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Evaluation of Antioxidants and Serum Immunoglobulins in Young Adult Females with Striae Distensae.

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ABSTRACT

Objective: Striae distensae constitute linear dermal scars that are accompanied by epidermal atrophy not yet associated with any significant medical problem, but can cause significant distress to its sufferers. The study aimed to analyze the relationship between antioxidants and total antioxidant capacity and Striae distensae in young female adults.

Materials and methods: Using a random sampling technique, sixty (60) young adult females with Striae distensae (test samples) were selected and age-matched with sixty (60) young adult females without Striae distensae, who served as the control group. Ten milliliters (10ml) of venous blood was collected from each participant, it was dispensed into plain containers and was allowed to cloth, and then centrifuged to obtain serum, which was stored at $-20℃$ before use. The antioxidant effect of the samples was studied using ABTS (2, 2'-azino-bis-3-ethyl benthiazoline-6-sulphonic acid) radical; cation decolorization assay. While the immunoglobulins were determined using the Elisa technique, data obtained were subjected to statistical analysis using SPSS 21, and the $P < 0.05$ level of significance was adopted.

Results: The results reveal significantly lower serum levels of GPx(0.1588 ± 0.0264), Catalase(0.4627 ± 0.2608), TAC (10.12 ± 2.186) and SOD (0.8897 ± 0.20708) in young adult females with SD compared to controls with GPx (0.1819 ± 0.474), Catalase (0.6535 ± 0.3112), TAC (11.82 ± 2.991) and SOD (1.0389 ± 0.1994). There are also non-significant correlation of GPx with IgG (r = 0.054, p = 0.684), IgE (r = -0.189, p = 0.148) and IgM (r = 0.058, p = 0.661) as well as there are non-significant correlation of serum SOD with IgG (r = -0.221, p = 0.089), IgE (r = 0.018, p = 0.891) and IgM (r = 0.088, p = 0.504) in young adult females with SD. The results also reveal non-significant correlation of TAC with IgG (r = -0.143, p = 0.275), and IgM (r = -0.071, p = 0.589), IgE (r = 0.180, p = 0.170) and non-significant positive correlation of Catalase with IgG (r = 0.168, p = 0.200) and IgE (r = 0.062, p = 0.637), IgM (r = -0.147, p = 0.262) in young adult females with SD.

Conclusion: This study concludes that Striae distensae is associated with reduced activities of enzymatic antioxidants in young adult females.

I. Introduction

Striae distensae (SD), also known as stretch mark is a common skin condition that is not yet associated with any significant medical problem but can cause significant distress to its sufferers. Striae distensae represent linear dermal scars that are accompanied by epidermal atrophy, as a natural result of the skin stretching, which may diminish over time but will not disappear completely. They are indented; reddened streaks that usually appear on the skin from rapid weight gain or weight change [1].

The classic anatomical sites affected include the abdomen and breast for pregnancy-related striae, the outer thighs or lumbosacral regions for adolescent boys, and the buttocks, thighs, upper arms, and breasts for adolescent girls [2, 3]. Striae progress through three different stages of maturation: the acute stage is characterized by red and slightly raised striae rubrae, the sub-acute stage is characterized by purpuric stage, and the chronic stage is characterized by hypopigmented and atrophic striae albae [4]. Histological studies of mature striae reveal stretched collagen fibers aligned parallel to the skin surface, followed by subsequent loss of collagen and increased flattening of rete ridges [3].

Immunoglobulins (Igs) are glycoprotein molecules called antibodies (Abs), that are produced in response to foreign substances entering the living body-antigens or immunogens (viruses, bacteria, or toxins, etc.), binding them and forming antigen-antibody complexes resulting in antigen (Ag) elimination and protection of the body of the host. Igs are produced by the lymphocytes and are found in infractions of blood called gamma globulin [5]. Igs are synthesized with a molecular arrangement that fits the shape of molecules on the antigens or immunogens, to allow effective binding of the Abs. Igs binding to Ags helps to inactivate, weaken, or enhance the phagocytosis of Ags [6]. They act as a critical part of the immune response by specifically recognizing and binding to particular antigens, and aiding in their destruction [7]. The antibody immune response is highly complex and exceedingly specific. The various immunoglobulin classes and subclasses (isotypes) differ in their biological features, structure, target specificity, and distribution. Hence, the assessment of the immunoglobulin isotype can provide useful insight into complex humoral immune responses [8].

