Journal of Carcinogenesis
Covering Genesis to Therapeutics

Journal of Carcinogenesis, ISSN: 1477-3163
2025; Vol 24: Issue 1s, https://carcinogenesis.com/

CXCL10 As A Potential Marker of Disease Activity in Pediatric Vitiligo

Faten Abd Elwadoud Abd Elkawy[1]*, Doaa Aly Abdel-Fatah[2], Esraa Nabil Abd-Allah[1], Shaimaa Hassan Mohamed[1]

[1]Dermatology and venereology Department, Faculty of Medicine for Girls - Al-Azhar University, Cairo, Egypt.

[2]Department of Clinical Pathology, Faculty of Medicine for Girls - Al-Azhar University, Cairo, Egypt.

Corresponding Author: Shaimaa Hassan Mohamed

Email: shaimaadiab08@gmail.com

ABSTRACT

Introduction: Vitiligo is a common acquired depigmentary inflammatory disorder associated with negative psychosocial impacts in children and their caregivers. C-X-C motif chemokine ligand 10 [CXCL10] is related to the Th1 response that facilitates T cell migration in many autoimmune conditions & augments melanocyte-targeted cytotoxic T lymphocyte infiltration into the epidermis to assault melanocytes, leading to melanin deficit.

Objective: The aim of this research was to estimate serum CXCL10 concentration in pediatric vitiligo cases as an indicator of disease activity.

Methods: Serum CXCL10 level was measured in 60 pediatric vitiligo patients [segmental vitiligo [num.=twenty], non-segmental active vitiligo [num.=22] and non-segmental stable vitiligo [n=18]], & 30 age and sex matched healthy control using enzyme- linked immunosorbent assay [ELISA]. Vitiligo severity was estimated by vitiligo area scoring index [VASI] score & vitiligo disease activity [VIDA] score.

Results: Serum CXCL10 level was significantly greater in cases [median=42.5] than control group [median=25.8] with highest serum level in non-segmental active vitiligo group [median=53.55] followed by segmental vitiligo patients [median=41.45] then non-segmental stable vitiligo patients [median=32.3]. Statistically significant positive correlations were found between serum CXCL10 level with VIDA score and VASI score [p=0.001].

Conclusion: Serum CXCL10 increased significantly in pediatric vitiligo patients, with significant positive correlation with VIDA and VASI scores.

Keywords: *Pediatric vitiligo, CXCL10, VIDA score, VASI score*

How to Cite: Faten Abd Elwadoud Abd Elkawy , Doaa Aly Abdel-Fatah , Esraa Nabil Abd-Allah , Shaimaa Hassan Mohamed , (2025) CXCL10 as a potential marker of disease activity in pediatric vitiligo, *Journal of Carcinogenesis*, *Vol.24, No.1s*, 133-140

1. INTRODUCTION

Vitiligo is a persistent, acquired pigmentation condition that is found globally. The occurrence of vitiligo is one percent and fluctuates between 0.1 percent and over 8.8 percent across various global regions **[1]**.

In vitiligo, there is a targeted & gradual depletion of active melanocytes from the basal layer of the epidermis & hair follicles, resulting in clinically observable depigmented areas on the skin and mucous membranes **[2]**.

Non-segmental vitiligo, which includes vitiligo vulgaris, vitiligo universalis, and acro-facial vitiligo, segmental vitiligo, mixed vitiligo, and unclassified vitiligo, which includes focal and mucosal vitiligo, are the clinical classifications that are used to describe vitiligo. It is also possible to have follicular vitiligo, vitiligo punctata, and hypochromic vitiligo, which are all additional subtypes. **[1]**.

Pediatric vitiligo (PV) is characterized as vitiligo that commences prior to the age of 12. It is prevalent and may vary from adult vitiligo regarding epidemiology, clinical manifestation, associated conditions, and therapeutic alternatives **[3]**.

© 2025 Journal of Carcinogenesis | Published for Carcinogenesis Press by Wolters Kluwer-Medknow

The etiology of vitiligo includes the interaction of intrinsic and extrinsic melanocyte abnormalities, innate immunological inflammation, and T cell-induced melanocyte annihilation **[4]**.

