Leptin in Vitiligo: An Exploration of its Role in Disease Pathogenesis

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Abstract

Background: Vitiligo is an important depigmenting skin disease, in which there is selective loss of melanocytes, which in turn leads milky white skin lesions.

Objectives: To evaluate serum leptin levels in vitiligo patients with and without obesity, compared to obese non-vitiligo patients and healthy controls.

Patients and methods; in this case –control study, a total of 344 patients aged > 18 years who were eligible for inclusion in the current study. They were categorized into four groups (Group A: vitiligo patients with obesity, Group B: vitiligo patients without obesity, Group C: Obese patients without vitiligo and Group D: control participants without vitiligo or obesity).

Results: Serum leptin level was higher in obese patient. In the obese group (Group C), leptin levels showed significant positive correlations with age, anthropometric measures (weight, BMI, waist circumference), and a negative correlation with smoking. The regression analysis revealed strong independent association between serum leptin levels and obesity in vitiligo patients, there were strong positive correlation between serum leptin levels and disease duration, VASI score, and markers of obesity (BMI, waist circumference, central obesity index).

Conclusion: Elevated serum leptin level in vitiligo patient either (obese or non-obese) than obese non-vitiligo individuals and healthy controls. This elevation is significantly higher in obese vitiligo patients compared to others with strong association between leptin levels and markers of obesity, vitiligo duration, and severity suggests that leptin dysregulation may play a crucial role in the pathogenesis and progression of vitiligo, particularly in the context of obesity.

Keywords: Serum Leptin; Vitiligo; obesity.

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Introduction

Vitiligo is the commonest acquired depigmentary skin disorder manifested by circumscribed macules or patches of loss of pigment . It usually has devastating emotional and social consequences. **(Nguyen et al.,2016).**

Obesity is associated with proinflamatory state in which enlarged fat cells and adipose tissue-resident cells release an increased amount of proinflammatory cytokines. In obese individuals ,fat tissue cells secret large amount of leptin, a cytokine-like hormone that subsequently stimulates autocrine secretion of variable proinflammatory cytokines, tumor necrosis factor-α and interleukin-6 which create a state of chronic inflammation that increases the risk of autoimmune diseases (**Fidan et al.,2016).** In vitiligo, tissue biopsy revealed a significant high expression of proinflammatory cytokines in lesional and perilesional tissue. These cytokines play an essential role in melanocyte damage and loss. Contradictory findings have been observed at a systemic level (**Camara-Lemarroy and Salas-Alanis, 2013).** Elevated level of serum interleukin-17A, interleukin-21 levels and transforming growth factor-β1 have been identified at the serum in patients with vitiligo **(Zhou et al.,2015).**

Leptin decreases expansion of regulatory T cells and suppress its function and so amplifies the processes of inflammation through the attraction and accumulation of macrophages , mast cells and CD8+ Th1 lymphocytes and stimulates secretion of multiple inflammatory mediators (**Procaccini et al.,2011)**.

This chronic low-grade inflammation can explain the relationship between a obesity and the presence of inflammation in vitiligo lesions (**Dragoni et al.,2017).**

The aim of this work was to evaluate serum leptin levels in vitiligo patients with and without obesity, compared to obese non-vitiligo patients and healthy controls, and assessing its correlation to the body mass index and disease dermographic data.

Patients and methods

This case-control study was approved by the Research and Ethical Committee of the Faculty of Medicine, Sohag University (IRB Registration No: soh-med-22-09-02). An informed written consent was obtained from all cases.

This study included 172 vitiligo patients attending the Dermatology, Venereology, and Andrology outpatient clinic and other 172 non vitiligo controls. Inclusion criteria were All patients with vitiligo who are above the age of 18 years. While Exclusion criteria were Patients with other autoimmune diseases, pregnant or lactating patients.

Participants were categorized into four groups: Group A: 86 vitiligo patients with obesity; Group B: 86 vitiligo patients without obesity; Group C: 86 obese individuals without vitiligo; Group D: 86 healthy controls.

