Official Journal of Erciyes University Faculty of Medicine

DOI: 10.14744/cpr.2025.82711
J Clin Pract Res 2025;47(1):53–59

Investigation of Interleukin-6 -572G/C Gene Polymorphism in Vitiligo Patients

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ABSTRACT

Objective: Vitiligo is a complex autoimmune condition characterized by the progressive destruction of melanocytes, resulting in patches of skin depigmentation. Interleukin-6 (IL-6), a multifunctional cytokine, has been linked to the development of various autoimmune diseases, including vitiligo. This study aims to investigate the association between the -572G/C polymorphism in the IL-6 gene and susceptibility to vitiligo.

Materials and Methods: The study included 50 vitiligo patients and 50 healthy controls. Genetic analysis was conducted using DNA extracted from blood samples, followed by genotyping through polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP) techniques.

Results: The GG genotype was associated with a 2.53-fold increased risk of vitiligo (p=0.04, odds ratio (OR) [95% confidence interval (CI)] = 2.53 [1.03–6.23]), whereas the CG genotype significantly reduced this risk (p=0.04, OR [95% CI] = 0.39 [0.16–0.97]). A significant difference in allele frequencies was observed, with the G allele increasing the risk of vitiligo (p=0.03, OR [95% CI] = 2.39 [1.06–5.38]) and the C allele demonstrating a protective effect (p=0.03, OR [95% CI] = 0.42 [0.19–0.94]).

Conclusion: The findings suggest that the GG genotype and G allele of the IL-6 -572G/C polymorphism are associated with an increased risk of vitiligo in the Turkish population, underscoring a potential role for IL-6 in the pathogenesis of the disease.

Keywords: Gene variants, interleukin-6, polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP), polymorphism, vitiligo.

Cite this article as:
Yukcu F, Akcilar R, Dizen Namdar N. Investigation of Interleukin-6 -572G/C Gene Polymorphism in Vitiligo Patients. J Clin Pract Res 2025;47(1):53–59.

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Submitted: 02.09.2024
Revised: 28.11.2024
Accepted: 06.01.2025
Available Online: 03.02.2025

Erciyes University Faculty of Medicine Publications - Available online at www.jcpres.com

INTRODUCTION

Vitiligo is a chronic autoimmune skin disorder characterized by the progressive loss of melanocytes, resulting in depigmented patches on the skin.[1] It affects approximately 1.3% of adults worldwide and, while not life-threatening, significantly impacts patients' appearance and quality of life.[2,3]

Although the precise etiology of vitiligo remains unclear, it is believed to result from an interplay of genetic predisposition with metabolic, immune system, and environmental factors.[4] Familial clustering of vitiligo cases highlights the critical role of hereditary factors in its development.

Interleukin-6 (IL-6) is a glycoprotein cytokine with a molecular weight ranging from 21 to 26 kDa, composed of 212 amino acids and a 28-amino-acid signal peptide.[5] It is secreted by various cell types, including monocytes, endothelial cells, T cells, and fibroblasts, and plays a pivotal role in both local and systemic inflammatory responses.[6,7] Elevated levels of IL-6 have been associated with numerous autoimmune disorders, including vitiligo. By activating and differentiating T cells and modulating B-cell activity, IL-6 contributes to immune processes that may result in the destruction of melanocytes.[7] Research suggests that IL-6 can induce the production of intercellular adhesion molecule-1 (ICAM-1) on melanocytes, facilitating interactions with leukocytes and leading to melanocyte injury and subsequent depigmentation.[6,8,9] Despite these findings, research on IL-6 gene polymorphisms, such as -572G/C (rs1800796), in vitiligo is limited, necessitating further investigation into their potential role in disease susceptibility and pathogenesis.[10]

Investigating the potential association between IL-6 gene polymorphism and vitiligo could provide valuable insights into the progression of the disease, given the critical role of immune and inflammatory mechanisms in its pathogenesis. This study aims to explore the relationship between the IL-6 -572G/C gene polymorphism and susceptibility to vitiligo in the Turkish population, considering the prominent involvement of immunological and inflammatory factors in the disease's development.

