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Evaluation of oxidative stress burden in vitiligo patients: A case-control study from the North Indian Cohort

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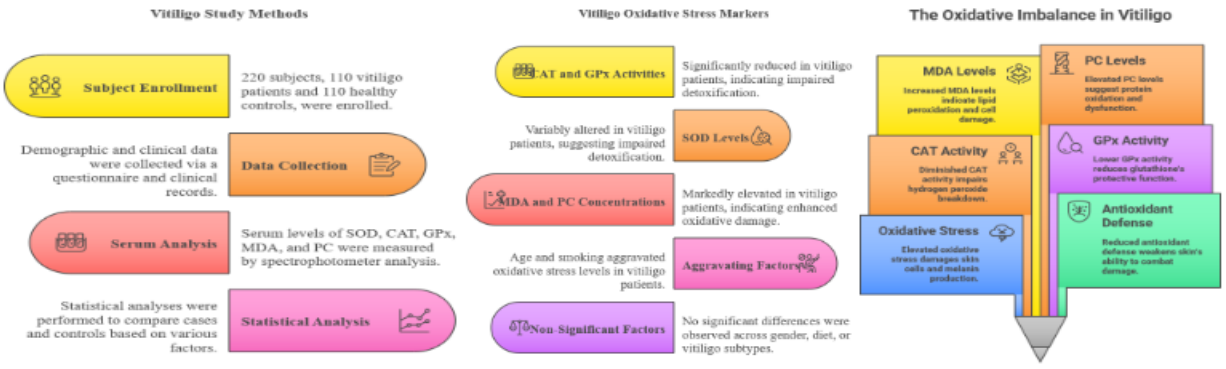
Abstract

Background: Vitiligo is an acquired disorder characterized by skin depigmentation. Although autoimmune mechanisms are central to its pathogenesis, accumulating evidence highlights that oxidative stress is a pivotal contributor to melanocyte destruction.
Methods: A total of 220 subjects (110 vitiligo patients and 110 healthy controls) were enrolled. Demographic and clinical data were collected via a well-designed questionnaire and clinical records. The serum levels of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), malondialdehyde (MDA), and protein carbonyl (PC) were measured by spectrophotometer analysis. Statistical analyses were performed to compare cases and controls based on age, gender, diet, alcohol/smoking habits, and clinical subtype/activity of vitiligo.
Results: Vitiligo patients showed significantly reduced CAT and GPx activities compared to controls, while SOD levels were variably altered, suggesting impaired hydrogen peroxide (H_2O_2) detoxification. The MDA and PC concentrations were markedly elevated in patients, indicating enhanced lipid and protein oxidative damage. Age and smoking aggravated oxidative stress levels in vitiligo patients, while no significant differences were observed across gender, diet, or vitiligo subtypes.
Conclusion: An imbalance between elevated oxidative stress and impaired antioxidant defense, particularly diminished CAT and GPx activities, while elevated MDA and PC levels, underscores the pathogenic role of oxidative damage in vitiligo. These biomarkers may serve as useful indicators of disease progression and targets for antioxidant-based therapeutic strategies.

Graphical abstract



1.INTRODUCTION

Over the past few decades, there has been a marked increase in the incidence of autoimmune diseases, and vitiligo is recognized as one of the most frequently occurring autoimmune pigmentary disorders. Vitiligo is characterized by the progressive loss of functional melanocytes. While autoimmune processes are widely recognized as central to its pathogenesis, increasing attention has been directed toward oxidative stress as a critical initiator and potentiator of melanocyte damage (Picardo et al., 2015; Speeckaert et al.,2017). Oxidative stress describes the disequilibrium between reactive oxygen species (ROS) formation and antioxidant defense mechanisms, culminating in oxidative damage to essential cellular components. In individuals with vitiligo, numerous studies have documented elevated ROS levels, particularly superoxide anions (O_2^-) and hydrogen peroxide (H_2O_2) in both lesional skin and systemic circulation (Zhou et al.,2019). Excessive accumulation of these reactive species disrupts vital cellular processes, promotes melanocyte apoptosis, and facilitates the release of immunogenic antigens, thereby triggering or exacerbating autoimmune responses. Enzymatic antioxidant systems play a primary role in controlling ROS. Superoxide dismutase (SOD) catalyzes the conversion of superoxide radicals into H_2O_2, which is subsequently decomposed into water by glutathione peroxidase (GPx) and catalase (CAT), thereby limiting oxidative damage. Alterations in the activities of these enzymes have been consistently observed in vitiligo patients. Specifically, increased SOD activity in conjunction with decreased CAT and GPx levels impairs effective detoxification of H_2O_2, resulting in cumulative oxidative stress (Gianfaldoni et al., 2018; Shaikh et al., 2021). The resulting hydrogen peroxide overload inhibits tyrosinase, the key enzyme in melanogenesis, and adversely affects mitochondrial function, thereby accelerating melanocyte degeneration.