All participants in this study were subjected to clinical evaluation through: Complete history taking in the form of Personal history; included age, sex, marital status, occupation and special habit of medical importance. History of present illness; included onset of disease, its course, duration, previous treatment received. and Family history of vitiligo

General examination Was made to detect systemic diseases and assessment of body Weight in (kg), Height in (cm) and calculation of Body mass index" BMI” . Also Waist

circumference "WC" (cm) was measured with a tape at the midpoint between the iliac crest and the subcostal plane of the abdomen in a standing position. Also Index of central obesity "ICO" was calculated; it equals to the ratio of WC to height. A cutoff of 0.5 was set for the ICO. **(Parikh et al., 2007)**.Obesity was diagnosed based on BMI ≥30 and an index of central obesity (ICO) >0.5.

Dermatological examination involved local Examination of vitiligo patients to detect Type of lesions and Distribution and calculation of VASI score for disease evaluation. Vitiligo Area Severity Index (VASI): The area of vitiligo lesions is measured in terms of hand units. each hand unit is equal to 1% of the total body surface area. The level of pigmentation is estimated to the nearest one of the following percentages: 100% - complete depigmentation ; 90% - specks of pigment present; 75% - depigmented area exceeds the pigmented area; 50% - pigmented and depigmented areas are equal; 25% - pigmented area exceeds depigmented area; and 10% - only specks of depigmentation present **(Hamzavi et al.,2004).** The VASI for each body region is determined by the product of multiplication of the area of vitiligo in hand units and the extent of depigmentation within each hand unit measured patch. Total body VASI = S All body sites.

Investigations

All patients were subjected to blood samples and biochemical assay:

A) Blood samples: Three millilitres venous blood were withdrawn and evacuated into serum gel separator tubes, samples allowed to be clotted for 30 min. at 37ºC, then centrifugation at 3500 rpm for 5 min was done. Then the sera were separated into 1 ml cryotubes, and stored at - 80ºC till biochemical assays of leptin

B) Biochemical assays of serum leptin: using commercially available ELISA assay kits supplied by Sunred Biological Technology Co., Ltd., Shanghai, China, catalogue number 201-12-1560, using microplate ELISA reader (EMR-500, USA).

Statistical analysis

It was performed using IBM-SPSS ver. 24, categorical variables were presented as frequency and percentages, Chi-square test was used for comparison of proportions between groups . Quantitative variables were presented as mean, and standard deviation (SD). Test of normality, Shapiro-Wilk was used to test the normality of continuous variables and independent sample t-test was calculated to test the mean differences between groups. For continuous variables with more than two categories ANOVA test was calculated to test the mean differences, post-hoc test was calculated using Bonferroni corrections for pairwise comparisons between the two study groups. to investigate the independent relationship between s. leptin level and obesity among vitiligo patients multivariable logistic regression analysis was calculated (Odds Ratio -OR-, 95% confidence interval -95% CI- and p-value-). Pearson correlation was calculated to examine the relationship between s. leptin and disease determinants. A p-value <0.05 was considered significant.

Results

As regard vitiligo clinical characteristics in both group A, B, both groups showed sudden onset of the disease, it was with progressive course in 72.1% of group (A) and 87.2% of group (B) with insignificant difference. Vitiligo duration

was significantly longer in Group A than in Group B, while VASI scores were insignificantly higher in Group B than in Group A. Positive family history was in 38.4%, 48.8% of group (A) and (B) respectively. Majority of vitiligo patients were on topical and phototherapy treatment. All the clinical criteria of vitiligo in vitiligo groups are shown in (**Table.1**).

