Original Research Article

BLOOD GLUCOSE AND HAEMATOLOGICAL CHANGES IN YOUNG ADULT FEMALES WITH STRIAE DISTENSAE IN OWERRI, NIGERIA.

ABSTRACT

Striae distensae is a skin condition associated with cosmetic morbidity and psychological distress, particularly in young adult females. This study was designed to appraise if there are alterations in blood glucose level and hematological profile of young adult females with Striae distensae. Fouty (40) young adult females with striae distensae and 40 young adult female controls participated in this study. Via venipuncture technique, 5 milliliters of blood were collected from all participants. Two (2) milliliters were dispensed into fluoride oxalate container, while 3 milliliters were dispensed into Ethylene Diamine Tetracetic Acid (EDTA) container, and were analyzed for blood glucose and haematological profile respectively. IBM SPSS version 21 was employed in determination of student t-test and Pearson correlation. Values were accepted to be statistically significant at p<0.05. The result shows significantly lower (p < 0.05) total WBC, Platelets, MCV, MCH, MCHC and PDW levels in young adult females with striae distensae $(5.14 \pm 1.02 \times 10^{9})$ L, $140.10 \pm 45.74 \times 10^{9}$ L, 84.14 ± 5.58 fL, 28.25 ± 2.25 pg, 335.70 ± 6.20 g/L and 15.84 \pm 0.59 respectively) compared to the controls (6.18 \pm 1.32 x 10^9/L, 195.70 \pm 85.79×10^{9} L, 87.00 ± 4.71 fL, 29.82 ± 1.28 pg, 343.00 ± 5.69 g/L and 16.40 ± 0.39 respectively). Blood Glucose, Eosinophils and PCT levels were significantly higher (p < 0.05) in young adult females with striae distensae (101.85 \pm 12.69 mg/dL, 2.01 \pm 1.32 % and 2.00 \pm 0.88 mL/L respectively) compared to the controls (85.80 \pm 12.12 mg/dL, 1.16 \pm 0.17 %, 1.47 \pm 0.48 mL/L respectively). There was non-significant difference (p > 0.05) in Neutrophils, Lymphocytes, Monocytes, RBC, Haemoglobin, HCT and MPV levels in young adult females with striae distensae compared to the controls. There was a significant negative correlation of total WBC with Eosinophil (r = -0.651, p = 0.002) in young adult females with striae distensae. In conclusion, the higher levels of blood glucose, eosinophils, MCHC and PCT parallels lower levels of total WBC, platelets, MCV, MCH and PDW observed in young adult females with striae distensa. This may be associated with the development of striae distensae in this environment.

Key Words: striae distensae, blood glucose, hematological profile, young-adult-females, Nigeria.

INTRODUCTION

Striae distensae (SD) or Stretch marks are observable lined scars that grow in parts of dermal damage as a outcome of extreme widening of the skin. It has an estimated prevalence of 50-80% [1]. Striae distensae generally develop in several physiological conditions including gestation, puberty associated growth or speedy alteration in proportion of precise body areas as seen in overweight or weight loss and weight lifters [2]. SD sometimes manifest in pathological situations with high cortisol level like Cushing's syndrome and hereditary ailments like Marfan syndrome [3].[4]. Numerous factors are involved in the development of SD, yet the precise etiopathogenesis of SD still remains contentious. Main pathology rests in changed dermal connective tissue framework connecting components of extracellular matrix (ECM) specifically collagen, fibronectin, fibrillin, and elastin [5].

Blood Glucose is the end product of carbohydrate metabolism and hence the principal source of energy production in humans. Special transport proteins are involved in the active transport of glucose into body cells, which occurs simultaneously with sodium ions uptake [6].

Some hormones interplay in the careful maintenance of blood glucose concentration of healthy humans, by participating in glycolysis and glycogenolysis. Hence disease conditions are associated with alterations in blood glucose level as seen in liver failure and consequently hypoglycemia. There are also indications that pro-inflammatory situations that encourage leukocytosis may be associated with hyperglycemia [7].