In addition to enzymatic defenses, non-enzymatic oxidative stress biomarkers provide valuable insight into molecular damage. Malondialdehyde (MDA), a lipid peroxidation end-product, is significantly elevated in the serum and skin of vitiligo patients and shows a strong association with disease activity and severity (Karadag et al., 2020). Similarly, protein carbonyls (PC), formed through irreversible oxidative modification of proteins, impair protein structure and biological function, contributing further to melanocyte dysfunction and apoptosis (Yang et al., 2021). Emerging transcriptomic and proteomic studies have reinforced the oxidative stress paradigm in vitiligo by demonstrating downregulation of genes encoding antioxidant enzymes and upregulation of oxidative damage markers in lesional tissues (Cui and Liu, 2023). The present study focuses on evaluating both enzymatic and non-enzymatic oxidative stress markers in vitiligo. Impairment of this antioxidant defense predisposes melanocytes to degeneration under oxidative conditions. Therefore, supporting the skin's antioxidant systems through coordinated enzymatic and non-enzymatic mechanisms may be essential for protecting melanocytes from oxidative damage and limiting the progression of vitiligo.

2. METHODOLOGY

Subject's recruitment

In our study, a total of 220 participants were enrolled and categorized into two groups: 110 vitiligo patients clinically diagnosed at the Department of Dermatology, PGIMS, Rohtak, Haryana, and 110 healthy subjects without any personal or familial history of vitiligo. Participants aged 18 years and above, regardless of gender, were recruited for the study. Those with a history of infectious, immunological, or autoimmune conditions, along with pregnant or breastfeeding women, were excluded. Information regarding participants' age, gender, vitiligo type and duration, family history, and the extent of skin involvement was gathered through a structured questionnaire and verified using clinical documentation.

Sample collection

From vitiligo patients and controls, 2 mL of venous blood samples were drawn via venipuncture and collected in vacuum-filled vacutainer tubes. The coagulated blood was allowed to stand for 30 minutes before centrifugation at 3000 rpm for 10-15 minutes to separate the serum, subsequently followed by biochemical analysis.

Analysis of Oxidative Stress Biomarkers

Superoxide dismutase (SOD) activity

The enzymatic activity of superoxide dismutase (SOD) in serum samples was evaluated following the method described by Marklund and Marklund (1974) with minor modifications (Marklund and Marklund, 1974). The assay is based on the ability of SOD to inhibit the autoxidation of pyrogallol, which generates superoxide radicals under alkaline conditions. The reaction mixture consisted of 100 µL of serum, 2.8 mL of 0.1 M Tris–HCl buffer (pH 8.2), and 0.1 mL of 2 mM pyrogallol as the substrate. Pyrogallol oxidation was monitored by recording the change in absorbance at 420 nm using a UV-visible spectrophotometer.

Catalase (CAT) Activity

Catalase (CAT) activity was determined using the method described by Aebi (1984) with minor modifications (Aebi, 1984). The assay is based on the enzymatic decomposition of hydrogen peroxide (H_2O_2), which results in a decrease in absorbance at 240

nm. Absorbance measurements were recorded at 10 s intervals for 3 min using a UV-visible spectrophotometer. A freshly prepared H_2O_2 solution was obtained by diluting 0.06 mL of 30% H_2O_2 in 50 mL of phosphate buffer (pH 7.0). For each reaction, 0.98 mL of the working H_2O_2 solution was used. The baseline absorbance of the H_2O_2 solution was adjusted to 0.50 ± 0.01 at 240 nm, with phosphate buffer serving as the reagent blank. The decrease in absorbance at 240 nm corresponds to the enzyme-mediated decomposition of H_2O_2. Catalase activity was calculated based on the principle that a decline of 0.04 absorbance units per minute corresponds to the breakdown of 1 µmol of hydrogen peroxide per minute.