Table 1. Clinical characteristics of the studied vitiligo groups

Variables	Group-A (n = 86)	Group-B (n = 86)	P-value
Sudden Onset	86 (100%)	86 (100%)	----
Disease course			
Progressive	62 (72.1%)	75 (87.2%)	**0.014****
Stationary	24 (27.9%)	11 (12.8%)	
Disease Duration/years	9.01 ± 1.6	5.62 ± 1.4	**< 0.001***
VASI Score	4.03 ± 1.4	5.53 ± 1.8	0.094*
Family History			
Negative	53 (61.6%)	44 (51.2%)	0.166**
Positive	33 (38.4%)	42 (48.8%)	
Treatment Type			
Topical + Phototherapy	53 (61.6%)	46 (47.5%)	0.735**
Topical	28 (32.6%)	31 (36%)	
Laser	5(5.8%)	9(10.5%)	

Group A: vitiligo patients with obesity and Group B: vitiligo patients without obesity; VASI: Vitiligo area scoring index. *Independent Sample T-test was used to compare the difference in mean between groups. **Chi-square test was used to compare frequency between group

There was significant ($p < 0.001$) difference in the mean level of serum leptin level between groups; Serum leptin levels were significantly higher in Group A (7.71±1.3) compared to Groups B (5.5±1.1), C (5.18±0.7), and D (3.09±0.9) ($p < 0.001$). Also group (B) or group (C) showed significant higher serum leptin level (5.50 ±1.1, 5.18± 0.7 respectively) than control group (3.09±0.9) (**Table.2,Fig. 1**).

Table 2. Serum leptin level difference between the studied groups

Variables	Group A (n=86)	Group B (n=86)	Group C (n=86)	Group D (n=86)	P-value
Serum leptin (ng/ml)	7.71 ± 1.3	5.50 ± 1.1	5.18 ± 0.7	3.09 ± 0.9	**< 0.001***
P-value**	A vs B<0.001	B vs C=0.224	C vs D<0.001	A vs D<0.001	
	A vs C<0.001	B vs D<0.001			

Group A: vitiligo patients with obesity, Group B: vitiligo patients without obesity, Group C: Obese patients without vitiligo and Group D: control participants without vitiligo or obesity. *ANOVA test was used to compare the mean difference between groups ; **Post-hoc test with Bonferroni Corrections was used to compare the mean difference between groups; ***Chi-square test was used to compare frequency between groups