The immune system is pivotal in the development of vitiligo via Th17, Th1, cytotoxic & regulatory T cells, as well as dendritic cells **[5]**. CXCL 10, Interferon-γ, and interleukin-22 are asserted to have a crucial function **[6]**.

The C-X-C motif chemokine receptor 3 [CXCR3] and its ligands, primarily CXCL 10 together with CXCL 11 and CXCL 9, are proposed as a significant chemokine axis that facilitates T-cell migration in many inflammatory and autoimmune conditions **[7]**.

CXCL 10 is a chemokine synthesized in response to IFN-γ. It interacts with its receptor, CXCR-3, resulting in the recruitment and self-activation of CD8+ T cells, which may contribute to the obliteration of melanocytes in vitiligo-affected areas **[8]**.

Prior research indicated an increase in serum CXCL 10 levels among non-segmental vitiligo (NSV) cases relative to healthy controls, especially during the progressive phases **[9]**. Additional research indicated that CXCL 10 is essential for the advancement and sustenance of depigmentation in a mouse model of vitiligo. **[10]**.

2. OBJECTIVES

The aim of the current research was estimation of serum CXCL10 as a marker of disease activity in pediatric vitiligo.

3. METHODS

This is a case control research that has been done on 90 Egyptian children divided into 60 vitiligo cases and 30 healthy individuals as control group. Cases have been divided in to 3 subgroups, segmental vitiligo [n=18], non-segmental active vitiligo [num.=22] and non-segmental stable vitiligo [num.=twenty]. The cases were randomly selected from the Dermatology outpatient clinic of Al-Zahra University Hospital, through the period from September 2022 to February 2024.

Patients known to have any systemic or dermatological disorders that increase CXCL10 level as: Lupus erythematous, psoriasis or extensive dermatitis. Patients with other autoimmune diseases as T1DM, alopecia aerate and thyroid disease, and patients who received systemic or topical treatment for vitiligo one month prior to the study, were excluded from the research.

All the cases have been subjected to complete history taking involving: sex, name and the age. Onset, site, course, duration of vitiligo. possible causes, exacerbating factors, previous lines of treatment, previous investigations, concurrent medical problems, past history and family history of similar condition.

The patients were examined clinically to determine the site, shape, size, number and distribution of the Vitiligo and then photographed with digital camera for documentation. Wood's light examination was done in order to give better information about the extent and activity of vitiligo patches. Also, routine laboratory investigations were done to exclude unfit patients.

VIDA score as well as, VASI score, were calculated to assess disease activity, the extension of depigmentation and the percentage of vitiligo involvement.

In relation to the assessment of CXCL-10, two milliliters of venous blood were obtained from all participants under stringent aseptic conditions utilizing a plastic disposable syringe, thereafter transferred to EDTA-coated anticoagulant tubes. samples were subsequently subjected to centrifugation for twenty minutes at 2000-3000 RPM. Subsequently, the supernatant devoid of sediment was gathered into Eppendorf tubes and preserved at -80°C until the assay was conducted. The plasma levels of CXCL10 were assessed with a CXCL10 ELISA kit. The plate has been pre-treated with Human CXCL10 antibody. CXCL10 present in the specimen is introduced and attaches to antibodies. Subsequently, biotinylated Human CXCL10 Antibody is introduced and attaches to CXCL10 present in the sample. Subsequently, Streptavidin-HRP is added and binds to the Biotinylated CXCL10 antibody. Following incubation, unbound Streptavidin-HRP is eliminated using a washing procedure. The substrate solution is subsequently introduced, leading to color development that correlates with the quantity of Human CXCL10. The concentration of CXCL10 has been determined based on the optical density value of the sample in relation to the standard curve.

© 2025 Journal of Carcinogenesis | Published for Carcinogenesis Press by Wolters Kluwer-Medknow

Ethical Considerations:
The research has been conducted with the endorsement of the local Ethics Committee at the Faculty of Medicine, Al-Azhar University, Cairo, Egypt. Informed written consent has been acquired from all cases. The research has been performed according to the Helsinki guidelines as updated in 2013.