Glutathione peroxidase (GPx) is the general name of an enzyme family with peroxidase activity whose main biological role is to protect the organism from oxidative damage. The biochemical function of glutathione peroxidase is to reduce lipid hydroperoxides to their corresponding alcohols and to reduce free hydrogen peroxide to water [5]. Glutathione (GSH) is an antioxidant in plants, animals, fungi, and some bacteria and Archaea bacteria. Glutathione is capable of preventing damage to important cellular components caused by reactive oxygen species such as free radicals, peroxides, lipid peroxides, and heavy metals [9]. Catalase is an enzyme that brings about (catalyzes) the reaction by which hydrogen peroxide is decomposed to water and oxygen. Found extensively in organisms that live in the presence of oxygen, catalase prevents the accumulation of and protects cellular organelles and tissues from damage by peroxide, which is continuously produced by numerous metabolic reactions. Typical catalases comprise the most abundant group found in Eubacteria, Archaea bacteria, Protista, Fungi, Plantae, and Animalia, whereas catalase–peroxidases are not found in plants and animals and exhibit both catalatic and peroxidative activities [10].

It also has industrial uses to prevent certain contaminants in food as a disinfectant for contact lenses and as a cleansing agent in some other products. Commercial catalases also are used to break down hydrogen peroxide in wastewater. Superoxide dismutase (SOD) is an enzyme that alternately catalyzes the dismutation (or partitioning) of the superoxide (O_2^-) radical into ordinary molecular oxygen (O_2) and hydrogen peroxide (H_2O_2). Superoxide is produced as a by-product of oxygen metabolism and, if not regulated, causes many types of cell damage [11]. Hydrogen peroxide is also damaging and is degraded by other enzymes such as catalase. Thus, SOD is an important antioxidant defense in nearly all living cells exposed to oxygen. One exception is Lactobacillus plantarum and related lactobacilli, which use a different mechanism to prevent damage from reactive (O_2^-) [12]. SOD comprises a family of metalloproteins primarily classified into four groups: copper, zinc-containing SOD (Cu, Zn-SOD), manganese-containing SOD (Mn-SOD), iron-containing SOD (Fe-SOD), and nickel-containing SOD (Ni-SOD) [13]. This study aims to evaluate the serum levels of some immunoglobulins (IgG, IgE, and IgM), the correlation between serum IgM with IgG and IgE, and to determine and compare the body mass index of young adult females with striae distensae and control.

II. Pathogenesis of striae distensae

The pathogenesis of striae distensae is unknown but probably relates to changes in the components of the extracellular matrix, including fibrillin, elastin, and collagen. Elastic fiber is a primary target of the pathological process and the abnormalities extend as far as 3cm beyond the lesion into the normal skin [14]. It has been noted that striae are prevalent in cachetic states, such as tuberculosis, and typhoid, and after intense slimming diets. They may also be seen in anorexia nervosa [15].

Striae or "stretch marks" begin as reddish or purple lesions, which can appear anywhere on the body, but are most likely to appear in places where larger amounts of fat are stored; the most common places are the abdomen (especially near the navel), breasts, upper arms, underarms, back, thighs (both inner and outer), hips and buttocks. Over time, they tend to atrophy and lose pigmentation [16].

The affected areas appear empty and are soft to the touch. Stretch marks occur in the dermis, the resilient middle tissue layer that helps the skin retain its shape [17]. No stretch marks will form as long as there is support within the dermis; stretching plays a role in where the stretch marks occur and in what direction they run, however, there are several contributing factors to their formation. They can (but do not always) cause a burning itching sensation, as well as emotional distress. They pose no health risk in and of themselves, and do not compromise the body's ability to function normally and repair itself; however, they are often considered a cosmetic nuisance [18].