KEY MESSAGES

- This study identifies the interleukin-6 (IL-6) gene as a potential genetic factor influencing susceptibility to vitiligo in the Turkish population.
- The G allele and GG genotype of the IL-6 -572G/C polymorphism are significantly associated with an increased risk of developing vitiligo.
- A notable proportion of patients (34%) demonstrated a familial predisposition to vitiligo, as evidenced by the presence of the disease in their first-degree relatives.

MATERIALS AND METHODS

Study Cohort

This investigation included 50 vitiligo patients aged between 18 and 70 years who were clinically diagnosed with vitiligo in the Dermatology outpatient clinic of Kutahya Health Sciences University Faculty of Medicine. These patients were not on any medication and had no inflammatory diseases. The control group consisted of 50 healthy volunteers with a similar age and gender distribution who did not have vitiligo or any inflammatory dermatosis. The study excluded adults with autoimmune disorders, infectious diseases, liver or kidney diseases, neoplastic diseases, diabetes mellitus, inflammatory conditions associated with familial hypercholesterolemia, chronic illnesses, or immunocompromised states. Additionally, vitiligo patients who had undergone photochemotherapy, systemic medication within the past three months, or local treatment in the preceding month were also excluded.

Approval for this study was granted by the Ethics Committee of Kutahya Health Sciences University Faculty of Medicine (approval date: October 11, 2023; approval number: 2023/11-26). The research was conducted in accordance with the ethical standards outlined in the Declaration of Helsinki, and informed consent was obtained from all participants.

The study was conducted over a six-month period, from October 2023 to April 2024, utilizing a case-control design. Molecular analyses were carried out in the Physiology Laboratory of the Faculty of Medicine at Kutahya Health Sciences University.

DNA Isolation

Genomic DNA was isolated from participants' blood samples using a standardized phenol-chloroform extraction method. Initially, cell lysis was performed using a designated lysis buffer. Proteins and other contaminants were removed with phenol-chloroform-isoamyl alcohol (25:24:1). Following centrifugation at 12,000 rpm, the DNA-rich aqueous layer was carefully separated. DNA precipitation was induced using cold ethanol, and the resulting sediment was washed with 70% ethanol to eliminate residual impurities. Finally, the DNA was dissolved in the TE buffer and assessed for purity and concentration using a spectrophotometer before being stored at -20°C for future use.

Analysis of IL-6 -572G/C Gene Polymorphism

Genotyping of the IL-6 -572G/C polymorphism was performed using polymerase chain reaction (PCR) with primers specifically designed for this locus, followed by analysis through restriction fragment length polymorphism (RFLP). The primer sequences and PCR conditions are provided in Table 1. The PCR reaction mixture was prepared to a final volume of 25 µl.[2,11] Amplified

Table 1. Primer sequences and reaction conditions for genotyping interleukin-6 (IL-6) -572G/C polymorphism

Polymorphism (db SNP ID)	**IL-6 -572G/C (rs1800796)**
PCR primer sequences	**F:** 5′-GTG CCC CAG TGA AAC AGT G-3′
	R: 5′-CAA GCC TGG GAT TAT GAA GA-3′
PCR reaction conditions	94°C for 5 min,
	30 cycles of 94°C for 30 s,
	55°C for 30 s, 72°C for 30 s,
	72°C for 7 min

db SNP ID: Database identifier; SNP: Single nucleotide polymorphism; PCR: Polymerase chain reaction; F: Forward primer; R: Reverse primer.

DNA fragments of 358 base pairs were digested with the BSrBI restriction enzyme (Thermo, Catalog No: ER1271) at 37°C for 16 hours. The digested products were resolved on 2% agarose gels (Biomax, Catalog No: HS-8000), stained with ethidium bromide (BioShop, Catalog No: ETB444.50), and visualized under ultraviolet (UV) illumination using a 100 bp DNA marker (abm, Catalog No: G193). Genotypes were identified based on the band patterns: CC (358 bp), GG (285 bp and 73 bp), and CG (358 bp, 285 bp, and 73 bp), as illustrated in Figure 1.

