vitiligo patients with and without active disease (161.4±22.3 and 156.3±21.0, respectively, p=0.9). Furthermore, the catalase activity did not differ between vitiligo patients who received and who did not receive Psoralen plus ultraviolet A (PUVA) therapy in Table 2.
Table 1. Clinico-demographic characteristics of vitiligo patients (N=48)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
<td>33.7 (13.2)</td>
</tr>
<tr>
<td>Gender, N (%)</td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>13 (27.1)</td>
</tr>
<tr>
<td>Females</td>
<td>35 (72.9)</td>
</tr>
<tr>
<td>Education level, N (%)</td>
<td></td>
</tr>
<tr>
<td>Elementary</td>
<td>9 (18.87)</td>
</tr>
<tr>
<td>High school</td>
<td>27 (56.3)</td>
</tr>
<tr>
<td>Diploma</td>
<td>8 (16.7)</td>
</tr>
<tr>
<td>University</td>
<td>5 (10.4)</td>
</tr>
<tr>
<td>Localization of vitiligo, N (%)</td>
<td></td>
</tr>
<tr>
<td>Segmental</td>
<td>3 (6.3)</td>
</tr>
<tr>
<td>Non-segmental</td>
<td>45 (93.7)</td>
</tr>
<tr>
<td>Disease activity, N (%) (assessed in 39 patients)</td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>21 (53.9)</td>
</tr>
<tr>
<td>Stable</td>
<td>18 (46.1)</td>
</tr>
<tr>
<td>Presence of risk factors for vitiligo, N (%)</td>
<td></td>
</tr>
<tr>
<td>Emotional stress</td>
<td>36 (75.0)</td>
</tr>
<tr>
<td>Chemicals exposure</td>
<td>3 (6.3)</td>
</tr>
<tr>
<td>Sun exposure</td>
<td>2 (4.2)</td>
</tr>
<tr>
<td>Others</td>
<td>7 (14.6)</td>
</tr>
<tr>
<td>Positive family history of vitiligo, N (%)</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>13 (27.1)</td>
</tr>
<tr>
<td>No</td>
<td>35 (72.9)</td>
</tr>
<tr>
<td>Presence of autoimmune diseases, N (%)</td>
<td></td>
</tr>
<tr>
<td>Thyroid disorder</td>
<td>7 (14.6)</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>1 (2.1)</td>
</tr>
</tbody>
</table>

Table 2. Comparison of blood catalase activity in vitiligo patients according to treatment with PUVA therapy

<table>
<thead>
<tr>
<th>PUVA therapy</th>
<th>N*</th>
<th>Mean (±SD)</th>
<th>p**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment-naive</td>
<td>12</td>
<td>156.2 (21.9)</td>
<td></td>
</tr>
<tr>
<td>&lt;6 months treatment</td>
<td>13</td>
<td>157.9 (17.5)</td>
<td>0.9</td>
</tr>
<tr>
<td>&gt;6 months treatment</td>
<td>11</td>
<td>162.3 (27.4)</td>
<td></td>
</tr>
</tbody>
</table>

*The total number of patients was less than 48 due to missing data

** by one-way ANOVA test
**Genotype & Sequencing results**

Figures 2-4 show sequencing results for the complementary strand of CAT gene. Data were analyzed by using Chromas program; one black band means CC genotype, one green band means TT genotype and 2 bands mean CT genotype.

As shown in Table 3, the most prevalent genotype in both vitiligo cases and controls was CC genotype (56.2% and 62.7%, respectively). There was no difference in all three genotypes distribution between the vitiligo patients and the controls (p=0.7). Similarly, the allele distribution did not differ between the vitiligo patients and the controls (p=0.6).

**Table 3. Genotype and allele frequencies among vitiligo patients and controls**

<table>
<thead>
<tr>
<th></th>
<th>Cases (N=48), N (%)</th>
<th>Controls (N=51), N (%)</th>
<th>Total</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Genotype</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CC</td>
<td>27 (56.2)</td>
<td>32 (62.7)</td>
<td>59 (59.6)</td>
<td>0.7*</td>
</tr>
<tr>
<td>CT</td>
<td>18 (37.5)</td>
<td>15 (29.4)</td>
<td>33 (33.3)</td>
<td></td>
</tr>
<tr>
<td>TT</td>
<td>3 (6.2)</td>
<td>4 (7.8)</td>
<td>7 (7.1)</td>
<td></td>
</tr>
<tr>
<td><strong>Allele</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>72 (75.0)</td>
<td>79 (77.5)</td>
<td>151 (76.3)</td>
<td>0.6**</td>
</tr>
<tr>
<td>T</td>
<td>24 (25.0)</td>
<td>23 (24.0)</td>
<td>47 (23.7)</td>
<td></td>
</tr>
</tbody>
</table>