III. Epidemiology of Striae distensae

The exact etiology remains controversial and this is partly due to variability in the clinical situations in which striae arises. They are the result of various physiologic states, including pregnancy, adrenocortical excess, and changes in the body habitus, as seen in rapid weight change [19]. Striae distensae affect skin that is subjected to continuous and progressive stretching; increased stress is placed on the connective tissue due to the increased size of various parts of the body. It occurs on the abdomen and the breast of pregnant women, on the shoulders of builders, in adolescents undergoing their growth, and in individuals who are overweight [20].

IV. Method

I. Study Design and Sample Collection

The period of subject enrollment, classification, sample collection, and laboratory determination of serum immunoglobulins (IgG, IgE, IgM), and antioxidant enzymes (Glutathione peroxidase, catalase, and superoxide dismutase) lasted for three months.

Ten (10) milliliters of venous blood were collected from each participant. It was dispensed into a plain container to obtain serum. The samples were refrigerated at $-20℃$ and analyzed within 1 week.

II. Laboratory Procedures

Reagents were commercially purchased and the manufacturer's operational instruction was followed. Determination of Serum Human Immunoglobulin IgE, IgG, and IgMusing ELISA Kit as Modified by Melsin Medical Co., Limited [21]. To measure the concentration of IgE, IgG, and IgM in the sample, the ELISA kit includes a set of calibration standards. The calibration standards are assayed at the same time as the samples and allowed to produce a

standard curve of Optical Density (O.D) versus IgE, IgG, and IgM concentrations. The concentration of IgE, IgG, and IgM in the samples was then determined by comparing the O.D. of the samples to the standard curve.

SOD was assayed according to the method of [22] based on the inhibition of the formation of NADH-phenazine methosulphate-nitro blue tetrazolium formazon. The color formed at the end of the reaction was sampled into butanol and measured at 560mm.

Catalase activity was assayed following the method of [23] where the UV absorption of hydrogen peroxide was measured at 240nm, and absorbance decreased when degraded by the enzyme catalase. From the decrease in absorbance, the enzyme activity was calculated.

The method proposed by [24] was adopted for assaying the activity of glutathione peroxidase in the presence of the hydrogen donor pyrogallol, where peroxidase converted $H_2 0_2$ to $H_2 O$ and 0_2. The oxidation of pyrogallol to a colored product called purpurogalliwas followed spectro-photometrically at 430nm.

The antioxidant effect of the samples was studied using ABTS (2, 2'-azino-bis-3-ethyl benthiazoline-6-sulphonic acid) radical; cation decolorization assay according to the method of [25].

III. Statistical Analysis

All data generated from this study was subjected to statistical analysis. The mean, standard deviation, students' P–Test, and Pearson's correlation were determined using SPSS version 21 Statistical Package software for Windows. Results were expressed as Mean± SD. The 5% ($P<0.05$) level of significance was adopted for significant findings.

V. Results

Table 1: Mean ±SD values of Glutathione peroxidase, Catalase, TAC, and Superoxide Dismutase in young adult females with striae distensae and control.

Variables (Mean ± SD)	StraieDistensae (n = 60)	Control (n = 60)	t-value	p-value
Glutathione peroxidase (K/mg)	0.1588 ± 0.0264	0.1819 ± 0.474	-3.280	0.002
Lower 95% C.I	0.1520	0.1695		
Upper 95% C.I	0.1656	0.1942		
Catalase (K/mg)	0.4627 ± 0.2608	0.6535 ± 0.3112	-3.632	0.001
Lower 95% C.I	0.3953	0.5731		
Upper 95% C.I	0.5301	0.7339		
TAC (K/mg)	10.12 ± 2.186	11.82 ± 2.991	-7.223	0.000
Lower 95% C.I	9.555	11.05		
Upper 95% C.I	10.69	12.59		
Superoxide Dismutase (K/mg)	0.8897 ± 0.20708	1.0389 ± 0.1994	-4.038	0.000
Lower 95% C.I	0.8362	0.9874		
Upper 95% C.I	0.9432	1.0905		

Table 2: Pearson correlation of TAC with SOD, Catalase, and Glutathione peroxidase in young adult females with striae distensae.