Statistical Analysis

Statistical analyses were conducted using SPSS software version 27.0.1 (IBM Corp., Armonk, NY, USA). The study methodology referenced the work of Wang D. et al.[2]. Power analysis was performed with the G*Power software (version 3.1.9.7; Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany), assuming an odds ratio (OR) of 3.167 and a significance level of 0.05 (95% confidence interval). To achieve a statistical power of 86.5% for detecting differences in genotype frequencies between the patient and control groups, a total sample size of 100 participants (50 per group) was determined to be adequate.

Binomial logistic regression analysis was conducted to assess differences in genotype frequencies between groups, with results presented as OR and 95% confidence intervals. The Pearson Chi-Square (χ^2) test was used to compare genotype and allele frequency distributions of the IL-6 -572G/C gene polymorphism between the groups. Additionally, the χ^2 test was applied to evaluate Hardy-Weinberg equilibrium for allele and genotype frequencies. The association between the clinical features of vitiligo patients and the genetic distribution of this polymorphism was analyzed using the χ^2 test. Clinical data were further assessed using the Independent Samples T-test, with statistical significance set at $p<0.05$.

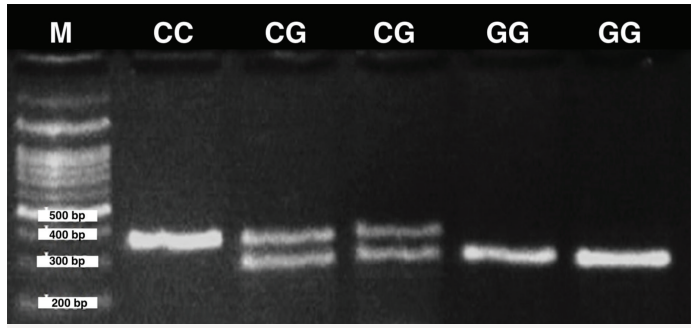

Figure 1. Electrophoresis of the interleukin-6 (IL-6) (-572G/C) gene polymorphism by enzyme digestion. The product sizes were as follows: 285 bp for the GG genotype, 285 bp and 358 bp for the CG genotype, and 358 bp for the CC genotype.

M: 100 bp DNA molecular weight marker (abm, Catalog No: G193).

RESULTS

Table 2 provides an overview of the demographic characteristics and clinical attributes of the participants. Among the 50 vitiligo patients, 19 (38%) were male and 31 (62%) were female, with an average age of 40.5±17.6 years. The control group comprised 19 males (38%) and 31 females (62%), with an average age of 40.7±13.5 years. No statistically significant differences were found in gender or age distribution between the groups. Additionally, 34% of patients (17 individuals) reported having first-degree relatives with vitiligo. Regarding disease classification, 80% of patients had generalized vitiligo, 12% had localized vitiligo, and 8% presented with the acrofacial form of the disease. Statistical analyses performed using the Independent Samples T-test and the χ^2 test confirmed that these variables were comparable ($p\leq0.05$).

The genotype and allele distributions for the IL-6 -572G/C polymorphism are presented in Table 3. In the vitiligo group, the genotype frequencies were 80% GG, 20% CG, and 0% CC, compared to 60% GG, 38% CG, and 2% CC in the control group. While the differences in genotype frequencies were not statistically significant ($p=0.07$), the GG genotype was associated with a 2.53-fold increased risk of vitiligo [$p=0.04$, OR (95% confidence interval, CI) = 2.53 (1.03–6.23)]. In contrast, the CG genotype exhibited a protective effect [$p=0.04$, OR (95% CI) = 0.39 (0.16–0.97)]. A significant difference was observed in allele frequencies ($p=0.03$). The G allele was more prevalent among vitiligo patients (53.3%) compared to controls (46.7%), conferring an increased risk of the disease [$p=0.03$, OR (95% CI) = 2.39 (1.06–5.38)]. Conversely, the C allele was less frequent in patients (32.3%) compared to controls (67.7%) and was associated with a reduced risk of vitiligo [$p=0.03$, OR (95% CI) = 0.42 (0.19–0.94)].

