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Original Research Article

Assessment of inflammatory cytokines (IL-6, TNF-α) in patients with Vitiligo: A Cross-Sectional study

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Abstract:

Background: Vitiligo is a chronic autoimmune depigmenting disorder characterized by melanocyte destruction, with increasing evidence supporting the role of inflammatory cytokines in its pathogenesis.

Aim: To compare serum levels of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) in patients with vitiligo and healthy controls, and to assess their correlation with disease activity.

Methodology: This hospital-based comparative cross-sectional study included 80 participants (40 vitiligo patients and 40 age- and sex-matched controls). Serum IL-6 and TNF-α levels were measured using ELISA. Disease activity was evaluated using the Vitiligo Disease Activity (VIDA) score. Statistical analysis was performed using SPSS version 27.0.

Results: Mean serum IL-6 (18.62 ± 6.84 pg/mL) and TNF-α (32.15 ± 9.72 pg/mL) levels were significantly higher in vitiligo patients compared to controls (9.47 ± 3.95 pg/mL and 17.36 ± 5.88 pg/mL, respectively; $p < 0.001$). Both cytokines showed significant positive correlation with VIDA score (IL-6: $r = 0.58$; TNF-α: $r = 0.49$).

Conclusion: Elevated IL-6 and TNF-α levels and their association with disease activity support the role of systemic inflammation in vitiligo and highlight their potential as biomarkers.

Keywords: Vitiligo, IL-6, TNF-α, Cytokines, Inflammation, VIDA score.

Introduction

Vitiligo represents a long-lasting skin disorder which causes skin color loss through its progressive destruction of skin cells that produce pigment, resulting in the formation of distinct white skin patches and white patches on mucous surfaces [1]. The medical community has transitioned its view of vitiligo from a mere cosmetic issue to a complex disease which combines autoimmune and inflammatory properties and creates various psychological and social and systemic health effects. The global prevalence of vitiligo ranges from 0.5% to 2% of the population, affecting individuals of all ages, genders, and ethnic backgrounds, with no clear predilection [2]. The disease can start at any age, but most people develop it during their twenties and thirties.

Vitiligo etiopathogenesis is multifactorial and not well understood. A number of theories have been put forward such as autoimmune, oxidative stress, neural, genetic and melanocyte self-destruction theories [3]. Of these, the autoimmune hypothesis has received the most support, as can be seen through the presence of circulating autoantibodies to melanocyte-specific antigens, association to other autoimmune diseases like autoimmune thyroid disease and type 1 diabetes mellitus, and positive response to immunomodulatory treatment. There is growing evidence to support the idea that vitiligo is caused by an overreaction of the immune system against melanocytes, which culminates in the destruction of melanocytes by the cytotoxic effects of T lymphocytes and inflammatory cytokines.

Inflammation is instrumental in the process of melanocytic loss activation and sequence in vitiligo. Over the last few years, the role of the pro-inflammatory cytokines in orchestrating immune response that causes melanocyte damage has been in focus. Cytokines are soluble small sized proteins that are released by immune and non-immune cells which control immunity, inflammation and hematopoiesis. Interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-a) are among the other group of cytokines that

have been found to mediate the pathogenesis of vitiligo [4].

Interleukin-6 (IL-6) is a pleiotropic cytokine that is expressed by the keratinocytes, macrophages, T cells and fibroblasts in reaction to infection, stress and tissue damage [5]. It is crucial in both innate and adaptive immune responses because it enhances the B-cell differentiation, T-cell activation, and acute-phase protein synthesis. Within the framework of vitiligo, it was demonstrated that IL-6 levels in lesional skin and serum of patients are increased and imply its role in local and systemic inflammation [6]. It is thought that IL-6 plays a role in the pathogenesis of melanocyte dysfunction by aggravating inflammatory infiltration of cells, stimulating the production of Th17, and enhancing its autoimmune reactions to melanocytes. Also, IL-6 can disrupt the process of melanogenesis and the survival of melanocytes thus worsening depigmentation.