Glutathione Peroxidase (GPx) Activity

Glutathione peroxidase (GPx) activity in serum was determined spectrophotometrically using the method described by Sharma et al. (2020) (Sharma et al., 2020). The assay is based on a glutathione reductase–coupled reaction, wherein oxidized glutathione (GSSG) is reduced to reduced glutathione (GSH) in the presence of NADPH. The regenerated GSH reacts with 5,5′-dithiobis(2-nitrobenzoic acid) (DTNB) to form a yellow-colored 5-thionitrobenzoic acid (TNB) chromophore, measurable at 412 nm. The reaction mixture consisted of 0.4 M phosphate buffer (pH 7.0), 10 mM sodium azide, and 2 mM GSH, which was pre-incubated for 5 min. The reaction was initiated by adding 2.4 mM H_2O_2 and subsequently incubated at 37°C for 10 min. The reaction was terminated with 10% trichloroacetic acid (TCA), followed by centrifugation at 3500 rpm for 15 min. The resulting supernatant was reacted with DTNB, and the absorbance of TNB was recorded at 412 nm using a UV-visible spectrophotometer.

Malondialdehyde (MDA) Estimation

Serum MDA concentration was determined spectrophotometrically according to the method described by Vineetha and Palakkal (2020) with minor modifications (Vineetha and Palakkal, 2020). The assay employed trichloroacetic acid (TCA) and thiobarbituric acid (TBA) reagents for the assessment of lipid peroxidation. Following the reaction, the samples were centrifuged at 8000 rpm for 30 s, and the absorbance of the resulting supernatant was recorded at 530 nm using a UV-visible spectrophotometer.

Protein Carbonyl (PC) Estimation

Serum PC content was estimated following the method of Levine et al. (1990) with minor modifications (Levine et al., 1990). Briefly, samples were treated with hydrochloric acid (HCl), and proteins were precipitated using trichloroacetic acid (TCA). The carbonyl groups were quantified by measuring the absorbance of the supernatant within the wavelength range of 360-370 nm using a UV-visible spectrophotometer.

Statistical Analysis

Statistical analysis was performed using SPSS software (version 26). Normality of continuous variables was assessed using the Kolmogorov-Smirnov test. Continuous demographic variables, such as age, were expressed as mean ± standard deviation using Student's *t*-test. Categorical variables were analyzed using the Chi-square test. As the biochemical data were not normally distributed, results are expressed as median and interquartile range (IQR), and non-parametric tests were applied. The Mann-Whitney U test was used to compare oxidative stress biomarkers and antioxidant enzyme activities between vitiligo patients and healthy controls, as well as between gender groups. Differences across age categories and vitiligo types were evaluated using the Kruskal-Wallis test. A p-value less than 0.05 was considered statistically significant.

3. RESULTS

Table 1: Demographic and clinical characteristics of controls and vitiligo patients

Variables	vitiligo patients (N=110)	Control (N=110)	p-Value
Age in years (mean ± SD)	34.49 ± 12.8	30.60 ± 10.2	0.028[b*]
Age group			
18-30	50	74	0.004[c*]
31-45	42	23	
46-60	18	13	
Gender (N/%)			
Male	45 (40.9)	69 (62.73)	0.003 [c*]
Female	65 (59.0)	41 (37.27)	
Smoking (N/%)			
Yes	13 (11.81)	5 (4.54)	0.118 [c*]
No	97 (88.18)	105 (95.45)	
Diet			
Vegetarian	94	92	0.580 [c*]
Non Vegetarian	16	18	
Types of vitiligo			
Segmental	4	-	NA
Non segmental	93	-	
Intermediate (segmental+non-segmental)	13	-	

Continuous variables (age) are presented as mean ± standard deviation (SD) and were analyzed using the independent Student's t-test. Categorical variables were compared using the Chi-square (x^2) test.

NA = not applicable, b = student t-test, c = Chi-square test, *p<0.05 is statistically significant.

The demographic and clinical characteristics of vitiligo patients and controls are summarized in Table 1. The mean age of vitiligo patients (34.49 ± 12.8 years) was significantly higher than that of controls (30.60 ± 10.2 years; p = 0.028). Distribution across age categories showed that the majority of controls (67.3%) were in the 18-30 year group, whereas most vitiligo patients (38.2%) were in the 31-45 year category, showing a significant age-related difference (p = 0.004). Gender distribution revealed a higher proportion of females (59.1%) among vitiligo patients compared to controls (37.3%), with males being more represented in the control group (62.7%; p = 0.003). Smoking status and dietary habits did not differ significantly between patients and controls (p = 1.000 and p = 0.580, respectively). Among the clinical types of vitiligo, the non-segmental form was predominant (84.5%), followed by intermediate type (11.8%) and segmental (3.6%) types (p < 0.001).