Table 2. Clinical data of the studied vitiligo patients and controls

Characteristics	Vitiligo patients (n=50)		Controls (n=50)		p
Age (years)	40.5±17.6		40.7±13.5		0.97
Age of disease onset (years)	33.0±18.6		–		–
Disease duration (months)	90.4±140.3		–		–
	n	%	n	%	
Gender					
Male	19	38	19	38	1.000
Female	31	62	31	62	
Family history of vitiligo					
Yes	17	34	–	–	–
No	33	66	–	–	–
Type of vitiligo					
Generalized	40	80	–	–	–
Localized	6	12	–	–	–
Acrofacial	4	8	–	–	–
Age of vitiligo onset					
Early-onset (<20 years)	15	30	–	–	–
Late-onset (≥20 years)	35	70	–	–	–

Age, age of disease onset, and disease duration are presented as mean±standard deviation (SD) and were analyzed using the Independent Student t-test. Proportions n (%) were analyzed using the Chi-Square (χ^2) test. Significance between groups: p≤0.05.

Table 3. Genotype and allele distribution in the vitiligo patient and control groups for the IL-6 (-572 G/C) gene polymorphism

Genotype	Vitiligo patients		Controls		OR (95% CI)	OR (95% CI)	p
	n	%	n	%			
GG	40	80	30	60	2.53 (1.03–6.23)	1 (reference)	0.04
CG	10	20	19	38	1 (reference)	0.39 (0.16–0.97)	
CC	0	0	1	2	–		
	χ^2=5.22, df=2, p=0.07						
Allele							
G	90	90	79	79	2.39 (1.06–5.38)	1 (reference)	0.03
C	10	10	21	21	1 (reference)	0.42 (0.19–0.94)	
	χ^2=4.61, df=1, p=0.03						

Significance between groups: p≤0.05. Analysis performed using Pearson Chi-Square (χ^2). IL-6: Interleukin-6; OR: Odds ratio; CI: Confidence interval; df: Degrees of freedom.

Table 4 summarizes the associations between the IL-6 -572G/C polymorphism and clinical characteristics in vitiligo patients. Statistical analysis revealed no significant relationships between genotype distribution and variables such as gender, family history, type of vitiligo, or age of disease onset (p>0.05).

DISCUSSION

Vitiligo is a complex autoimmune disorder characterized by the destruction of melanocytes, leading to the development of depigmented patches on the skin.[12] The pro-inflammatory cytokine IL-6 has been recognized as a critical contributor to the

Table 4. Clinical characteristics of vitiligo patients in relation to their IL-6 (-572G/C) genotypes

	GG		CG		p
	n	%	n	%	
Gender					
Male	16	40	3	30	0.56
Female	24	60	7	70	
Family history of vitiligo					
Yes	13	32.5	4	40	0.65
No	27	67.5	6	60	
Type of vitiligo					
Generalized	33	82.5	7	70	0.64
Localized	4	10	2	20	
Acrofacial	3	7.5	1	10	
Age of vitiligo onset					
Early-onset (<20 years)	12	30	3	30	1.00
Late-onset (≥20 years)	28	70	7	70	

Data were analyzed using Pearson Chi-Square (χ^2). IL-6: Interleukin-6.

development of vitiligo and other autoimmune disorders.[13] By activating polyclonal B cells, IL-6 may disrupt melanocyte growth and promote immune-mediated damage, ultimately resulting in depigmented areas.[14] Recent studies have emphasized the interaction between genetic predisposition and environmental factors in the pathogenesis of vitiligo.[2] While the IL-6 gene has been implicated in various diseases, the specific role of the -572G/C polymorphism in vitiligo remains insufficiently explored.