Statistical methods:
The data processing was done utilizing SPSS [Statistical Package for Social Sciences] version 28.0 for Windows. Continuous parameter expressed as mean ± standard deviation [SD]. Categorical parameters displayed as frequencies and percentages. The Chi-square test has been utilized to analyze differences across several groups about categorical parameters. Fisher's Exact or Monte Carlo adjustment tests have been utilized for chi-square analysis when over twenty percent of the cells had an anticipated count below 5. The F-test [ANOVA] has been utilized to analyze differences among several groups for quantitative parameters that follow a normal distribution. The Mann Whitney test has been utilized to compare two study groups for quantitatively variable data that is not regularly distributed. P-values below 0.05 have been deemed significant.

4. RESULTS

In the patient group, the children age ranged from 4-16 years with a mean± SD of 10.58 ± 2.81. Whereas the age of controls ranged from 6-17 years with a mean ± SD of 11.87 ± 3.44. There was a female predominance in both groups, also, most of children had skin phototype III and IV. With statistically insignificant difference between patient and control groups according to sex, age and skin photo type [p= 0.155, 0.062 and 0.436], respectively. (**Table 1)**

As regard clinical presentation of vitiligo among the studied patients in (**Table 2)**, 20 patients [33.3%] had segmental vitiligo, 18 patients [30.0%] had non-segmental stable vitiligo and 22 patients [36.7%] had non-segmental active vitiligo. The range of VIDA score was [-1 - 4] with median [IQR]: 2[-1 -3], and the range of VASI score was 0.02-6.25 with median [IQR]: 0.5 [0.15 – 1.71].

As shown in (**Table 3)** With reference to estimation of serum level of CXCL10, there was highly statistically significant rise in serum CXCL-10 concentration in cases when than control group [p= 0.005].

Moreover, Receiver operative characteristic [ROC] data of serum CXCL10 showed that the best cut off point for serum CXCL10 concentration to distinguish among patient & control group was >25.9 with sensitivity 86.67, specificity 53.33 and AUC 0.682, as shown in (**Figure 1).**

With reference to relation among serum level of CXCL10 & disease activity, there was greatly statistically significant variance among cases with non-segmental active vitiligo, segmental vitiligo, non-segmental stable vitiligo and control group in serum CXCL10 [p= 0.003]. Non segmental active vitiligo patients had the highest serum CXCL10 level with median [IQR]: 53.55 [42.1 - 102.4] followed by segmental vitiligo with median [IQR]: 41.45[32.7 - 58.2] then non-segmental stable with median [IQR]: 32.3 [22.9 - 46.6] in comparison to control group with median [IQR]:25.8 [18.7 - 43.7], as shown in (**Table 4).**

Moreover, ROC data of serum CXCL10 revealed that the best cut off point for serum CXCL10 to differentiate among non-segmental active and non-segmental stable vitiligo patient was >42 with sensitivity 77.27, specificity 66.67 and AUC 0.708, as shown in (**Figure 2).**

With reference to relation among serum level of CXCL10 and disease severity, there was a statistically significant positive association among VIDA and serum CXCL10 in non-segmental active vitiligo patients [p equal to 0.000]. There was also statistically significant positive association among VASI score and serum CXCL10 in non-segmental active vitiligo patients [p= 0.005], as shown in (**Table 5).**

Table [1]: Demographic data of the studied patients.

		Patients group	**Control group**	**P-value**
		No. = 60	**No. = 30**	
Sex	Female Male	37 [61.7%] 23 [38.3%]	23 [76.7%] 7 [23.3%]	0.155
Age [years]	Mean ± SD Range	10.58 ± 2.81 4 – 16	11.87 ± 3.44 6 – 17	0.062
Skin Photo type	Type III Type IV	23 [38.3%] 37 [61.7%]	9 [30%] 21 [70%]	0.436

© 2025 Journal of Carcinogenesis | Published for Carcinogenesis Press by Wolters Kluwer-Medknow

Table [2]: Clinical presentation of vitiligo in patient group.