Hematological Profile, frequently referred as Full Blood Count (FBC) consist of a list of clinical laboratory test that proffer information of blood cells in humans. It specifies Haemoglobin (Hb) concentration, white blood Cell (WBC) count, Platelets count, packed cell volume (PCV), red blood cell (RBC) count, red blood cell indices, differential white blood cell count, and blood cell morphological abnormalities [8]. Hematological profile is frequently employed in the over-all assessment of human health. It is among the most regularly requested medical laboratory test for hospital patients.

Interpretation of hematological profile result in association with clinical presentation of the patient is decisive in diagnosis and management [9]. Hematological profile may be affected by numerous factors viz gender, age, disease conditions, diet, genetic factors, stress and environmental factors [10].

There is poor understanding of the factors that contribute to the development of striae distensae. There are divergent views on the causes of striae distensae, one report is of the view that striae develops due to stressful rupture of framework of the connective tissue [11]. Another report is of the opinion that striae effortlessly develop due to presence of raised amount of rigid cross-linked collagen on skin [12]. Also, raised adrenal cortical stimulations and secretions as seen in Cushing syndrome has been opined as a main factor consequential to development of striae distensae [12, 13].

Furthermore, adjustments of the cellular and extracellular matrix (ECM) that arbitrate the clinical manifestation of striae distensae is not completely understood [14].

Numerous exhaustive studies have assessed the effects of SD and its temporary treatments [15, 16], yet the pathogenesis of striae distensae remains a sheer assumption.

A successful mechano-chemical devised model study has demonstrated a remarkable similarity of platelets mediated wound healing process with the pattern forming process of striae distensae [17]. Thus, the need for a detailed study on association of haematological profile with development of striae distensae. Besides, there is scarcity of report in blood glucose level and haematological profile of Striae distensae Sufferers worldwide and particularly in Nigeria. Therefore, this effort was made to examine possible role of blood glucose level and hematological profile in the development and prognosis of Striae distensae in Owerri, Nigeria.

METHODOLOGY

Area of study

Imo State University, Owerri, Nigeria was selected as the area of study.

Study Population

By random sampling method, 40 young adult females within the range of 18 to 25 years who had striae distensae were selected from students of Imo State University, Owerri Nigeria. They were age matched with 40 young adult females without striae distensae who served as controls.

Inclusion Criteria:

- I) Young adult females with striae distensae.
- II) Subjects that gave informed consent.
- III) Subjects within the age range of 18 to 25 years.
- IV) Apparently healthy students.

3.5. Exclusion Criteria:

- I) Subjects having other skin diseases like eczema.
- II) Subjects having chronic diseases.
- III) Subjects that did not give their informed consent.

Specimen Collection and Processing

Five (5ml) of venous blood was collected from the subjects by venipuncture using sterile needle and syringes. About 2 milliliters was dispensed into fluoride oxalate container while 3 milliliters were dispensed into Ethylene Diamine Tetracetic Acid (EDTA) container. The containerswere

properly labelled before commencement of analytical procedures. They were stored refrigerated at 2-8 °C until analysed within 5 hours after collection.

Analytical Methods

All reagents used were commercially prepared and procured and the manufacturer's standard operating procedures were strictly followed. Blood glucose was determined employing Glucose GOD-PAP method **as described by** Ambade et al., (1998) [18] using reagent kits (Cat no GL364) manufactured by Randox diagnostics.

Glucose is oxidized to gluconic acid and hydrogen peroxide by the catalytic action of glucose oxidase (GOD). Further catalytic action by peroxidase (POD), breaks down the hydrogen peroxide to liberate oxygen which combines with phenol and 4-aminophenazone (4-aminoantipyrine) to generate a pinkish coloration., whose absorbance is measured at 520nm via a spectrophotometer.