This study investigated the potential influence of the IL-6 -572G/C polymorphism on vitiligo patients and its associations with demographic and clinical characteristics. The findings provide valuable insights into the intricate autoimmune mechanisms underlying vitiligo. Although no statistically significant differences in IL-6 genotypes were identified between the control and vitiligo groups, the results suggest a potential link between this polymorphism and increased susceptibility to vitiligo. The -572G/C polymorphism appears to significantly affect disease development, with the GG genotype identified as a prominent risk factor, conferring a 2.53-fold higher likelihood of vitiligo onset. These findings highlight the IL-6 gene's involvement in the disease's pathogenesis. Supporting these results, Singh et al.[13] observed a similar association in the Gujarat population, reporting a higher frequency of the GG genotype among vitiligo patients.

Our findings revealed a higher prevalence of the G allele in vitiligo patients compared to the control group, suggesting that the G allele of the IL-6 -572G/C polymorphism significantly increases the risk of developing vitiligo. A similar study conducted among vitiligo patients in the Chinese Han population also reported an increased frequency of the G allele in affected individuals. Their findings indicated that carriers of the G allele faced a 1.718-fold higher risk of developing vitiligo compared to those with the C allele. Similar to our observations, the Chinese study did not identify any association between the IL-6 rs1800796 polymorphism and the clinical features of vitiligo patients.[2]

The IL-6 -572G/C gene polymorphism has been studied in relation to various diseases, yielding mixed outcomes. Research conducted in Pakistan and Egypt reported an increased prevalence of the variant genotypes (CG+CC) at the -572G/C locus of the IL-6 gene among individuals with acne.[15,16] Interestingly, significant differences were observed between these populations. In the Pakistani cohort, the CG polymorphism was strongly associated with acne vulgaris, whereas in the Egyptian population, the CC polymorphism was markedly more frequent in patients compared to controls. These discrepancies may reflect the influence of diverse ethnic backgrounds in the respective studies.

A meta-analysis focusing on patients with systemic lupus erythematosus (SLE) identified a significant association between the IL-6 -572G/C polymorphism and the recessive form of the disease. However, no such association was observed when analyzing alleles or under the dominant genetic model.[17] Another meta-analysis reinforced the association between IL-6 polymorphism and an increased risk of SLE, indicating that the -572G/C variant might contribute to susceptibility to this condition.[18]

In contrast, a study conducted on Chinese children concluded that the IL-6 -572G/C polymorphism does not influence susceptibility to atopic dermatitis, underscoring the variability in the role of this polymorphism across different diseases.[19]

Melanocytes produce IL-6, which promotes the expression of intercellular adhesion molecule-1 (ICAM-1) on their surface. This elevated ICAM-1 expression facilitates the attachment of leukocytes to melanocytes, potentially leading to melanocyte damage and contributing to the pathogenesis of vitiligo.[20] Although serum IL-6 levels were not assessed in our study, previous research has consistently reported elevated IL-6 levels in the serum of vitiligo patients. One study specifically observed increased IL-6 levels in the unaffected skin of vitiligo patients.[21] Moreover, multiple studies have confirmed significant elevations in serum IL-6 concentrations among individuals with vitiligo.[22,23]

Our study indicates that the IL-6 -572G/C gene polymorphism, particularly the GG genotype and the G allele, is associated with an increased risk of developing vitiligo in the Turkish population. These findings highlight the potential involvement of IL-6-mediated pathways in the pathogenesis

of the disease.[20,22] Identifying such genetic associations may pave the way for personalized diagnostic strategies, including genetic screening to detect high-risk individuals, especially those with a familial history of vitiligo. Early identification of genetic susceptibility could facilitate targeted surveillance and the implementation of preventive measures.