		Patients group
		No. = 60
Clinical type	Segmental	20 [33.3%]
	Non segmental *Stable* *Active*	**40 [66.7%]** 18 [30%] 22 [36.7%]
Duration [years]	Median [IQR] Range	2 [1 – 2] 0.17 – 7
VIDA	Median [IQR] Range	2 [-1 – 3] -1 – 4
VASI	Median [IQR] Range	0.5 [0.15 – 1.71] 0.02 – 6.25

Table [3]: Comparison between patient and control groups regarding serum level of CXCL10.

Serum CXCL10 [ng/L]	**Patients group**	**Control group**	**P-value**
	No. = 60	**No. = 30**	
Median [IQR]	42.5 [31.15 – 63.2]	25.8 [18.7 – 43.7]	0.005
Range	3.74 – 560	1.26 – 211.6	

Table [4]: Relation between serum CXCL-10 and disease activity.

Serum CXCL10 [ng/L]	**Segmental**	**Non segmental active**	**Non segmental stable**	**Control group**	**P-value**
	No. = 18	**No. = 22**	**No. = 20**	**No. = 30**	
Median [IQR]	41.45 [32.7 – 58.2]	53.55 [42.1 – 102.4]	32.3 [22.9 – 46.6]	25.8 [18.7 – 43.7]	0.003
Range	22.8 – 560	4.98 – 451	3.74 – 534.5	1.26 – 211.6	

Table [5]: Correlation between serum CXCL10 with VIDA and VASI score.

	Serum CXCL10 (ng/L)							
	All patients		**Segmental**		**Non segmental active**		**Non segmental stable**	
	r	**P-value**	**r**	**P-value**	**r**	**P-value**	**r**	**P-value**
VIDA score	**0.470****	**0.000**	-0.002	0.992	**0.842****	**0.000**	0.426	0.078
VASI score	**0.401****	**0.001**	-0.149	0.530	**0.578****	**0.005**	0.152	0.547

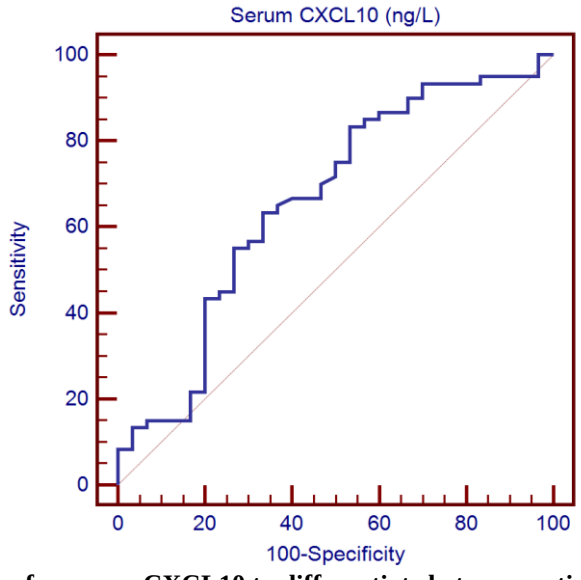

Figure [1]: ROC curve for serum CXCL10 to differentiate between patient and control groups.

© 2025 Journal of Carcinogenesis | Published for Carcinogenesis Press by Wolters Kluwer-Medknow

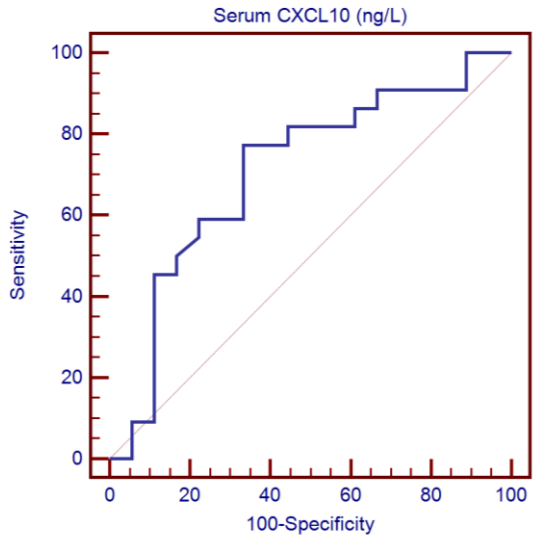

Figure [2]: ROC curve of CXCL10 to differentiate between non-segmental active vitiligo patients and non-segmental stable vitiligo patients.