Hematological profile was determined by the automation method as previously described by Yun-A et al, (2013) [19] using *Mindray BC 6800 Automated Hematology Analyzer (Mindray, China)*, supplied by Med Sing Long Global Group Co LTD, GuangZhou City, China)

Statistical Analysis

IBM SPSS version 21 was employed in determined of student t-test and Pearson correlation. The obtained values were stated as mean \pm standard deviation, Values with p < 0.05 were accepted to be statistically significant. The data obtained in the present study is normal distribution.

RESULTS

Blood Glucose, Total WBC, Neutrophils, Lymphocytes, Monocytes, Basophils, Eosinophils, RBC, HB and Platelets in Young Adult Females with Striae Distensae Versus Controls in Owerri, Imo State.

There were significant decreases in the mean levels of serum total white blood cells and Platelets (p = 0.005 and p = 0.026 respectively) in young adult females with striae distensae when compared to controls. Blood Glucose and Eosinophils were significantly increased in young female adult striae distensae sufferers (p = 0.000 and p = 0.010 respectively) when compared to the controls.

There were non-significant differences in Neutrophils, Lymphocytes, Monocytes, Red blood cells and Haemoglobin (p = 0.385, p = 0.227, p = 0.732, p = 0.084 and p = 0.612 respectively) in young adult females with striae distensae when compared to the controls.

The mean levels of Basophil in young adult females with striae distensae and controls was zero, hence it was not highlighted in the table (Table 1).

Blood HCT, MCV, MCH MPV, PDW and PCT in Young Adult Females with Striae Distensae Versus Controls in Owerri, Imo State.

There were significant decreases in the mean levels of Mean Cell Volume, Mean Cell Haemoglobin, Mean Cell Haemoglobin Coefficient and Platelet Distribution Width (p = 0.034, p = 0.005, p = 0.003 and p = 0.004 respectively) in young adult females with striae distensae when compared to controls. The mean level of Platecrit was significantly increased in young adult females with striae distensae (p = 0.040) when compared to the controls.

There were non-significant differences in Hematocrit and Mean Platelet Volume (p = 0.750 and p = 0.920 respectively) in young adult females with striae distensae when compared to the controls (Table 2).

Pearson Correlation of Blood Glucose with WBC, RBC, Hb and Platelets in Young Adult Females with Striae Distensae in Owerri, Imo State.

There was non-significant correlation of blood glucose with White blood cells ($\mathbf{r} = -0.195$, $\mathbf{p} = 0.409$), Red blood cells ($\mathbf{r} = 0.267$, $\mathbf{p} = 0.255$), Haemoglobin ($\mathbf{r} = -0.150$, $\mathbf{p} = 0.529$) and Platelets ($\mathbf{r} = -0.056$, $\mathbf{p} = 0.815$) in young adult females with striae distensae in Owerri, Imo state (Table 3).

Pearson Correlation of WBC with Neutrophil, Lymphocyte, Monocytes and Eosinophil in Young Adult Females with Striae Distensae in Owerri, Imo State.

There was a significant negative correlation of Total White blood cells with Eosinophil (r = -0.651***, p = 0.002) in young adult females with striae distensae in Owerri, Imo State.

There was non-significant correlation of Total White blood cells with Neutrophil ($\mathbf{r} = 0.261$, $\mathbf{p} = 0.266$) Lymphocyte ($\mathbf{r} = -0.154$, $\mathbf{p} = 0.517$) and Monocyte ($\mathbf{r} = 0.024$, $\mathbf{p} = 0.921$) in young adult females with striae distensae in Owerri, Imo State (Table 4).