Table 2. Antioxidant enzyme activity levels in vitiligo patients and controls according to demographic characteristics

Variable	Group (control =110) (vitiligo =110)	SOD (U/ml) Median(IQR)	CAT (µmol H_2O_2/min) Median(IQR)	GPx (U/ml) Median(IQR)
18-30	Control	3.80(2.63-5.02)	4.77(4.31-5.24)	33.22(22.49-39.93)
	vitiligo	1.76(1.18-2.36)	1.91(1.29-2.51)	18.16(11.01-24.35)
31-45	Control	3.52(3.18-4.84)	5.02(4.25-5.29)	31.42(22.70-42.83)
	vitiligo	1.80(1.53-2.32)	2.30(1.79-2.44)	18.76(13.10-24.91)
45-65	Control	4.88(3.21-5.68)	3.65(3.51-4.37)	24.11(20.49-33.43)
	vitiligo	1.67(1.33-2.44)	1.82(1.15-2.65)	19.81(15.83-26.84)
Male	Control	3.66(2.81-5.21)	4.85(4.19-5.24)	29.25(20.61-37.60)
	vitiligo	1.89(1.18-2.45)	2.16(1.35-2.44)	17.52(12.05-24.91)
Female	Control	4.14(2.59-5.02)	4.66(4.24-5.26)	34.55(27.64-42.91)
	vitiligo	1.70(1.40-2.36)	1.99(1.50-2.52)	19.04(12.70-25.87)
Segmental	Control	NA	NA	NA
	vitiligo	2.36(1.26-2.45)	2.42(1.29-2.77)	16.23(12.42-27.84)
Non-segmental	Control	NA	NA	NA
	vitiligo	1.79(1.34-2.38)	2.16(1.40-2.52)	18.72(12.37-25.75)
Intermediate (segmental+non-segmental)	Control	NA	NA	NA
	vitiligo	1.65(1.52-2.33)	1.86(1.55-2.28)	19.20(12.23-22.18)

Values of SOD, CAT, and GPx are presented as median IQR. Comparisons between vitiligo patients and healthy controls were performed using the Mann-Whitney U test. A p-value < 0.05 was considered statistically significant.

SOD- superoxide dismutase, CAT- catalase, GPx- glutathione peroxidase, IQR- interquartile range, NA- not applicable

Antioxidant enzymatic activities, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), were compared between vitiligo patients and healthy controls across age groups, genders, and vitiligo subtypes (Table 2). The patients showed lower mean ± SD values than the controls. Vitiligo patients consistently exhibited lower median activities of these enzymes compared to controls in all age categories (18-30, 31-45, and 45-65 years). For instance, in the 18-30 years group, median SOD activity was 1.76 U/ml (IQR: 1.18-2.36) in patients versus 3.80 U/ml (IQR: 2.63-5.02) in controls, with similar reductions noted for CAT and GPx activities. Gender-wise analysis also showed decreased antioxidant activities in both male and female patients relative to their controls; males with vitiligo had a median SOD activity of 1.89 U/ml (IQR: 1.18-2.45) compared to 3.66 U/ml (IQR: 2.81-5.21) in controls, and females exhibited 1.70 U/ml (IQR: 1.40-2.36) vs. 4.14 U/ml (IQR: 2.59-5.02). Among clinical subtypes, segmental and non-segmental vitiligo patients showed reduced CAT and GPx activities compared to controls, with median CAT activities of 2.36 µmol H_2O_2/min (IQR: 1.26-2.45) and 1.79 µmol H_2O_2/min (IQR: 1.34-2.38), respectively.

Table 3. Comparison of lipid peroxidation and protein oxidation markers between vitiligo patients and controls.

Variable	Group (control =110) (vitiligo =110)	MDA (nmol/mL)	PC (nmol/mg)
18-30	Control	0.80(0.55-1.26)	1.59(1.14-2.27)
	vitiligo	1.43(0.69-2.28)	3.41(2.50-5.45)
31-45	Control	0.83(0.57-1.05)	1.59(1.25-2.16)
	vitiligo	1.18(1.53-2.32)	3.30(2.27-4.32)
45-65	Control	0.82(0.56-1.69)	0.91(0.91-2.05)
	vitiligo	1.09(0.48-1.43)	3.07(2.50-3.86)
Male	Control	0.88(0.57-1.27)	1.59(1.14-2.16)
	vitiligo	1.12(0.63-2.26)	3.41(2.27-4.55)
Female	Control	0.72(0.54-1.44)	1.59(1.14-2.27)
	vitiligo	1.35(0.74-2.08)	3.41(2.50-4.77)
Segmental	Control	NA	NA
	vitiligo	1.06(0.65-2.43)	3.18(2.50-5.80)
Non-segmental	Control	NA	NA
	vitiligo	1.19(0.66-2.11)	3.41(2.50-4.66)
Intermediate (segmental+non-segmental)	Control	NA	NA
	vitiligo	1.41(0.88-2.26)	3.53(2.56-5.05)