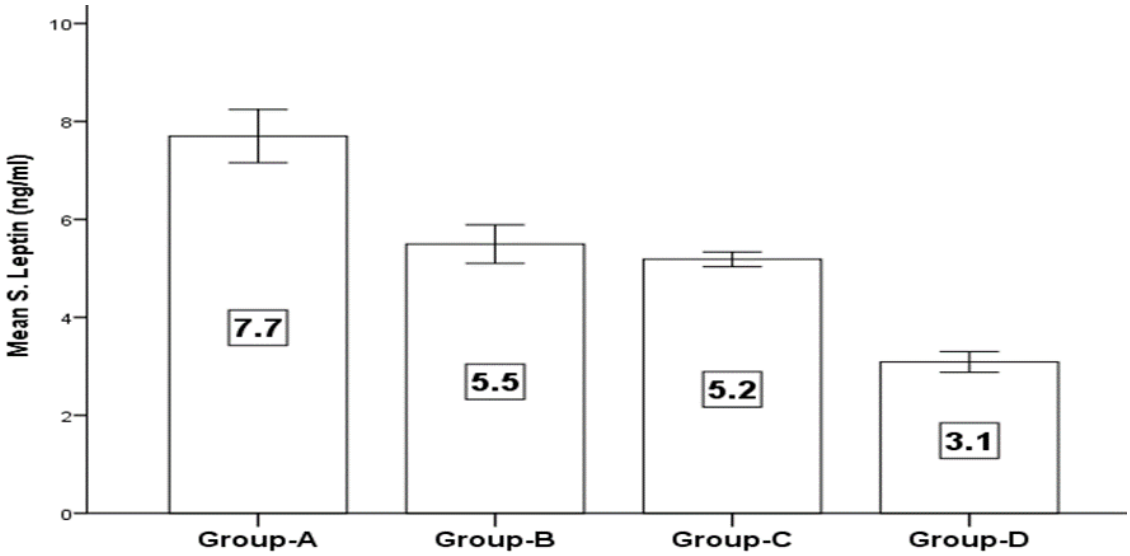

Fig. 1. Mean level of serum leptin of the studied groups

Multivariable logistic regression model of the independent association between obesity and S. Leptin level among vitiligo patients revealed that each year increase in the patient's age produces 2.5% (OR = 1.025, 95% CI; 1.001 – 1.048) increase in the chance of being obese and this was statistically significant (p = 0.038). Moreover, married patients had 3 folds increased risk (OR = 2.970, 95% CI; 1.043 – 8.463, p = 0.042) of being obese than single cases. Likewise, the probability of being obese was 3.2 times (OR = 3.185, 95% CI; 1.198 –8.468, p = 0.020) in smokers compared with non-smokers. Likely, patients with skilled work were less liable for being obese by 32% (OR = 0.678, 95% CI; 0.467 – 0.985, p = 0.041). Furthermore, the possibility of obesity was three times more among patients with progressive disease course (OR = 2.913, 95% CI; 1.163 – 7.309, p = 0.023) than those with stationary course. Also, with one-year increase in the disease duration, there was 19% (OR = 1.191, 95% CI; 1.089 – 1.302) increase in the obesity likelihood and this was statistically insignificant (p < 0.001). Similarly, with one-point increase in the VASI score there was 29% (OR = 1.291, 95% CI; 1.034 – 2.970, p = 0.006) increase in the obesity likelihood. Notably, with one-ng/ml increase in the s. leptin level there was 63% (OR = 1.626, 95% CI; 1.357 – 1.947, p < 0.001) increase in the obesity likelihood, (**Table.3**)

Table 3. Independent association between obesity and S. Leptin level among vitiligo patients (group A and B): multivariable logistic regression model

Variables	OR (95% CI) *	P-value
Age/years	1.025 (1.001 – 1.048)	**= 0.038**
Sex (Male)	0.735 (0.390 – 1.348)	= 0.341
Marital Status (Married)	2.970 (1.043 – 8.463)	**= 0.042**
Skilled Worker	0.678 (0.467 – 0.985)	**= 0.041**
Disease Course (Progressive)	2.913 (1.163 – 7.301)	**= 0.023**
Disease Duration	1.191 (1.089 – 1.302)	**< 0.001**
VASI	1.291 (1.034 – 2.970)	**= 0.006**

Serum Leptin Level	1.626 (1.357 – 1.947)	**< 0.001**

OR=Odds Ratio; CI, Confidence Interval; VASI: Vitiligo area scoring index

The univariate correlation between s. leptin and disease determinants among vitiligo cases showed significant (< 0.001) positive moderate to high correlation between s. leptin and age, smoking, weight, BMI, waist circumference, index of central obesity, disease duration and VASI score (r = 0.572, 0.283, 0.410, 0.451, 0.423, 0.449, 0.939 and 0.402). In other words, increase in s. leptin level was associated with increase in the values of these parameters. Likewise, significant negative mild and moderate correlation was observed between s. leptin and height (r=-0.163, p = 0.017) i.e., increase in s. leptin level was associated with decrease in height,(**Table. 4; Fig. 2**).

Table 4. Univariate Correlation of S. Leptin and Disease Determinants in Vitiligo patients (group A&B)

Variables (n = 172)	S. Leptin (ng/ml)
	r* (P-value)
Age	**0.572 (< 0.001)**
Weight	**0.410 (< 0.001)**
Height	**-0.163 (= 0.017)**
Waist Circumference	**0.423 (< 0.001)**
Index of Central Obesity	**0.449 (< 0.001)**
Body mass index	**0.451 (< 0.001)**
Disease Duration	**0.939 (< 0.001)**
VASI Score	**0.402 (< 0.001)**