* By Fisher exact test

** By independent-sample t-test

**Figure 2. Genotype sequencing (CT)**
As shown in Table 4, the occurrence of at least one T was not different between the vitiligo patients and controls (p=0.5), likewise, the occurrence of at least one C did not differ between the two study arms (p=0.8).
Table 4. Recessive and dominant genotype distribution among vitiligo patients and controls

<table>
<thead>
<tr>
<th>Study group</th>
<th>At least one T model</th>
<th>At least one C model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CC</td>
<td>CT+TT</td>
</tr>
<tr>
<td>Cases, N (%)</td>
<td>27 (56.3)</td>
<td>21 (43.7)</td>
</tr>
<tr>
<td>Controls, N (%)</td>
<td>32 (62.7)</td>
<td>19 (37.3)</td>
</tr>
</tbody>
</table>

DISCUSSION:

Importance of the study

Among the hypotheses for vitiligo, oxidative stress is considered as the initial pathogenic event in melanocyte destruction[7]. It was recently demonstrated that oxidative stress, in addition to producing multiple forms of melanocyte death, can induce the formation of the inflammatory cytokines, such as IL-6, IL-8, CXCL12, CCL5, CXCL10, and CXCL16 from stressed melanocytes or keratinocytes, resulting in initiation or promotion of the autoimmune reaction toward melanocytes[31].

It is widely accepted that there is a genetic background in the pathogenesis of vitiligo. A few studies were conducted in Jordan to investigate associations of SMOC2[32], NALP1[33], PTPN22 1858C/T[34], PTPN22 and SMOC2[35] and NLRP1[36]genes, the latter two in relation to autoimmune thyroid disease. However, the association of catalase gene with vitiligo has not been investigated in Jordanian population, therefore, we decided to focus on the CAT gene polymorphism 262 C/T as it is less frequently reported than other CAT gene polymorphisms. It was previously shown that polymorphisms in catalase gene may affect the enzyme activity and result in less functional catalase and accumulation of hydrogen peroxide and, on the other hand, these polymorphisms were associated with vitiligo in some populations. However, it was suggested that the controversy concerning the CAT activity in vitiligo patients may be at least partially related to the polymorphisms in the catalase gene[31]. A meta-analysis demonstrated that the 389 C/T polymorphisms in CAT were not associated with the risk of vitiligo in Asians and Turks, on the contrary, the CT genotype was proposed to be a genetic risk factor for susceptibility to vitiligo, while in Western Europeans the CC genotype might decrease the risk of vitiligo[37]. A number of studies demonstrated that CAT -89A/T, -262G/A, and -262T/C polymorphisms in the promoter region and -20T/C in 5'-untranslated region have detrimental effects on the catalase expression or function[21,24,38,39]. In both Chinese and Indian populations, CAT -89A/T variants were associated with a significant decrease in CAT activity and a genetic predisposition for vitiligo, especially in active and generalized vitiligo patients[40,41]. On the other hand, the CAT –262G/A variant showed no change in CAT activity or risk of vitiligo in Indian population[41]. Furthermore, in Northwestern Mexicans, CAT 419 C/T gene polymorphism was not informative, -89 A/T was associated with risk, while 389 C/T conferred protection against vitiligo along with AT haplotype. Additionally, although the serum CAT activity was lower in vitiligo patients, there was no association with any of the polymorphisms[42].

Previous studies demonstrated that the CAT 262 C>T polymorphism may affect the transcription of reporter genes and the binding of transcription factors[4,40,43]. Thus, the purpose of this study was to determine whether there is association between the CAT 262 polymorphism, as well as the blood catalase activity, with vitiligo in Jordanians.
Study findings

Our study showed that 27.1% of patient had family history of vitiligo, lower than reported in a retrospective analysis conducted in Saudi Arabia (42.8%)[43] but higher than in the previous study from Jordan (19.2%)[32] and the US and UK populations (6.1%)[44].

In our study, 16.7% of patients had autoimmune disease in line with the Saudi study where 90.5% of patient had no history of autoimmune disease[43]. Our data regarding prevalence of thyroid autoimmune disease (14.6%) are comparable with another report where thyroid issues were present in 21.1% of vitiligo patients[45].

Genotype

Our results reveal that CC genotype was predominant in both study groups (58.3% in vitiligo patients vs. 62.7% in control). This frequency was somewhat in between the Hungarian and the English populations (Hungarian 43% in vitiligo patients and 36% in controls[17], English 72.3% in vitiligo patients and 75.1% in controls[27]), but the lack of difference in genotype frequency between the vitiligo patients and the controls was consistent among the three studies. Notably, a recent comprehensive meta-analysis and prioritization study to identify vitiligo associated coding and non-coding single-nucleotide variants (SNV) using web-based bioinformatics tools prioritized CAT gene, among 13 SNVs, from a set of 291 SNVs, as a candidate contributing to vitiligo pathogenesis[46].