5. DISCUSSION

A prior investigation indicated that both human vitiligo & a murine model of vitiligo exhibits an IFN-γ-specific Th1 immune response in the dermis, which includes the IFN-γ-dependent chemokines CXCL9, 10, and 11 **[11]**. Furthermore, prior research has emphasized the significance of CXCL10 in the immunopathogenesis of vitiligo **[11-12]**. Consequently, we have initiated this current investigation to assess serum CXCL10 as a biomarker for disease activity in pediatric vitiligo, aiming for improved therapy of this condition.

The present investigation involved 60 pediatric cases with vitiligo, contrasted with a control group of 30 subjects matched for age and sex. A blood specimen has been collected from every participant to assess serum CXCL10 concentration using the ELISA method. At the conclusion of the research, we detected a markedly significant elevation in serum CXCL-10 concentrations in cases [42.5] relative to the control group [25.8] [p= 0.005]. Additionally, we conducted ROC analysis and determined that the optimal cutoff for serum CXCL10 concentrations to distinguish between the case and control groups was above 25.9, with a sensitivity of 86.67, specificity of 53.33, and an AUC of 0.682. The findings indicate that serum CXCL10 serves as a valuable biomarker for predicting vitiligo.

To the best of our knowledge, the present research is the 1st to assess the serum level of CXCL10 in pediatric vitiligo and also the first to compare CXCL10 levels in segmental versus non-segmental vitiligo patients. Moreover, the results in the current study came in agreement with several previous studies of adult vitiligo patients indicating the pivotal role of CXCL10 in the pathogenesis of vitiligo.

Our finding comes in agreement with a case-control research conducted on 60 Egyptian vitiligo cases [thirty had active vitiligo & thirty had stable vitiligo] & thirty healthy age and sex matched controls to measure the blood concentrations of CXCL10 in vitiligo and to relate it with disease activity and degree. Vitiligo extent score [VES] has been calculated. CXCL10 has been measured in venous blood samples utilizing ELISA. The outcomes of their research shown that CXCL10 concentrations were significantly greater among vitiligo cases than controls [P below 0.001] **[12].**

This goes in line with previous study they conducted a case-control research on 30 Egyptian vitiligo cases, and 30 apparent healthy controls with similar demographics, to examine the possible role of CXCL10 [using ELISA method] in the pathogenesis of vitiligo. The outcome of the research revealed increased concentrations of CXCL10 in the vitiligo cases than controls **[13].**

These findings correspond to a case-control research involving 40 Egyptian cases diagnosed with non-segmental vitiligo [NSV; comprising 20 cases exhibiting active disease and 20 stable patients] alongside 20 healthy controls matched for age and sex, aimed at assessing the influence of CXCL10 and CXCL12 thru the ELISA technique in the etiology of vitiligo and examining their relation to disease activity. At the conclusion of the research, it has been determined that serum CXCL10 concentrations have been markedly elevated in individuals with vitiligo in comparison to the control group **[14]**. In alignment with prior findings, a research involving 80 vitiligo cases and 40 controls presents clinical evidence that supports and elucidates the role of CXCL10 in the recruitment of T-lymphocytes in vitiligo. Serum CXCL10 concentrations

© 2025 Journal of Carcinogenesis | Published for Carcinogenesis Press by Wolters Kluwer-Medknow

were markedly greater in cases with vitiligo than the control group. The findings of the present research, in conjunction with earlier studies, bolster the significance of CXCL10 in the pathogenesis of vitiligo when contrasted with healthy controls. **[15]**.