Dependent Variables	n	r-value	P-value
SOD			
Catalase	60	0.038	0.776
Glutathione peroxi			

Table 3: Pearson correlation of SOD, Catalase, and Glutathione peroxidase with each other in young adult females with striae distensae.

	SOD	Catalase	Glutathione Peroxidase
SOD			
r-value	1	-0.315*	-0.048
p-value		0.014	0.715
N	60	60	60
Catalase			
r-value	-0.315*	1	-0.039
p-value	0.014		0.767
N	60	60	60
Glutathione Peroxidase			
r-value	-0.048	-0.039	1
p-value	0.715	0.767	
N	60	60	60

Table 4: Pearson correlation of SOD with IgG, IgE, and IgM in young adult females with striae distensae

Dependent Variables	n	r-value	P-value
IgG	60	−0.221	0.089
IgE	60	0.018	0.891
IgM	60	0.088	0.504

Table 5: Pearson correlation of TAC with IgG, IgE, and IgM in young adult females with striae distensae.

Dependent Variables	n	r-value	P-value
IgG	60	−0.143	0.275
IgE	60	0.180	0.170
IgM	60	−0.071	0.589

Table 6: Pearson correlation of Catalase with IgG, IgE, and IgM in young adult females with striae distensae.

Dependent Variables	n	r-value	P-value
IgG	60	0.168	0.200
IgE	60	0.062	0.637
IgM	60	−0.147	0.262

Table 7: Pearson correlation of Glutathione peroxidase with IgG, IgE, and IgM in young adult females with striae distensae.

Dependent Variables	n	r-value	P-value
IgG	60	0.054	0.684
IgE	60	−0.189	0.148
IgM	60	0.058	0.661

The result in Table 1 showed that there were significantly lower serum levels of Glutathione peroxidase (0.1819 ± 0.474), Catalase(0.4627 ± 0.2608), TAC (10.12 ± 2.186), and Superoxide Dismutase (0.8897 ± 0.20708) in young adult females with SD compared with controls with p = 0.002, 0.001 and 0.000 for each case.

Table 2 shows that there was a non-significant correlation of TAC with Catalase (r = 0.038, p = 0.776), and a non-significant correlation with SOD (r = -0.128, p = 0.332) and Glutathione peroxidase (r = -0.224, p = 0.086) in young adult females with SD.

There was a significant correlation of SOD with Catalase (r = -0.315, p = 0.014), and a non-significant correlation with Glutathione peroxidase (r = -0.048, p = 0.715) while there was a non-significant correlation of Catalase with Glutathione peroxidase (r = -0.039, p = 0.767) in young adult females with SD as revealed in Table 3.

There was a non-significant correlation of serum SOD with IgG (r = -0.221, p = 0.089), IgE (r = 0.018, p = 0.891), and IgM (r = 0.088, p = 0.504) in young adult females with SD as shown in Table 4.

Table 5 revealed that there was a non-significant correlation of TAC with IgG (r = -0.143, p = 0.275), and IgM (r = -0.071, p = 0.589), IgE (r = 0.180, p = 0.170) while Table 6 revealed that there were non-significant positive correlation of Catalase with IgG (r = 0.168, p = 0.200) and IgE (r = 0.062, p = 0.637), IgM (r = -0.147, p = 0.262) in young adult females with SD.

There was a non-significant correlation of Glutathione peroxidase with IgG (r = 0.054, p = 0.684), IgE (r = -0.189, p = 0.148), and IgM (r = 0.058, p = 0.661) in young adult females with SD as shown in Table 7.