Tumor necrosis factor-alpha (TNF-α) serves as a strong pro-inflammatory cytokine which activated macrophages and dendritic cells and T lymphocytes primarily produce [7]. TNF-α produces multiple biological effects which include apoptosis induction and immune cell activation control and inflammatory signaling pathway alteration. TNF-α causes vitiligo by blocking melanocyte growth and preventing their pigment production while it causes their cell death through NF-κB activation together with other signaling pathways. Researchers have found that vitiligo patients show increased serum and tissue TNF-α levels which correspond to their disease activity and extent of skin lesions [8]. The beneficial effects which anti-TNF drugs provide for specific inflammatory conditions demonstrate that this cytokine plays a critical role in diseases which involve immune-mediated mechanisms.

The combination of IL-6 and TNF-a should be of special interest because these two cytokines are capable of interacting to enhance the intensity of the inflammatory cascades [9]. TNF-a is able to provoke IL-6 generation of the keratinocytes and other local skin cells and this helps to maintain a chronic inflammatory microenvironment. This chronic inflammation can result in further lesion of melanocytes and further development of depigmented areas. Besides, the established factor of vitiligo pathogenesis is oxidative stress that may also potentiate the release of cytokines, making the inflammatory process and cell damage a vicious cycle.

Research shows that vitiligo leads to cytokine imbalance which affects both the skin and the entire immune system of the body. The increased levels of IL-6 and TNF-α in the bloodstream indicate that vitiligo produces wider immunological effects which extend beyond the visible skin discoloration. The comparison of serum cytokine levels between vitiligo patients and healthy subjects will produce insights which help to understand the disease process and its current status and potential treatment points. The biomarkers IL-6 and TNF-α can function as disease progression indicators which help to develop personalized treatment plans and provide early diagnostic support.

Across different studies, researchers have found inconsistent results about inflammatory cytokines in vitiligo because the studies used different participant numbers and disease duration and clinical subtype and research methods. The research needs to compare cytokine levels between vitiligo patients and healthy controls because this comparison will help establish how these substances specifically drive disease development and it will provide stronger proof of their role.

The research compares the levels of inflammatory cytokines IL-6 and TNF-α between vitiligo patients and their age- and sex-matched healthy controls. The study investigates vitiligo-related systemic inflammation by measuring serum cytokine levels which also serve as potential disease activity biomarkers. Researchers aim to understand vitiligo inflammatory environments because it will help them discover immunopathogenic mechanisms and create new immunotherapy treatments".

Methodology

Study Design: This hospital-based comparative cross-sectional study was conducted to evaluate and compare serum levels of inflammatory cytokines, interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), in patients with vitiligo and healthy controls.

Study Area: The study was carried out in the Department of Skin & VD and Biochemistry, Sri Krishna Medical College and Hospital, Muzaffarpur, Bihar, India.

Study Duration: The duration of the study was 7 months from March 2025 to September 2025.

Study Participants: A total of 80 participants were enrolled in the study and divided into two groups: 40 clinically diagnosed cases of vitiligo and 40 age- and sex-matched healthy controls.

Inclusion Criteria

- Patients of either gender aged 18 years and above.
- Clinically diagnosed cases of vitiligo attending the Dermatology outpatient department.
- Patients who had not received systemic or topical immunomodulatory therapy for at least 6 weeks prior to enrollment (wash-off period).
- Patients willing to provide written informed consent.

- For controls: Healthy individuals without any dermatological or systemic inflammatory disease and willing to participate.

Exclusion Criteria

- Patients with other inflammatory or autoimmune skin disorders such as psoriasis.
- Patients with systemic inflammatory diseases, chronic infections, or malignancies.
- Patients on immunosuppressive or corticosteroid therapy within the last 6 weeks.
- Pregnant or lactating women.
- Individuals unwilling to provide consent.

Sample Size: The total sample size was 80 participants, comprising 40 patients with vitiligo and 40 healthy controls.

Procedure: Eligible patients attending the Dermatology outpatient department were screened for inclusion. A detailed clinical history was recorded, and a thorough dermatological examination was performed to confirm the diagnosis of vitiligo. Classification of vitiligo was done based on the extent of involvement into localized (focal, segmental, and mucosal) and generalized (vulgaris, acrofacial, and universalis) types. Disease activity was assessed using the Vitiligo Disease Activity (VIDA) score, a six-point scale ranging from +4 (activity ≤6 weeks) to −1 (stable with spontaneous repigmentation for ≥1 year). Stable vitiligo was defined as no new lesions and no progression of existing lesions for at least one year, whereas active vitiligo included cases with new or enlarging lesions.