In the current research we found a high statistically significant variance among cases with segmental vitiligo, non-segmental stable vitiligo and non-segmental active vitiligo in serum CXCL10. Non segmental active vitiligo had the highest serum CXCL10 level [53.55] followed by segmental vitiligo [41.45] then non-segmental stable [32.3] [p= 0.003]. Moreover, we did ROC analysis and we found that the best cut off point for serum CXCL10 concentration to distinguish among non-segmental active and non-segmental stable vitiligo patient was >42 with sensitivity 77.27, specificity 66.67 and AUC 0.708. Our results suggest that increased serum level of CXCL10 had a positive relationship to active vitiligo, which can be utilized to monitor disease activity and predict vitiligo treatment response and outcome.

Our results corresponded with prior research indicating that CXCL10 levels in blister fluid samples have been markedly reduced in stable cases compared to those with active conditions. The researchers conducted ROC analysis and demonstrated that CXCL10 levels were both sensitive and specific for identifying active vitiligo **[15].**

Likewise, it was disclosed that serum concentrations of CXCL-10 were elevated in active vitiligo than stable vitiligo, and the expression of CXCL-10+ cells in tissue was more pronounced in active instances than in stable ones. To establish a threshold for the specificity and sensitivity of the activity indicators in their research, the authors conducted ROC analysis, revealing that CXCL-10 was the most specific serum indicator for distinguishing between active and stable cases, with a ninety-five percent confidence interval of 0.927 [0.823 - 0.980] [sensitivity 83.3%, specificity 96.0%]. These findings were consistent with ours **[8].**

Numerous studies corroborate that CXCL10 concentrations in vitiligo cases were markedly elevated in the active cohort relative to the stable cohort [P below 0.001] [13]. In a separate investigation, cases in the progressive phase exhibited elevated levels of CXCL10 compared to those in the stable phase, indicating a potential mechanistic function of CXCL10 in vitiligo **[14].**

This aligns with a cross-sectional research with 54 vitiligo cases , aimed at assessing the efficacy of systemic CXCL10 as an indicator of illness advancement and therapeutic response. The researchers exhibited increased CXCL10 concentrations in affected skin and in the bloodstream. Following intervention with psoralen with narrowband UVB [NB-UVB] phototherapy, UVA [PUVA] phototherapy, and systemic steroids [SS], a notable reduction in CXCL10 concentrations was seen, indicating that serum CXCL10 could serve as a biomarker for vitiligo **[16]**.

Contrary to our results, CXCL10 does not influence the activity of vitiligo. The researchers sought to evaluate serum CXCL10 concentrations in cases with active vitiligo versus those with stable vitiligo. A total of 35 individuals with non-segmental active vitiligo and thirty cases with non-segmental stable vitiligo have been enrolled. The research indicated that there was no notable relationship between serum CXCL10 levels and disease activity among the cases studied. It is noteworthy that the authors did not juxtapose serum CXCL10 levels with those of healthy control subjects. Variations in patient ethnicity, sample dimensions, disease progression, and measurement techniques may elucidate these conflicting findings **[17].**

This research revealed a statistically significant positive correlation among serum CXCL10 and both VIDA and VASI ratings in cases with non-segmental active vitiligo [p equal to 0.000 and 0.005, correspondingly]. These results indicate that elevated serum levels of CXCL10 correlate positively with disease severity and inform the management of progressing vitiligo.

Our findings indicate that the serum concentration of CXCL10 in Vitiligo cases exhibited a moderate positive correlation with the degree of the disease, as measured by the VASI and VIDA scores, implying the prospective value of serum CXCL10 as a new biomarker for assessing disease activity and a beneficial measure of treatment efficacy in progressive vitiligo **[14].**

These results closely resembled a descriptive cross-sectional investigation designed to assess serum CXCL 10 concentrations in 16 blood specimens from cases identified as having vitiligo. At the conclusion of the research, the authors indicated that serum CXCL 10 concentrations were elevated in individuals exhibiting active vitiligo lesions, and a strong positive connection was seen between VASI and CXCL 10. [P below 0.05**][16].** This aligns with the observation that blood CXCL10 concentrations correlated with elevated VASI ratings in cases with progressing vitiligo and diminished following effective therapy. These findings were consistent with ours **[15].**

Additionally, a statistically significant positive correlation has been seen among CXCL10 concentrations and the VES

© 2025 Journal of Carcinogenesis | Published for Carcinogenesis Press by Wolters Kluwer-Medknow

score [P below 0.003]. This outcome aligned with our findings; although, we utilized the VASI score instead of the VES score.