Table 1: Blood Glucose, Total WBC, Neutrophils, Lymphocytes, Monocytes, Basophils, Eosinophils, RBC, HB and Platelets in Young Adult Females with Striae Distensae and Controls

Variables Young Adult Females Controls <u>t-v</u>alues <u>p-v</u>alues with Striae Distensae

Blood Glucose (mg/dI	L) 101.85 ± 12.69	85.80 ± 12.12	4.465	0.000
Lower 95% C.I	95.91	80.12		
Upper 95% C.I	107.78	91.47		
11				
Total WBC (10^9/L)	5.14 ± 1.02	6.18 ± 1.32	-3.138	0.005
Lower 95% C.I	4.66	5.55		
Upper 95% C.I	5.62	6.80		
Neutrophils (%)	48.84 ± 7.22	47.24 ± 4.70	0.889	0.385
Lower 95% C.I	45.45	45.03	4	
Upper 95% C.I	52.22	49.44		
Lymphocytes (%)	43.44 ± 6.94	45.74 ± 5.92	-1.249	0.227
Lower 95% C.I	40.19	42.96		
Upper 95% C.I	46.68	48.51		
Monocytes (%)	5.71 ± 1.82	5.86 ± 1.63	-0.348	0.732
Lower 95% C.I	4.85	5.09		
Upper 95% C.I	6.56	6.62		
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Eosinophil (%)	2.01 ± 1.32	1.16 ± 0.17	2.872	0.010
Lower 95% C.I	1.38	1.07		
Upper 95% C.I	2.63	1.24		
		7		
RBC (10^12/L)	4.53 ± 0.54	4.33 ± 0.39	1.823	0.084
Lower 95% C.I	4.28	4.15		
Upper 95% C.I	4.79	4.52		
	10.77 . 1.45	10.04 - 1.01	0.516	0.610
Hb (g/dL)	12.77 ± 1.45	12.94 ± 1.31	-0.516	0.612
Lower 95% C.I	12.09	12.32		
Upper 95% C.I	13.44	13.55		
Platelets (10^9/L)	140.10 ± 45.74	195.70 ± 85.79	-2.420	0.026
Lower 95% C.I	140.10 ± 45.74 118.69	195.70 ± 85.79 155.54	-2.420	0.020
Upper 95% C.I	161.50	235.85		

(n = 40) (n = 40)

 $(Mean \pm SD)$

Table 2: Blood HCT, MCV, MCH MPV, PDW and PCT in Young Adult Females with Striae Distensae Versus Controls in Owerri, Imo State.

Variables	Young Adult Femal with Striae Distensa		<mark>t</mark> -v <mark>alues</mark>	p - values
$(Mean \pm SD)$	$(\mathbf{n} = 40)$	(n = 40)		
HCT (%)	38.06 ± 4.28	37.76 ± 4.11	0.323	0.750
Lower 95% C.I	36.05	35.83		
Upper 95% C.I	40.06	39.68		
MCV (fL)	84.14 ± 5.58	87.00 ± 4.71	-2.278	0.034
Lower 95% C.I	81.52	84.79		
Upper 95% C.I	86.75	89.20		
MCH (pg)	28.25 ± 2.25	29.82 ± 1.28	-3.158	0.005
Lower 95% C.I	27.19	29.82 ± 1.28	-3.136	0.003
Upper 95% C.I	29.30	3042		
Opper 95% C.1	29.30	3042		
MCHC (g/L)	335.70 ± 6.20	343.00 ± 5.69	-3.451	0.003
Lower 95% C.I	332.79	340.33		
Upper 95% C.I	338.60	345.66	Y	
MPV (fL)	10.16 ± 1.13	10.12 ± 0.80	0.102	0.920
Lower 95% C.I	9.63	9.74		
Upper 95% C.I	10.68	10.49		
PDW (-)	15.84 ± 0.59	16.40 ± 0.39	-3.255	0.004
Lower 95% C.I	15.56	16.21		
Upper 95% C.I	16.11	16.58		
PCT (mL/L)	2.00 ± 0.88	1.47 ± 0.48	2.211	0.040
Lower 95% C.I	1.58	1.24		
Upper 95% C.I	2.41	1.70		

Table 3: Pearson Correlation of Blood Glucose with WBC, RBC, Hb and Platelets in Young Adult Females with Striae Distensae in Owerri, Imo State.