*Pearson Correlation Coefficient; VASI: Vitiligo area scoring index

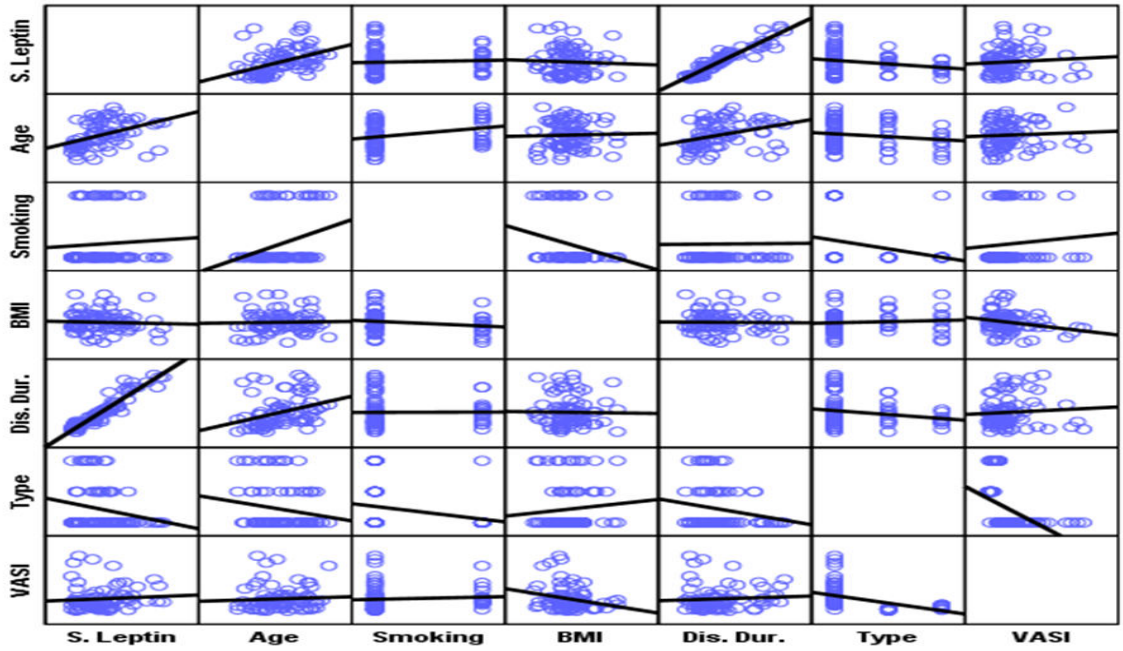

Fig. 2. Univariate correlation of S. Leptin and disease determinants.
BMI: body mass index; Dis. Dur: disease duration; VASI: Vitiligo area scoring index.

The univariate correlation between s. leptin and disease determinants among Obese group (Group-C) revealed significant (< 0.001) positive mild to high correlation between s. leptin and age, weight, WC, ICO and BMI (r = 0.195, 0.365, 0.483, 0.393 and 0.417). In other words, increase in s. leptin level was associated with increase in the values of these parameters. On the other hand, significant negative moderate and mild correlation was observed between s. leptin and smoking (r = -0.190) smoking was associated with decrease in the level of s. leptin, (**Table.5; Fig.3**).

Table 5. Univariate Correlation of S. Leptin and Disease Determinants in Obese patients (group C)

Variables (n=86)	S. Leptin (ng/ml)
	r* (P-value)
Age	**0.195 (= 0.036)**
Weight	**0.365 (< 0.001)**
WC (waist circumference)	**0.483 (< 0.001)**
ICO (index of central obesity)	**0.393 (< 0.001)**
BMI (body mass index)	**0.417 (< 0.001)**