After informed consent and under strict aseptic precautions, 5 mL of venous blood was collected from each participant (both cases and controls). Blood samples were allowed to clot and were centrifuged to separate the serum. The separated serum was stored at −20°C until analysis. Serum levels of IL-6 and TNF-α were measured using commercially available enzyme-linked immunosorbent assay (ELISA) kits based on the sandwich ELISA principle. Cytokine estimation was performed using an ELISA microplate reader according to the manufacturer's instructions. All assays were carried out in duplicate to ensure accuracy and reliability of results.

Healthy controls were selected randomly from individuals accompanying patients or hospital staff after ensuring they had no history of vitiligo or other inflammatory conditions. Blood collection and cytokine estimation procedures for the control group were performed in the same manner as for the cases to maintain uniformity.

All clinical and laboratory data were recorded systematically in a predesigned proforma for subsequent statistical analysis.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 27.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. The independent t-test was used to compare mean cytokine levels between vitiligo patients and healthy controls. The chi-square test was applied for categorical variables. Correlation analysis was performed to assess the relationship between cytokine levels and disease activity (VIDA score). A p-value of less than 0.05 was considered statistically significant".

Result

Table 1 shows the demographic characteristics of the study participants, including 40 patients with vitiligo and 40 healthy controls. The mean age of the vitiligo group was 34.8 ± 10.6 years, while that of the control group was 33.9 ± 9.8 years, and the difference was statistically non-significant (p = 0.68), indicating that both groups were comparable in terms of age distribution. Regarding gender distribution, 22 (55%) participants in the vitiligo group were males and 18 (45%) were females, whereas in the control group, 21 (52.5%) were males and 19 (47.5%) were females. The difference in gender distribution between the two groups was also statistically non-significant (p = 0.82). Overall, the findings suggest that the study groups were well matched for age and gender, minimizing potential confounding effects of these demographic variables on the study outcomes.

Table 1: Demographic Characteristics of Study Participants (N = 80)

Variable	Vitiligo (n = 40)	Controls (n = 40)	p-value
Age (years), Mean ± SD	34.8 ± 10.6	33.9 ± 9.8	0.68
Gender (Male), n (%)	22 (55%)	21 (52.5%)	0.82
Gender (Female), n (%)	18 (45%)	19 (47.5%)	0.82

Table 2 shows the clinical characteristics of vitiligo patients (n = 40). Among the study participants, generalized vitiligo was more common, observed in 24 patients (60%), whereas localized vitiligo was present in 16 patients (40%), indicating a predominance of widespread disease in the study population. Regarding disease activity assessed by the VIDA score, the highest proportion of patients belonged to the +2 category (3–6 months of activity), accounting for 10 cases (25%), followed by the +3 category (6 weeks–3 months) with 8 patients (20%). Stable disease for ≥1 year (VIDA 0) was also noted in 8

patients (20%). Recent and highly active disease (VIDA +4, ≤6 weeks) was seen in 6 patients (15%), while 6 patients (15%) had activity lasting 6–12 months (VIDA +1). Only 2 patients (5%) demonstrated stability with repigmentation (VIDA −1). Overall, most patients exhibited varying degrees of active disease rather than complete stability.

Table 2: Clinical Characteristics of Vitiligo Patients (n = 40)		
Variable	**Frequency (n)**	**Percentage (%)**
Type of Vitiligo		
Localized	16	40%
Generalized	24	60%
Disease Activity (VIDA Score)		
+4 (≤6 weeks)	6	15%
+3 (6 weeks–3 months)	8	20%
+2 (3–6 months)	10	25%
+1 (6–12 months)	6	15%
0 (Stable ≥1 year)	8	20%
−1 (Stable with repigmentation)	2	5%

Table 3 shows the comparison of serum IL-6 levels between vitiligo patients and healthy controls. The mean serum IL-6 level in the vitiligo group (n = 40) was 18.62 ± 6.84 pg/mL, which was markedly higher than that observed in the control group (n = 40), where the mean value was 9.47 ± 3.95 pg/mL. The calculated t-value of 7.12 indicates a strong difference between the two groups. Moreover, the p-value was <0.001, demonstrating that the difference in serum IL-6 levels was highly statistically significant. These findings suggest that serum IL-6 levels are significantly elevated in patients with vitiligo compared to healthy individuals, indicating a possible role of inflammatory cytokines in the pathogenesis of vitiligo.