Values are presented as median IQR. Comparisons between vitiligo patients and healthy controls were performed using the Mann-Whitney U test. Comparisons among vitiligo clinical subtypes (segmental, non-segmental, and intermediate) were carried out using the Kruskal-Wallis test analysis. p-value < 0.05 was considered statistically significant.

MDA- malondialdehyde, PC- protein carbonyl, IQR- interquartile range, NA- not applicable

Oxidative stress markers MDA and PC levels were significantly elevated in vitiligo patients compared to controls across the same subgroups (Table 3). In the 18-30 years age group, median MDA levels were 1.43 nmol/mL (IQR: 0.69-2.28) in patients versus 0.80 nmol/mL (IQR: 0.55-1.26) in controls, while PC levels were 3.41 nmol/mg (IQR: 2.50-5.45) compared to 1.59 nmol/mg (IQR: 1.14-2.27). Both male and female patients demonstrated elevated MDA and PC concentrations relative to controls, with males showing a median MDA of 1.12 nmol/mL versus 0.88 nmol/mL, and females 1.35 nmol/mL versus 0.72 nmol/mL, respectively. Among vitiligo types, segmental and non-segmental groups had higher median PC levels (3.18 and 3.41 nmol/mg), with the intermediate group (segmental + non-segmental) showing a median of 3.53 nmol/mg.

Statistical comparison using the Mann-Whitney U test revealed significant decreases in CAT and GPx activities in vitiligo patients compared with controls ($p < 0.001$ for both). Markers of oxidative damage, MDA and PC, were significantly elevated in patients ($p = 0.002$ and $p < 0.001$, respectively), indicating increased lipid and protein oxidation in vitiligo. However, SOD activity did not differ significantly between groups ($p = 1.000$), suggesting selective impairment in antioxidant enzymes. The Kruskal-Wallis test assessed differences across age categories, showing no significant variation in SOD ($H = 1.823$, $p = 0.402$), GPx ($H = 2.348$, $p = 0.309$), MDA ($H = 0.176$, $p = 0.916$), or PC ($H = 1.119$, $p = 0.572$). In contrast, CAT activity significantly varied by age group ($H = 8.720$, $p = 0.013$), indicating age-related differences in catalase function. Gender comparisons using Mann-Whitney U tests demonstrated no significant differences in SOD ($p = 0.477$), GPx ($p = 0.905$), or MDA ($p = 0.677$). Catalase activity was significantly higher in females than in males ($p = 0.021$). Protein carbonyl levels showed a trend toward difference between genders but did not reach statistical significance ($p = 0.063$).

Lastly, oxidative stress markers and antioxidant enzyme activities did not differ significantly across vitiligo clinical types (segmental, non-segmental, intermediate), as indicated

by Kruskal-Wallis tests for SOD ($p = 0.350$), CAT ($p = 0.633$), GPx ($p = 0.939$), MDA ($p = 0.792$), and PC ($p = 0.936$).

4. DISCUSSION

The present study demonstrated a statistically significant decrease in the activities of SOD, CAT, and GPx, accompanied by substantial increases in MDA and PC levels in vitiligo patients compared with healthy controls. These findings indicate a pronounced oxidative imbalance, supporting the hypothesis that excessive production of ROS together with an impaired antioxidant defense system contributes to melanocyte injury and disease pathogenesis in vitiligo.

Our results are consistent with several earlier reports emphasizing the role of oxidative stress in vitiligo progression. Perumal et al. (2025) documented significantly elevated MDA levels along with significantly reduced SOD, CAT, and GPx activities in patients with generalized and active vitiligo, reporting strong correlations with disease severity and clinical activity (Perumal et al., 2025). Similarly, studies conducted on Indian populations by Arora et al. (2023) demonstrated increased MDA and PC levels coupled with decreased antioxidant enzyme activities, reinforcing the contribution of lipid peroxidation and protein oxidation to disease progression (Arora et al., 2025). These observations, in alignment with our findings, suggest that depletion of enzymatic antioxidants exacerbates ROS-mediated melanocyte destruction, thereby perpetuating depigmentation.