VI. Discussion

A greater percentage of SD subjects consider striae distensae as a medical disorder and it has caused some level of psychological distress to them. There were significantly lower serum levels of Superoxide Dismutase (SOD), Glutathione (GPX), Total antioxidant capacity (TAC), and Catalase compared with controls in each case.

SOD catalyzes the dismutation of two superoxide anions into hydrogen peroxide and molecular oxygen [26]. The decreased level of SOD activity may be due to its high utilization to defend the cells from the injurious effects of superoxide radicals [27].

Catalase is recognized to be a secondary antioxidant enzyme in peroxidative defense that hydrolyzes hydrogen peroxide into water and oxygen. The low level of Catalase could be attributed to no feedback effect of hydrogen peroxide on mRNA expression [28, 29]. Though several data suggested that oxidative stress and antioxidant imbalance could play a pivotal role in the pathogenesis of skin diseases, recent evidence has clarified that ROS generation activates both physiologic and pathologic mechanisms which are not peculiar to the skin but that should be considered general organism responses to pro-oxidant stimuli [30]. When free radicals are unable to steal an available electron from other atoms, they may start stealing them from our cells, ultimately causing a breakdown in the DNA of the skin, which can cause serious problems as “healthy” atoms are robbed of their electrons [31]. One of

the most visible signs of free radical-induced damage is premature aging of the skin, as well as inflammation and irritation. The damage also appears on the skin in the form of wrinkles, sagging, dryness, dullness, and unwanted pigmentation, like age spots and broken blood vessels [32]. In defending the cells from the injurious effects of superoxide radicals, young adult females with low serum levels of antioxidants may be highly prone to SD. Extrinsic skin damage develops due to several factors: ionizing radiation, severe physical and psychological stress, alcohol intake, poor nutrition, overeating, environmental pollution, and exposure to UV radiation (UVR). UV-induced generation of ROS in the skin develops oxidative stress when their formation exceeds the antioxidant defense ability of the target cell (Katiyar, and Mukhtar, 2001). Acute exposure to UVR depletes the catalase activity in the skin and increases protein oxidation (Sander, Chang, and Salzmann, 2002).

There was a non-significant correlation of TAC with Catalase, SOD, and Glutathione peroxidase. The SOD has a significant correlation with Catalase and a non-significant correlation with TAC and Glutathione in test samples.

A study on the same samples conducted by [33] showed that serum levels of IgE, IgG, and IgM in young adult females with striae distensae were significantly lower compared with the controls in each case.SOD also has a non-significant correlation with IgG, IgE and IgM. This reveals that the serum level of SOD may not affect the levels of Catalase, TAC, and Glutathione peroxidase. IgG decreases while IgE and IgM increase as SOD increases and vice-versa in subjects. The TAC has a non-significant correlation with IgE, IgG, and IgM. This implies that serum levels of TAC may not affect the levels of IgE, IgG, and IgM in SD subjects. There was a non-significant correlation of Catalase with IgM, IgG, and IgE. This reveals that serum Catalase level may not influence the serum levels of IgM, IgG, and IgE in SD subjects. There was also a non-significant correlation of Glutathione peroxidase with IgE, IgG, and IgM. This reveals that serum levels of Glutathione peroxidase may not affect the levels of IgE, IgG, and IgM in SD subjects.

VII. Conclusion

SD is associated with reduced activities of enzymatic antioxidants.