Based on prior research, the CXCL10 indicator could be clinically beneficial for personalized treatment strategies in pediatric vitiligo cases and for forecasting disease severity and activity in this demographic.

Moreover, serum and plasma concentrations of CXCL10 have been proposed as a prognostic indicator of disease activity in juvenile dermatomyositis, systemic lupus erythematosus, and type 1 diabetes **[18-19]**. Consistent with these findings, an earlier investigation also demonstrated that serum CXCL10 levels were markedly decreased during corticosteroid therapy in individuals with SLE and Graves' orbitopathy **[20]**. CXCL10 demonstrates a robust correlation with autoimmune conditions **[21]**.

In the present research, we did not see any noteworthy association between serum CXCL10 levels and case demographics [such as gender, age, skin phototype, and duration of vitiligo].

Numerous investigations similarly shown insignificant correlation among serum CXCL10 levels and case demographics. **[8, 13, 14, 15]**.

In conclusion, our research revealed that serum CXCL10 concentration is significantly increased in pediatric vitiligo cases especially in non-segmental active type. Serum CXCL10 level has significant positive association with both VIDA & VASI scores among non-segmental active vitiligo patients.

Funding: None.

Competing Interests: None.

Authorship: All authors have contributed significantly to this publication.

REFERENCES

[1] Khaitan BK and Sindhuja T. Autoimmunity in vitiligo: therapeutic implications and opportunities. Autoimmunity Reviews. 2022;21[1]:102932.

[2] Singh A, Gotherwal V, Junni P, Vijayan V, Tiwari M, Ganju P, et al. Mapping architectural and transcriptional alterations in non-lesional and lesional epidermis in vitiligo. Sci Rep. 2017;7[1]:9860.

[3] Nicolaidou E, Mastraftsi S, Tzanetakou V and Rigopoulos D. Childhood Vitiligo. American Journal of Clinical Dermatology. 2019;20:515–526.

[4] Jacquemin C, Rambert J, Guillet S, Thiolat D, Boukhedouni N, Doutre MS, et al. Heat shock protein 70 potentiates interferon alpha production by plasmacytoid dendritic cells: relevance for cutaneous lupus and vitiligo pathogenesis. British Journal of Dermatology. 2017;177[5]:1367-75.

[5] Wang XX, Wang QQ, Wu JQ, Jiang M, Chen L, Zhang CF, et al. Increased expression of CXCR3 and its ligands in patients with vitiligo and CXCL10 as a potential clinical marker for vitiligo. British Journal of Dermatology. 2016;174;1318–1326.

[6] Ferrari SM, Fallahi P, Santaguida G, Virili C, Ruffilli I, Ragusa F, et al. Circulating CXCL10 is increased in non-segmental vitiligo, in presence or absence of autoimmune thyroiditis. Autoimmunity reviews. 2017;16[9]:946-50.

[7] Abdallah M, El-Mofty M, Anbar T, Rasheed H, Esmat S, Al-Tawdy A, et al. CXCL-10 and Interleukin-6 are reliable serum markers for vitiligo activity: A multicenter cross-sectional study. Pigment Cell Melanoma Res. 2018;31[2]:330-336.

[8] Alrefai A, Naguib R, Rashed L and Ahmed M. Study of detection of serum CXCL 10 in vitiligo patients. 2022;3[1]:37-56.

[9] Richmond J, Strassner J, Rashighi M, Agrawal P, Grag M, Essien K, et al. Resident Memory and Recirculating Memory T Cells Cooperate to Maintain Disease in a Mouse Model of Vitiligo. 2019;139[4]:769-78.