IL-6's role in the inflammatory pathways associated with vitiligo suggests its potential as a therapeutic target. Interventions aimed at regulating IL-6 activity, such as monoclonal antibodies or cytokine inhibitors, could offer promising strategies to slow disease progression or prevent melanocyte damage.[20,22] Considering IL-6's critical role in inflammation, these treatments may represent innovative approaches for managing disease activity in vitiligo patients.[5] However, further research is necessary to evaluate the efficacy of IL-6-targeted therapies in halting or reversing the condition. While these findings are preliminary, they provide a foundation for future studies to investigate the potential benefits of IL-6 modulation in vitiligo treatment.

CONCLUSION

This study emphasizes the significant role of the IL-6 -572G/C polymorphism in determining susceptibility to vitiligo in the Turkish population. The G allele and GG genotype were found to be closely associated with an increased risk of developing the disease. These findings underscore the need for further research to explore IL-6-mediated pathways and their potential as therapeutic targets. Future studies involving larger and more diverse cohorts will help refine our understanding and support the development of novel interventions to mitigate disease progression and improve patient outcomes.

Acknowledgements: The authors would like to thank the patients and healthy subjects who willingly participated in the study.

Ethics Committee Approval: The Kutahya Health Sciences University Faculty of Medicine Non-interventional Clinical Research Ethics Committee granted approval for this study (date: 11.10.2023, number: 2023/11-26).

Author Contributions: Concept – FY, RA, NDN; Design – FY, RA, NDN; Supervision – FY, RA; Resource – FY, RA; Materials – FY, RA; Data Collection and/or Processing – FY, RA, NDN; Analysis and/or Interpretation – FY, RA, NDN; Literature Search – FY; Writing – FY; Critical Reviews – FY, RA.

Conflict of Interest: The authors have no conflict of interest to declare.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Use of AI for Writing Assistance: Not declared.

Financial Disclosure: The authors declared that this study has received no financial support.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Kang P, Wang Y, Chen J, Chang Y, Zhang W, Cui T, et al. TRPM2-dependent autophagy inhibition exacerbates oxidative stress-induced CXCL16 secretion by keratinocytes in vitiligo. J Pathol 2024; 262(4): 441-53. [CrossRef]
2. Wang D, Min S, Lin X, Jiang G. Association among MIF, IFIH1, and IL6 gene polymorphisms and non-segmental vitiligo in a chinese han population. Clin Cosmet Investig Dermatol 2022; 15: 1597-609. [CrossRef]
3. Liu L, Xue Y, Li Y, Chen Y, Pan X, Huang Y, et al. Genome-wide DNA methylation of lesional and peri-lesional skin in vitiligo: A comparative and integrated analysis of multi-omics in Chinese population. Hum Genet 2024; 143(2): 137-49. [CrossRef]
4. Perez-Bootello J, Cova-Martin R, Naharro-Rodriguez J, Segurado-Miravalles G. Vitiligo: Pathogenesis and new and emerging treatments. Int J Mol Sci 2023; 24(24): 17306. [CrossRef]
5. Tanaka T, Narazaki M, Kishimoto T. IL-6 in inflammation, immunity, and disease. Cold Spring Harb Perspect Biol 2014; 6(10): a016295. [CrossRef]
6. Swaroop AK, Negi P, Kar A, Mariappan E, Natarajan J, Namboori P K K, et al. Navigating IL-6: From molecular mechanisms to therapeutic breakthroughs. Cytokine Growth Factor Rev 2024; 76: 48-76. [CrossRef]
7. Shahub S, Lin KC, Muthukumar S, Prasad S. A proof-of-concept electrochemical skin sensor for simultaneous measurement of glial fibrillary acidic protein (GFAP) and interleukin-6 (IL-6) for management of traumatic brain injuries. Biosensors (Basel) 2022; 12(12): 1095. [CrossRef]
8. Farhan J, Al-Shobaili HA, Zafar U, Al Salloom A, Meki AR, Rasheed Z. Interleukin-6: A possible inflammatory link between vitiligo and type 1 diabetes. Br J Biomed Sci 2014; 71(4): 151-7. [CrossRef]
9. Desai K, Kumar HK, Naveen S, Somanna P. Vitiligo: Correlation with cytokine profiles and its role in novel therapeutic strategies: A case-control study. Indian Dermatol Online J 2023; 14(3): 361-5. [CrossRef]
10. Al-Harthi F, Huraib GB, Mustafa M, Al-Qubaisy Y, Al-Nomair N, Abdurrahman N, et al. Inducible nitric oxide synthase iNOS-954-G>C and Ex16+14-C>T gene polymorphisms and susceptibility to vitiligo in the Saudi population. Pharmgenomics Pers Med 2022; 15: 603-12. [CrossRef]
11. Barartabar Z, Nikzamir A, Sirati-Sabet M, Aghamohammadi E, Chaleshi V, Rostami Nejad M, et al. The relationship between 174 G/C and -572 G/C of IL-6 gene polymorphisms and susceptibility of celiac disease in the Iranian population. Prz Gastroenterol 2018; 13(4): 293-8. [CrossRef]