Catalase activity

There is controversy regarding changes in serum catalase activity in vitiligo patients. Patients with active localized vitiligo showed significant decrease in the catalase levels when compared with healthy controls in studies conducted in Turkey[47,48], Nepal[20] and India[7] Notably, lower catalase activity was found in segmental vitiligo patients, whereas in non-segmental vitiligo patients the enzyme activity was normal[49]. More recently, significantly reduced catalase activity, along with other oxidant-antioxidant systems changes were found in serum of Chinese patients with nonsegmental vitiligo compared with healthy controls (HC)[31]. The current study found no difference in blood catalase activity between the vitiligo patients and the control individuals, in line with the studies from Hungary[17] Tunisia[50] and Turkey[51]. Furthermore, in a study from India, there was a significant decrease in serum catalase activity in the active phase group (p value=0.044) but not in the static group (p=0.095) in comparison with healthy group[7], however, our data did not support this observation. Notably, blood catalase activity in our patients was not affected by PUVA therapy.

Study limitations

Selection bias might occur as this research was a hospital-based cross-sectional study. Female participants were represented more than males and the gender interference with genotype prevalence cannot be excluded. Furthermore, since the data on triggering factors for vitiligo were retrospectively collected, this may increase the risk of recall bias, especially for events that date back to years ago like excessive sun exposure or emotional stress. Additionally, catalase activity was detected only in the blood, and not in the epidermis, which is more invasive but could be more accurately detect the oxidative processes at the local disease site. Genomewide association study, the best type of investigation to detect genetic loci involved in vitiligo, was not conducted.

CONCLUSION

Genetic polymorphism CAT 262C>T is not related to the incidence of vitiligo. There is no difference in blood catalase activity between vitiligo patients and healthy individuals, between patients with active and stable disease, or between patients receiving PUVA therapy and those without such treatment.

ACKNOWLEDGEMENT

We are grateful to study participants as well as medical doctors and nurses who helped us in data and sample collection.
REFERENCES


عدم وجود ارتباط بين تعدد الأشكال الجيني الكاتليز (262السيتوزين) والثايمين) مع التعرض للبهاق بين الأردنيين: دراسة حالة مراقبة

ابتسام الحوامدة1، نايليا بولاتوفا1، المعتصم يوسف1، محمد العبادي1 و ايثار عمر1

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ملخص

تم جلب البهاق عن طريق فقدان الخلايا الصباغية الوظيفية وظهور على شكل بقع بيضاء قد تغطي جلد الجسم كله. هناك خلفية وراثية في التسبب في البهاق. تعدد الأشكال في أجزاء مختلفة من الجينات الكاتلاز قد تؤثر على نشاط المرض و يؤدي إلى تقليل وظيفة الكاتليز، وبالتالي، تراكم بروكسيد الهيدروجين، واحدة من العوامل المؤكسدة التي تضر الخلايا الصباغية، واحدة من العوامل المؤكسدة التي تضر الخلايا الصباغية. فمما يتبين تعدد الأشكال الجيني 262 من مرضى البهاق وراثي في البهاق باستخدام تقنية تفاعل البلمرة المتسلسل (PCR) مع واحد على الأقل سي ونموذج تي واحد على الأقل. وشملت الدراسة 48 مريض البهاق و 51 عينة ضابط. كان التاريخ العائلي للبهاق موجودا في 27.1% من المرضى وتم تشخيص أمراض المناعة الذاتية في 16.0% من المرضى. أفاد حوالي ثلاثة أرباع ضحايا البهاق (75.0%) أن التوتر النفسي كان العامل الرئيسي لمرضهم. كان النمط الجيني CC (56.2% لمرضى البهاق و 62.7% للطبخ) مع عدم وجود فرق كبير بين مجموعة الدراسة. (P=0.7) كان نشاط الكاتليز في الدم متقارب بين أطراف الدراسة (159.1 ± 151.3 وحدة/لتر في مرضى البهاق و 25.4 ± 151.3 وحدة/لتر في العينة الضابطة). (P=0.15) لذا نستطيع أن لا يرتبط تعدد الأشكال الجيني في البهاق

لا نشاط الكاتليز في الدم بالبهاق.

الكلمات الدالة: تعدد الأشكال الجيني، الكاتليز في الدم، البهاق، الأردنيون Cat 262 سيتوسين، تانين، الأردن

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