[10] Rashighi M, Agarwal P, Richmond JM, Harris TH, Dresser K, Su MW, Zhou Y, Deng A, Hunter CA, Luster AD and Harris JE. CXCL10 is critical for the progression and maintenance of depigmentation in a mouse model of vitiligo. Science translational medicine. 2014;6[223]: 223ra23.

[11] Harris JE, Harris TH, Weninger W, et al. A mouse model of vitiligo with focused epidermal depigmentation requires IFN-gamma for autoreactive CD8[+] T-cell accumulation in the skin. J Invest Dermatol. 2012;132[7]:1869–1876.

[12] Khashaba S; Elmogy MH; Mohamed HF; El-Nablaway M; and Ismael AF. Assessment of Serum Levels of CXCL10 in Patients with Non-Segmental Vitiligo. Mansoura Medical Journal: 2023;52[2]: 102-107.

[13] El-Rifaie AA, Naguib RM, Rashed LA and Ahmed MF. Study Of Detection Of Serum CXCL10 In Vitiligo Patients. Egyptian Journal of Medical Research [EJMR], 2022;3[1]:37-56.

[14] Gharib K, Gadallah H, Elsayed A, Chemokines in Vitiligo Pathogenesis: CXCL10 and 12. J Clin Aesthet Dermatol. 2021;14[9]:27–32.

[15] Putri AI, Sawitri, Indramaya DM, Listiawan MY. Correlation of Vitiligo Area Scoring Index with the amount of CXCL 10 serum in vitiligo patient. Journal of Pakistan Association of Dermatologists. 2019; 29[3]: 286-288.

[16] El-Domyati M, El-Din WH, Rezk AF,Chervoneva I, Lee B, Farber M, Uitto J, Igoucheva O, Alexeev V. Systemic CXCL10 is a predictive biomarker of vitiligo lesional skin infiltration, PUVA, NB-UVB and corticosteroid treatment response and outcome. Archives of Dermatological Research. 2022;314:275–284

[17] AL-Mousawi ZR, Alattabi AS, Hamza DM, AL-Hussiny FA: Serum Level of CCL20 and CXCL 10 in Patients with Vitiligo and their Association with Disease Activity. Indian Journal of Forensic Medicine & Toxicology. 2021; 15: 1147–1156.

[18] Corrado A, Ferrari SM, Ferri C et al. Type 1 diabetes and [C-X-C motif] ligand [CXCL] 10 chemokine. Clin Ter. 2014; 165:e181–5.

[19] Ruffilli I, Ferrari SM, Colaci M et al. IP-10 in autoimmune thyroiditis. Horm Metab Res 2014; 46:597–602.

[20] Rose T, Gr€utzkau A, Hirseland H et al. IFNa and its response proteins, IP-10 and SIGLEC-1, are biomarkers of disease activity in systemic lupus erythematosus. Ann Rheum Dis 2013; 72:1639–45.

[21] Dominguez-Gutierrez PR, Ceribelli A, Satoh M, Sobel ES, Reeves WH and Chan EK [2014]: Reduced levels of CCL2 and CXCL10 in systemic lupus erythematosus patients under treatment with prednisone, mycophenolate mofetil, or hydroxychloroquine, except in a high STAT1 subset. *Arthritis Research & Therapy;* 16:1-5.

[22] Kameda M, Otsuka M, Chiba H, Kuronuma K, Hasegawa T, Takahashi H and Takahashi H [2020]: CXCL9, CXCL10, and CXCL11; biomarkers of pulmonary inflammation associated with autoimmunity in patients with collagen vascular diseases–associated interstitial lung disease and interstitial pneumonia with autoimmune features. *PLoS One;* 15: e0241719.

[23] Wang XX, Wang QQ, Wu JQ, Jiang M, Chen L, Zhang CF and Xiang LH Increased expression of CXCR3 and its ligands in patients with vitiligo and CXCL10 as a potential clinical marker for vitiligo. British Journal of Dermatology.2016; 174:1318–1326.