In contrast, Sravani et al. (2009) reported increased SOD levels with decreased CAT activity in both lesional and non-lesional skin (Sravani et al., 2009). They proposed that enhanced SOD activity may represent an adaptive mechanism to neutralize rising superoxide radicals, whereas insufficient CAT activity may allow the accumulation of hydrogen peroxide (H_2O_2). However, in our cohort, both SOD and CAT activities were significantly reduced, indicating a failure rather than a compensatory response of the antioxidant defense system, possibly due to prolonged or overwhelming oxidative stress that exceeds endogenous antioxidant capacity. International studies further corroborate our results. Li et al. (2021), Xuan et al. (2022), and Chang et al. (2023) demonstrated marked elevations in MDA and PC levels with concurrent decreases in SOD, CAT, and GPx activities, reinforcing the consensus that oxidative dysregulation constitutes a fundamental biochemical abnormality in vitiligo (Li et al., 2021;), Xuan et al., 2022; and Chang et al., 2023). Similarly, Khan et al. (2020) and Prasad et al. (2021) reported diminished antioxidant enzyme activities and elevated oxidative damage markers in both serum and epidermal samples of vitiligo patients (Khan et al.,2020; Prasad et al., 2021).

Nevertheless, a limited number of studies have reported partially discordant findings. Passi et al. (1998) and Zedan et al. (2015) observed increased SOD activity in vitiligo patients, interpreting this elevation as an early adaptive response to oxidative burden (. Passi et al., 1998; Zedan et al., 2015). Additionally, investigations by Shajil et al. (2006) and Jain et al. (2018) described non-significant variations in GPx or CAT activities (Shajil et al., 2006; Jain et al., 2018). Such inconsistencies may reflect differences in disease duration, clinical subtype, ethnic or genetic background, sample source (serum versus tissue), or variations in assay methodologies across studies. Overall, the present findings further validate the central contribution of oxidative stress to vitiligo pathophysiology. The consistent elevation of lipid and protein oxidation markers (MDA and PC) combined with depletion of antioxidant defenses (SOD, CAT, and GPx) reflects a state of systemic redox imbalance that predisposes melanocytes to oxidative injury and apoptosis. Although some variability exists among reported studies, these discrepancies underscore the complex and dynamic nature of oxidative mechanisms operating during different disease stages and clinical forms. Collectively, our data support the clinical utility of oxidative stress biomarkers for disease monitoring and highlight antioxidant-based interventions as potential adjunctive therapeutic strategies in vitiligo management.

5. CONCLUSION

The results of this study reinforce the concept that oxidative stress is a fundamental pathological mechanism in vitiligo, supported by decreased activities of key antioxidant enzymes (SOD, CAT, GPx) and increased levels of markers of lipid and protein oxidation (MDA, PC) in affected individuals. Data from diverse Indian and global populations studies consistently demonstrate a disruption in redox homeostasis, which contributes to melanocyte damage and disease progression regardless of geographic, ethnic, or dietary variation. The significant association between impaired antioxidant systems and vitiligo pathogenesis underscores the importance of oxidative imbalance as a diagnostic and therapeutic target. Future research should focus on exploring novel antioxidant therapies and targeted interventions aimed at restoring redox equilibrium and protecting melanocytes, potentially leading to more effective management and improved quality of life for vitiligo patients worldwide.

Ethical Approval

The study was approved by the Institutional Human Ethics Committee of Maharshi Dayanand University, Rohtak, Haryana (HEC No. 2023/39). Participation was entirely voluntary, and written informed consent was obtained from all individuals before their inclusion in the study.

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Disclosure statement

The author declares no competing interests to disclose.

Data Availability: The authors confirm that the data supporting the findings of this study are available within the article.

Ethical approval and Informed consent

The study was approved by the Institutional Human Ethics Committee of Maharshi Dayanand University, Rohtak, Haryana (**HEC No. 2023/39**). Participation was entirely voluntary, and written informed consent was obtained from all individuals before their inclusion in the study.

Author Contributions

Conceptualization: Kumar A; **Original draft preparation/writing:** Jangra S; **Software analysis:** Jangra S and Gulia H; **Formal analysis:** Aggarwal K; Giri S.K; Singh G; **Review and editing:** Dang S. A and Verma N

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