Dependent Variables	n	<mark>r</mark> - value	p – value
WBC	40	-0.195	0.409
RBC	40	0.267	0.255
Hb	40	-0.150	0.529
Platelets	40	-0.056	0.815

Table4 Pearson Correlation of WBC with Neutrophil, Lymphocyte, Monocytes and Eosinophil in Young Adult Females with Striae Distensae in Owerri, Imo State.

Dependent Variables	n	<u>r</u> - value	<mark>p</mark> - value
Neutrophil	40	0.261	0.266
Lymphocyte	40	-0.154	0.517
Monocyte	40	0.024	0.921
Eosinophil	40	-0.651**	0.002

^{*} Correlation is significant at the 0.05 level (2-tailed).

^{*} Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

^{**} Correlation is significant at the 0.01 level (2-tailed).

DISCUSSION

Striae distensae is a skin disorder with no critical clinical problem, but of significant distress.to affected individuals [20]. In this study, the mean value of blood glucose was significantly higher in young adult females with striae distensae when compared to the controls. There is paucity of report to support the relationship of blood glucose with Striae distensae. However, it has been noted that blood glucose is considered a main risk factor in development of obesity and cardiovascular diseases, of which emerging scientific evidences signify close association with some skin diseases [21, 22]. The scientific link between glucose and Striae distensae is a subject of on-going investigations. The induction of chronic inflammation was initially thought as the bridging gap [23.22], but oxidative stress induction and the role of endocrine abnormalities were lately included as possible links between certain skin diseases, obesity and other components of Metabolic disorders such as diabetes mellitus [23.22].

The mean levels of Eosinophils and PCT were significantly higher in young adult females with striae distensae when compared to the controls in this study. Though eosinophilia is a common observation in cutaneous inflammation, nevertheless its role in the pathogenesis of cutaneous disease is not well understood. Current research on the structure, constituent, and actions of the eosinophil revealed that eosinophil has potent noxious proteins that can easily facilitate tissue damage [24,25,26]. Eosinophil can disrupt the tissue by depositing noxious protein granules, as shown by immunofluorescent localization of eosinophil granule proteins. This occurrence in numerous diseases is massively out of proportion to the number of recognizable cells and specifies that eosinophil participation in cutaneous disease cannot be arbitrated via the sum of

intact eosinophils in the tissue. In eczematous lichenified conditions with increased serum immunoglobulin E, there is precise deposition of granule proteins outside of eosinophils [27,28,29].

According to data from this study, there were significantly lower mean levels of WBC, Platelets, MCV, MCH, MCHC and PDW in young adult females with striae distensae when compared to controls. There is paucity of similar findings in the past, though platelet rich plasma (PRP) has been suggested in other studies as a treatment approach to Striae distensae [16]. Localized chronic inflammation is reportedly a crucial factor in the pathophysiology of striae distensae. This can be ameliorated by the wound healing properties of platelet-rich plasma by acting on the erythrocytes, endothelial cells and collagen This further suggests a decrease in platelets in Striae distensae sufferers, hence the need for a platelet rich plasma therapy [30,31].

The observed lower total WBC count in young female adults with striae distensae in this study may be due to presence of striae distensae in these subjects. Radiation therapy, chemotherapy, infection and hematopoietic stem cell aberration, disallowing normal bone marrow growth and maturation can grossly reduce WBC count. Neutropenia is mostly the cause of reduced WBC count. but in this study, it is observed that the decreased WBC count is due to lower mean levels of lymphocytes and monocytes [32,33]. However, previous report shows positive association of WBC count with some metabolic and inflammatory risk factors in healthy adult females [34]. This study also showed non-significant differences in Neutrophils, Lymphocytes, Monocytes, RBC, Haemoglobin, HCT and MPV in young adult females with striae distensae when compared to the controls.