Table 3: Comparison of Serum IL-6 Levels Between Groups			
Group	**Mean ± SD (pg/mL)**	**t-value**	**p-value**
Vitiligo (n = 40)	18.62 ± 6.84	7.12	<0.001
Controls (n = 40)	9.47 ± 3.95		

Table 4 shows the comparison of mean serum TNF-α levels between vitiligo patients and healthy controls. The mean serum TNF-α level in the vitiligo group (n = 40) was 32.15 ± 9.72 pg/mL, which was markedly higher than that observed in the control group (n = 40), where the mean value was 17.36 ± 5.88 pg/mL. The calculated t-value of 6.48 indicates a substantial difference between the two groups. Furthermore, the p-value of <0.001 demonstrates that this difference is highly statistically significant. These findings suggest that serum TNF-α levels are significantly elevated in patients with vitiligo compared to healthy individuals, indicating a possible role of inflammatory mechanisms in the pathogenesis of the disease.

Table 4: Comparison of Serum TNF-α Levels Between Groups			
Group	**Mean ± SD (pg/mL)**	**t-value**	**p-value**
Vitiligo (n = 40)	32.15 ± 9.72	6.48	<0.001
Controls (n = 40)	17.36 ± 5.88		

Table 5 shows the correlation between serum cytokine levels and disease activity measured by the VIDA score in vitiligo patients (n = 40). The analysis demonstrates a moderate to strong positive correlation between IL-6 levels and VIDA score (r = 0.58) with a highly significant p-value (<0.001), indicating that higher IL-6 concentrations are significantly associated with increased disease activity. Similarly, TNF-α levels also show a moderate positive correlation with the VIDA score (r = 0.49) and a statistically significant p-value (0.002), suggesting that elevated TNF-α is linked with greater disease progression. Overall, both cytokines exhibit significant positive correlations with disease activity, implying that IL-6 and TNF-α may play an important role in the inflammatory process and severity of vitiligo.

Table 5: Correlation of Serum Cytokine Levels with Disease Activity (VIDA Score) in Vitiligo Patients (n = 40)		
Variable	**Correlation Coefficient (r)**	**p-value**
IL-6 vs VIDA Score	0.58	<0.001
TNF-α vs VIDA Score	0.49	0.002

Discussion

The current investigation found that vitiligo patients showed increased serum IL-6 levels of 18.62 ± 6.84 pg/mL and TNF-α levels of 32.15 ± 9.72 pg/mL when compared to healthy controls who showed IL-6 levels of 9.47 ± 3.95 pg/mL and TNF-α levels of 17.36 ± 5.88 pg/mL ($p < 0.001$ for both) while showing positive correlation between cytokine levels and disease activity (IL-6: $r = 0.58$; TNF-α: $r = 0.49$). The findings provide strong evidence that vitiligo functions as an autoimmune disease which produces inflammation instead of being an isolated skin pigmentation disorder. The autoimmune basis of vitiligo has long been recognized with evidence showing that both humoral and cellular immunity become dysregulated (Ongenae et al., 2003) [10]. Our observation of elevated systemic cytokines aligns with this autoimmune paradigm".

Our research results show that TNF-α levels reached their highest point which matches the results found by Singh et al. (2012) [11] who observed that vitiligo patients showed increased serum TNF-α levels when compared to their control group although the two groups did not reach statistically significant differences. Our study results showed almost double the increase which produced strong statistical results indicating that our group experienced greater systemic inflammatory response. Moretti et al. (2002) [12] demonstrated through their research that TNF-α increased in lesional epidermis which showed its role in local disease development. Our research extends this finding through complete verification of systematic elevation which supports the theory that TNF-α causes melanocyte death through both local and systemic mechanisms. Yu et al. (1997) [13] reported that active vitiligo patients showed lower TNF-α production from their peripheral mononuclear cells which contradicts our research results. The difference between our study and other research works might emerge from three factors which include sample size and disease activity status and methodology since our research focused on active disease patients who reached VIDA levels of 2 and 3 which resulted in higher cytokine measurements.