Reference

1. Hussain, H.A. (2012). Stretch marks and treatment efficacies. *Journal of the American Academy of Dermatology*; 66 (suppl): AB29.
2. Chang, T.W., Wu P.C., Hsu C.L., and Hung A.F., (2007). Anti-IgE antibodies for the treatment of IgE-mediated allergic diseases. Advanced Immunology. 93:63–119.
3. Elsaie, M.L., Baumann, L.S., and Elsaaiee, L.T. (2009). Striae distensae (stretch marks) and different modalities of therapy: an update. *Dermatological Surgery.* 35(4):563-573. doi: 10.1111/j.1524-4725.2009.01094.x. PMID: 19400881.
4. Kim B.J., Lee D.H., Kim M.N., Song K.Y., Cho, W.I., Lee, C.K., Kim, J.Y., Kwon, O.S. (2008). Fractional photothermolysis for the treatment of striae distensae in Asian skin. *American Journal of Clinical Dermatology*. 2008;9(1):33-37. doi: 10.2165/00128071-200809010-00003. PMID: 18092841.
5. Atwal, G.S.S., Manku, L.K., Griffiths, C.E.M., and Polson, D.W. (2006). Striae gravidarum in primiparae. *British Journal of Dermatology*. doi.org/10.1111/j.1365-2133.2006.07427.x.
6. Hermanns, J.F., and Piérard, G.E., (2006). High-resolution chemiluminescence colorimetry of striae distensae. *Journal of European Academy of Dermatology and Venereology*; 20: 282–287.
7. Maverakis E., Kim, K., Shimoda, M., Gershwin, E.M, Patel, F.,Wilken, R., Raychaudhuri, S., Ruhaak, R.L., and Lebrilla, C.B. (2015). *Glycans in the immune system and* The Altered Glycan theory of Autoimmunity: A critical review. *Journal of* Autoimmunity.
8. Barclay, A.N. (2013). The immunoglobulin superfamily--domains for cell surface recognition. *Annual Review Immunology.* 6:381-405. doi: 10.1146/annurev.iy.06.040188.002121. PMID: 3289571.
9. Espindola, S., Gomes, D.S., Panek, A.D., and Eleutherio, E.C., (2003). The role of glutathione in yeast dehydration tolerance. Cryobiology. 2003;47:236–241.0011-2240 (2003)047.
10. Zamocky, M., Furtmüller, P. G., &Obinger, C. (2008). Evolution of catalases from bacteria to humans. *Antioxidants & redox signaling*, *10*(9), 1527–1548. https://doi.org/10.1089/ars.2008.2046
11. Hayyan, M., Hashim, M.A., Al Nashef, I.M., (2016). Superoxide Ion: Generation and Chemical Implications. Chem. Rev. 116 (5): 3029–3085.Garcia-Hidalgo, L., (2002). Dermatological complications of obesity. *American Journal of Clinical Dermatology*; 3:497-506.
12. McCord, J.M., Fridovich, I. (1988). Superoxide dismutase: the first twenty years (1968-1988). *Free Radical Biology & Medicine*. 5 (5–6): 363–369.
13. Xueji Zhang, Huangxian Ju, and Joseph Wang. (2008). Electrochemical Sensors, Biosensors, and their Biomedical Applications. Academic Press. ISBN: 978-0-12-373738-0. doi.org/10.1016/B978-0-12-373738-0.X5001-6
14. McAvoy, B.R. (2013). No evidence for topical preparations in preventing stretch marks in pregnancy. *British Journal of General Practice*, *63*(609), 212.

15. Robinson, P.N., Arteaga-Solis, E., Baldock, C., Collod-Béroud, G., Booms, P., De Paepe, A., and Godfrey, M. (2013). The molecular genetics of Marfan syndrome and related disorders. *Journal of Medical Genetics*, 769-787.

16. De Angelis, F., Kolesnikova, L., Renato, F. and Liquori, G., (2011). Fractional nonablative 1540-nm laser treatment of striae distensae in Fitzpatrick skin types II to IV: Clinical and histological results. *Aesthetic Surgery Journal, 31*(4): 411-419.

17. Hexsel, D. (2014). Superficial dermabrasion versus topical tretinoin on early striae distensae A randomized, pilot study. *Dermatologic Surgery*.; 40:537.

18. Al-Himdani, S., and Arteaga, S. (2014). Striae distensae: A comprehensive review and evidence-based evaluation of prophylaxis and treatment. *British Journal of Dermatology*. 170:52-7.