Conclusion

Higher levels of blood glucose, eosinophils, MCHC and PCT parallels lower levels of total WBC, platelets, MCV, MCH and PDW observed in young adult females with striae distensae. These alterations may be associated with the development of Striae distensae in this environment.

Recommendations

Expanding the scope of the study to include histological and other biochemical studies might show a bigger picture and understanding of striae distensae among young adult females.

ETHICAL APPROVAL AND CONSENT

The study protocol was approved by the Department of Medical Laboratory Science, Imo state University, Owerri, Nigeria, Research Ethics Committee with reference number MLS/IMSU/REC/2021/05. Written informed consent was obtained from all study participants prior to their enrolment and collection of blood samples in accordance with the "1964 Helsinki declaration" and its later amendments in 2000.

DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

REFERENCES

- [1]. Atwal GS., Manku LK, Griffiths CE, Polson DW. Striae gravidarum in primiparae. Br J Dermatol. 2006; 155:965-969.
- [2] Sisson WR. Colored striae in adolescent children. J Pediatr 1954; 45:520–30.

- [3] Nieman LK, Ilias I. Evaluation and treatment of Cushing's syndrome. Am J Med 2005; 118: 1340-1346.8.
- [4] Tunzi M. and Gray GR. Common skin conditions during pregnancy. Am Fam Physician 2007; 75: 211-218.
- [5] Tung JY, Kiefer AK, Mullins M, Francke U, Eriksson, N. Genome-wide association analysis implicates elastic microfibrils in the development of non-syndromic striae distensae. Journal of Investigative Dermatology. 2013;133(11):2628-31.
- (6) Navale AM, Paranjape AN. Glucose transporters: physiological and pathological roles. Biophysical Reviews, 2016; 8(1), 5–9.
- [7] Nagareddy PR, Murphy AJ, Stirzaker RA, Hu Y, Yu S, Miller RG, Ramkhelawon B, Distel E, Westerterp M, Huang LS, Schmidt AM, Orchard TJ, Fisher EA, Tall AR, Goldberg IJ. Hyperglycemia promotes myelopoiesis and impairs the resolution of atherosclerosis. Cell Metab. 2013 May 7;17(5):695-708.
- [8] Tefferi A, Hanson CA, Inwards DJ. How to interpret and pursue an abnormal complete blood cell count in adults. Mayo Clinic Proceedings. 2015;80 (7): 923–936.
- [9] Hansel A, Hong S, Camara RJ, Von Kanel R. Inflammation as a psychophysiological biomarker in chronic psychosocial stress. Neuroscience and Biobehavioral Review. 2010; 35:115-121.
- [10] Chmielewski PP, Strzelec B. Elevated leukocyte count as a harbinger of systemic inflammation, disease progression, and poor prognosis: a review. Folia Morphologica. 2017;77(2):171-178.
- [11] Agache P, Ovide MT, Kienzler JL, et al. Mechanical factors in striae distensae. In: Morettig G, Rebora A, editors. Stria Distensae. Milan: Brocades; 1976. p. 87-96.
- [12] Magiakou MA, Mastorakos G, Chrousos GP. Final stature in patients with endogenous Cushing's syndrome. *Journal of Clinical Endocrinology and Metabolism*. 1994; 79:1082-1085.
- [13] Cho S, Park ES, Lee DH, Li K., Chung JH. (2006). Clinical features and risk factors for striae distensae in Korean adolescents. Journal of European Academy of Dermatology and Venerology. 20(9): 1108–1113.
- [14] Alaiti S. (2017). Striae distensae differential diagnosis. Journal of the American Academy of Dermatology.195(4): 497-502.
- [15] Ud-Din S, McGeorge D, Bayat A. Topical management of striae distensae (stretch marks): Prevention and therapy of striae rubrae and albae. Journal of European Academy of Dermatology and Venerology. 2016; 30:211-22.