The research demonstrated that serum IL-6 levels increased significantly among vitiligo patients who participated in the study. Singh et al. (2012) found that vitiligo patients showed elevated IL-6 levels which became more pronounced in patients who had the disease for more than 15 years. The study showed that active cases made up most of the participants which caused the study to show higher IL-6 levels which reached 18.62 pg/mL. Abdallah et al. (2018) [14] discovered that IL-6 functions as a dependable serum indicator for vitiligo progress which showed higher IL-6 levels during active disease compared to stable disease. The study found a moderate-to-strong positive correlation between IL-6 and VIDA score ($r = 0.58$, $p < 0.001$) which shows that IL-6 reflects disease activity according to their research conclusion. The study by Sushama et al. (2019) [15] showed that vitiligo patients had higher IL-6 and TNF-α levels which supported the idea that pro-inflammatory cytokines cause damage to melanocytes.

The study found stronger correlations between IL-6 levels and disease activity than between TNF-α levels and disease activity. The study found that IL-6 serves as a more effective biomarker for tracking inflammatory illness progression. Studies that examined various pro-inflammatory cytokines showed indirect confirmation of this finding because they studied their connection with disease progression. Bhardwaj et al. (2017) [16] demonstrated that non-segmental vitiligo progression leads to increased systemic and epidermal IL-17A levels which indicate that Th17-mediated inflammation causes active disease. The study found no IL-17 data but researchers found that IL-6 levels rose which promotes Th17 differentiation and IL-6 elevation causes stronger Th17 responses during active vitiligo development.

Ma et al. (2013) [17] demonstrated that progressive vitiligo patients showed increased macrophage migration inhibitory factor (MIF) levels in both their serum and lesional skin which correlated with their disease severity and activity. Our research found that active disease shows increased levels of multiple pro-inflammatory mediators which includes elevated IL-6 and TNF-α that correspond to VIDA scores. The concordance between our results and previous studies on cytokine imbalance strengthens the concept that vitiligo pathogenesis involves a complex inflammatory network rather than a single cytokine pathway.

Some earlier studies produced different results because they studied IFN-γ and TNF-αboth through different sample sizes and disease classification methods which included segmental and non-segmental diseases and they used different laboratory procedures. The study results become more trustworthy because our research used a control group that matched the study group and shared similar demographic traits which reduced potential confounding factors.

Our investigation showed that elevated levels of TNF-α from our study provide evidence to support treatment approaches which target this cytokine. The case reports which Jaouad et al. (2021) [18] summarized show that anti-TNF agents lead to clinical improvement of vitiligo lesions which indicates that biologic therapy may benefit particular patients. The high levels of IL-6 which our study found in our group of subjects need further investigation through clinical trials which should test anti-IL-6 treatments.

The current research findings show strong agreement with existing research which demonstrates that vitiligo increases the production of pro-inflammatory cytokines. The significant differences in IL-6 and TNF-α levels between patients and controls, along with their positive correlations with disease activity, provide strong evidence that systemic inflammation parallels clinical progression. The observations support the autoimmune-inflammatory model of vitiligo because the researchers discovered that IL-6 and TNF-α serve as potential biomarkers which enable disease activity monitoring and as effective targets for immunomodulatory treatment.

Conclusion

The present study demonstrates that serum levels of IL-6 and TNF-α are significantly elevated in patients with vitiligo compared to healthy controls, highlighting the crucial role of systemic inflammation in the pathogenesis of the disease. The strong statistical significance observed for both cytokines, along with their positive correlation with disease activity (VIDA score), indicates that higher cytokine levels are associated with increased disease progression and activity. IL-6 showed a relatively stronger correlation with disease activity than TNF-α, suggesting its potential as a more sensitive biomarker for monitoring inflammatory status in vitiligo. Overall, these findings reinforce the concept of vitiligo as an immune-mediated inflammatory disorder and suggest that IL-6 and TNF-α may serve as valuable biomarkers and potential therapeutic targets in disease management.

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