12. Joge RR, Kathane PU, Joshi SH. Vitiligo: A narrative review. Cureus 2022; 14(9): e29307. [CrossRef]
13. Singh M, Jadeja SD, Vaishnav J, Mansuri MS, Shah C, Mayatra JM, et al. Investigation of the role of interleukin 6 in vitiligo pathogenesis. Immunol Invest 2022; 51(1): 120-37. [CrossRef]
14. Morelli JG, Norris DA. Influence of inflammatory mediators and cytokines on human melanocyte function. J Invest Dermatol 1993; 100(2): 191S-5. [CrossRef]
15. Younis S, Javed Q. The interleukin-6 and interleukin-1A gene promoter polymorphism is associated with the pathogenesis of acne vulgaris. Arch Dermatol Res 2015; 307(4): 365-70. [CrossRef]
16. Ragab M, Hassan EM, Elneily D, Fathallah N. Association of interleukin-6 gene promoter polymorphism with acne vulgaris and its severity. Clin Exp Dermatol 2019; 44(6): 637-42. [CrossRef]
17. Yang Z, Liang Y, Qin B, Zhong R. A meta-analysis of the association of IL-6 -174 G/C and -572 G/C polymorphisms with systemic lupus erythematosus risk. Rheumatol Int 2014; 34(2): 199-205. [CrossRef]
18. Cui YX, Fu CW, Jiang F, Ye LX, Meng W. Association of the interleukin-6 polymorphisms with systemic lupus erythematosus: A meta-analysis. Lupus 2015; 24(12): 1308-17. [CrossRef]
19. Huang S, Wang H, Zheng H, Li W, Shi J, Shen C, et al. Association between IL-6 polymorphisms and atopic dermatitis in Chinese Han children. Front Pediatr 2023; 11: 1156659. [CrossRef]
20. Abdallah M, El-Mofty M, Anbar T, Rasheed H, Esmat S, Al-Tawdy A, et al; Egyptian Vitiligo Group. CXCL-10 and interleukin-6 are reliable serum markers for vitiligo activity: A multicenter cross-sectional study. Pigment Cell Melanoma Res 2018; 31(2): 330-6. [CrossRef]
21. Kovacs D, Bastonini E, Ottaviani M, Cota C, Migliano E, Dell'Anna ML, et al. Vitiligo skin: Exploring the dermal compartment. J Invest Dermatol 2018; 138(2): 394-404. [CrossRef]
22. Singh S, Singh U, Pandey SS. Serum concentration of IL-6, IL-2, TNF-α, and IFNγ in Vitiligo patients. Indian J Dermatol 2012; 57(1): 12-4. [CrossRef]
23. Moretti S, Spallanzani A, Amato L, Hautmann G, Gallerani I, Fabiani M, et al. New insights into the pathogenesis of vitiligo: Imbalance of epidermal cytokines at sites of lesions. Pigment Cell Res 2002; 15(2): 87-92. [CrossRef]