- [16] Hague A and Bayat A. Therapeutic targets in the management of striae distensae: a systematic review. Journal of American Academy of Dermatology. 2017;77(3):559-568.
- [17] Maini P. Mechanochemical model for adult dermal wound contraction. Journal of Biological Systems. 2015; 3(4): 1021-1031.
- [18] Ambade VN, Sharma, YV, Somani BL. Methods for estimation of blood glucose: a comparative evaluation. Medical journal, Armed Forces India, 1998;54(2), 131–133.
- [19] Yun-A Jo, Miyoung Kim, Hyun Soo Kim, Hee Jung Kang, Young Kyung Lee (2013) Evaluation of the Mindray BC-6800 Complete Blood Counts Analyzer. Laboratory Medicine Online 3(3):131.
- [20] Sheu HM, Yu HS, Chang CH. Mast cell degranulation and elastolysis in the early stage of striae distensae. Journal of Cutaneous Pathology. 1991;18(6):410-416.
- [21] Kleinman WB. Distal radius instability and stiffness. Common complications of Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. (Adult Treatment Panel III). Circulation. 2002;106 (25):3143-421.
- [22] Tanmay PG. Metabolic syndrome and skin: psoriasis and beyond. Indian Journal of Dermatology. 2013;58(4):299-305.
- [23] Rasouli N, Kern PA. Adipocytokines and the metabolic complications of obesity. Journal of Clinical Endocrinology and Metabolism. 2008; 93(11): S64-73.
- [24] Erwin WG. Eosinophils in Human Disease. Eosinophils in Health and Disease, 2013; 431–536.
- [25] Zucker-Franklin, D. (). Eosinophil structure and maturation. In: Mahmoud AAF Austen KF editors. The eosinophil in health and disease. Grune and Stratton, Incorporated, New York: 1980 pp. 43-59.
- [26] Miller F, DeHarven E, Palade GE. The structure of the eosinophil leukocyte granules in rodents and man. Journal of Cell Biology. 1996;31: 349-362.
- [27] Gleich GJ, Loegering DA, Maldonado JE. Identification of a major basic protein in guinea pig eosinophil granules. Journal of Experimental Medicine. 1973;137: 1459-1471.
- [28] Parmley RT, Spicer SS. Cytochemical and ultrastructural identification of a small type granule in human late eosinophils. Laboratory Investigation. 1974; 30: 557-567.
- [29] Wasmoen TL, Bell MP, Loegering DA. Biochemical and amino acid sequence analysis of eosinophil granule major basic protein. Journal of Biology and Chemistry. 1988;263: 12559-125563.

- [30] Samadi P, Sheykhhasan M, Khoshinani HM. The use of platelet-rich plasma in aesthetic and regenerative medicine: a comprehensive review. Aesthetic and Plastic Surgery. 2018; 43:803-814.
- [31] Cho EB, Park GS, Park SS. Effect of platelet-rich plasma on proliferation and migration in human dermal fibroblasts. Journal of Cosmetic Dermatology. 2019;18(4):1105-1112.
- [32] Kim AH, Lee W, Kim M, Kim Y, Han K.. White blood cell differential counts in severely leukopenic samples: a comparative analysis of different solutions available in modern laboratory hematology. Blood Research. 2014;49(2):120-6.
- [33] Gulack BC, Englum BR, Lo DD, Nussbaum DP, Keenan JE, Scarborough JE. 2015. Leukopenia is associated with worse but not prohibitive outcomes following emergent abdominal surgery. Journal of Trauma and Acute Care Surgery. 79 (3):437-443.
- [34] Farhangi MA, Keshavarz SA, Eshraghian M, Ostadrahimi A, Saboor-Yaraghi AA. White blood cell count in women: relation to inflammatory biomarkers, haematological profiles, visceral adiposity, and other cardiovascular risk factors. Journal of Health Population and Nutrition. 2013;31(1):58-64.