19. Belo, V., Arceo-Cruz, M.C., and Buse E., (2009). The efficacy of fractional resurfacing using an erbium glass laser in the treatment of stretch marks on Asian skin. *Laser Surgery Medicine*. 41: 92.

20. Lebwohl, M.G. (2015). Striae. Treatment of Skin Disease: Comprehensive Therapeutic Strategies. 4th ed. *Philadelphia, Pa.: Elsevier*; 2014.

21. Per, K.E., and Bo, T.A. (1998). A Research Standard for Human Serum Immunoglobulins IgG, IgA and 1gM. *The Journal of allergy and clinical immunology*, *125*(2 Suppl 2), S41–S52.

22. Kakkar, P., Das, B., and Viswanathan, P.N. (1984). A modified spectrophotometric assay of superoxide dismutase. Indian Journal of Biochemistry and Biophysics. 21(2):130-132. PMID: 6490072.

23. Luck H., (1974). Methods in enzymatic analysis. Academic Press, New York, 885.

24. Reddy, C., Channa, P.M., Moriarty, Mary, F.P. and John L.B. (1995). Classical Selenium-Dependent Glutathione Peroxidase Expression Is Decreased Secondary to Iron Deficiency in Rats. *The Journal of Nutrition*, Volume 125, Issue 2, February 1995, Pages 293–301.

25. Shirwaikar, A., Prabhu, K.S. and Punitha, I.S.R. (2006). In vitro antioxidant studies of Sphaeranthus indicus (Linn). *Indian Journal of Experimental Biology (IJEB)*. Vol.44 (12) pp: 993-996.

26. Momen-Beitollahi J., Mansourian A., Momen-Heravi F., Amanlou M., Obradov S., and M. Sahebjamee (2010). Assessment of salivary and serum antioxidant status in patients with recurrent aphthous stomatitis. *Medicina Oral, Patologia Oral y CirugiaBucal*, vol. 15, no. 4, pp. e557–e561.

27. Gupta Ishita, Shetti Arvind, Keluskar Vaishali, and Bagewadi Anjana (2014). Assessment of Serum Enzymatic Antioxidant Levels in Patients with Recurrent Aphthous Stomatitis: A Case Control Study.Hindawi Publishing Corporation Enzyme Research. Volume 2014, Article ID 340819, pp41-46.

28. Karincaoglu Y., Batcioglu K., Erdem T., Esrefoglu T., and Gene M., (2005). The levels of plasma and salivary antioxidants in the patient with recurrent aphthous stomatitis. *Journal of OralPathology and Medicine*, vol. 34, no. 1, pp. 7–12, 2005.

29. Saxena S. (2011). Assessment of plasma and salivary antioxidant status in patients with recurrent aphthous stomatitis. *RSBO*, vol. 8, no. 3, pp. 261–265.

30. Briganti S., and Picardo M. (2003). Antioxidant activity, lipid peroxidation, and skin diseases. What's new? *Journal of the European Academy of Dermatology and Venereology.* 17(6):663-669.

31. Meffert, H., Diezel, W., and Sönnichsen, N. (2006). Stable lipid peroxidation products in human skin: detection, ultraviolet light-induced increase, pathogenic importance. *Experientia*. **32,** 1397–1398. doi.org/10.1007/BF01937397

32. Petersen, R.C., Reddy, M.S., and Liu P-R (2018). "Advancements in Free-Radical Pathologies and an Important Treatment Solution with a Free-Radical Inhibitor. *Journal of Biotechnology and Biomedical Engineering*, vol. 1, no. 1, 2018, pp. 1003.

33. Iwuamadi, O.C., Ikaraoha, C.I., & Nwadike, C.N., (2022). Evaluation of Serum Immunoglobulins and Body Mass Index in Young Adult Females with Striae Distensae in Imo State University, Owerri. *Asian Journal of Research in Dermatological Science*, 5(1), 36–43.