*Pearson Correlation Coefficient

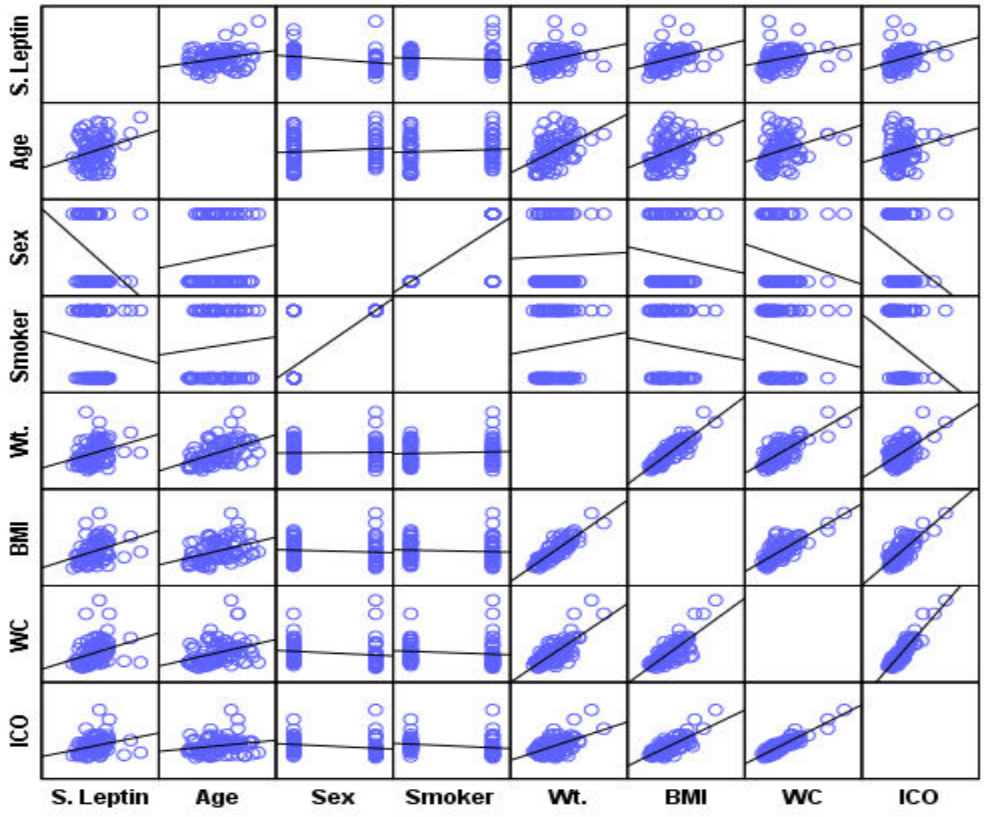

Fig. 3 . Univariate Correlation of S. Leptin and disease determinants in obese groups. Wt: weight; BMI: body mass index; WC: waist circumference; ICO : index of central obesity

Discussion

Our findings of higher serum leptin levels in obese individuals align with previous studies by **Al-Sultan and Al-Elq (2006)** who measured serum leptin level in obese individuals and normal weight persons, and assess the relation between its level and anthropometric parameters and metabolic indices. They concluded that there is a significantly higher serum leptin in obese patients compared to non-obese subjects. In harmony, **Al Maskari and Alnaqdy (2006)** who aimed to verify the link between serum leptin levels and body measures as weight, Body Mass Index (BMI) and fat percentage among Omani obese and non-obese persons. They found a significant increased serum leptin in obese patients than the control non-obese subjects. This also agrees with **Liu et al. (2020)** who reported that, the median leptin level was elevated in the obese group than in the normal BMI group.

In the obese group (Group C), leptin levels showed significant positive correlations with age, anthropometric measures (weight, BMI, waist circumference), and a negative correlation with smoking.

In accordance, **Al-Sultan and Al-Elq (2006)** reported that there is a significant positive correlation between serum leptin and BMI (r 0.440) and hip circumference (r 0.425) and **Erturk et al. (2004)** who reported that, serum leptin levels significantly correlated with body mass index (r = 0.649). **Al Maskari and Alnaqdy (2006)** reported that in obese patients, serum leptin level correlates positively with their weight (p=0.002), body fat percentage (p=0.0001) and BMI (p=0.001).

In harmony with what was ascertained by **Li et al.(2011)** there is association between elevated serum leptin metabolic risks, as well as MetS.

The key finding of our study was the significantly higher serum leptin levels in obese vitiligo patients (Group A) compared to all other groups, including obese non-vitiligo individuals (Group C). This suggests that vitiligo may be associated with altered leptin regulation or sensitivity, potentially contributing to the development or progression of the disease. Interestingly, both non-obese vitiligo patients (Group B) and obese non-vitiligo individuals (Group C) had nearly similar leptin level that is higher than healthy controls, indicating that either vitiligo or obesity alone can lead to increased leptin levels. The combination of vitiligo and obesity (Group A) appears to have an additive or synergistic effect on leptin dysregulation.

Previous studies have reported conflicting results regarding leptin levels in vitiligo mice they revealed that serum leptin is significantly decreased in vitiligo mice compared to normal healthy mice. **(Wu et al., 2023)** this can be due to difference between human and mice results. Also, in those mice vitiligo is induced by topical monobenzone. Not due to inflammatory sequence of pathogenesis.

In agreement with our study, **El-Hawary et al. (2022)** reported that the level serum leptin was significantly higher in vitiligo patients than controls.

Dragoni et al.(2017) have postulated that leptin is one of interleukin-6 family and elevated serum leptin levels plays a role in the development of a state of low grade chronic inflammation in some vitiligo patients with high BMI.

IL-6 was postulated as a sensitive marker of activity in vitiligo patients and as a marker of inflammation related

to the pathogenesis of development of type I diabetes mellitus in association with vitiligo. Considering leptin as one of IL-6 family, this could explain a possible relation between elevated serum leptin and pathogenesis of development of vitiligo as well as MTS (**Abdallah et al.,2018; Farhan et al.,2014).**

Adipose tissue contains melanocytes and there is a cross-talk between melanocytes and adipocytes through wnt/ beta-catenin pathway, this should be taken in consideration when deal with serum leptin in vitiligo patients. The process of melanogenesis within the adipose tissue contributes to oxidative radicals scavenging as well as suppress adipocyte hyperplasia and decreases obesity-related complications (**Abdallah et al.,2018; Farhan et al.,2014).**

Alteration in serum leptin level has also been found in obese patients and those with metabolic disorders , but not everyone with elevated serum leptin develops vitiligo **(Ramos and Donato, 2017).**

In our study, the regression analysis further confirmed the strong independent association between serum leptin levels and obesity in vitiligo patients, even after adjusting for potential confounders such as age, smoking, disease characteristics, and BMI. This finding supports the hypothesis that leptin may has an important role in the pathogenesis of vitiligo development, particularly in association with obesity (**Wu et al.,2023**).

In our study, the strong positive correlation between serum leptin levels and disease duration, VASI score, and markers of obesity (BMI, waist circumference, central obesity index) reinforces the potential link between leptin dysregulation and both vitiligo severity and obesity-related metabolic disturbances. These findings are consistent with the known pro-inflammatory and immunomodulatory effects of leptin, which could contribute to the autoimmune processes underlying vitiligo and the metabolic complications associated with obesity (**La Cava, 2017).**

To our knowledge, this is the first study to assess serum leptin levels in obese vitiligo patients, highlighting its potential role in disease pathogenesis.

We found it higher serum leptin in patient with vitiligo alone and patient with obesity alone. Thus, serum leptin may serve as a marker for metabolic susceptibility in vitiligo patients.

In accordance, to finding of our study **El-Hawary et al. (2022)** suggested that higher leptin levels, a hormone linked to fat cells, might be a consequence of having vitiligo rather than a cause. Leptin levels increased with age, age at vitiligo onset, and central obesity, but in disagreement to our finding that serum leptin level is positive correlation to VASI score and this not with disease severity.

Conclusion

Elevated serum leptin level in vitiligo patient either (obese or non-obese) than obese non-vitiligo individuals and healthy controls**.** This elevation is significantly higher in obese vitiligo patients compared to others with strong association between leptin levels and markers of obesity, vitiligo duration, and severity suggests that leptin dysregulation may play a crucial role in the pathogenesis and progression of vitiligo, particularly in the context of obesity. These findings highlight the importance of considering the interplay in pathogenesis between vitiligo,

metabolic disturbances, and adipokine dysregulation and the development of potential therapeutic strategies.

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