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Evaluation of Some Inflammatory Markers in Young Adults Females with Striae Distensae

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Abstract

Striae distensae (SD), commonly known as stretch marks, are dermal scars that develop due to rapid stretching of the skin, often observed in young adult females during periods of hormonal or physical changes such as puberty, pregnancy, or weight fluctuations. Although typically considered cosmetic, emerging evidence suggests a possible link between SD and systemic alterations in inflammatory and hematological parameters. This study aimed to evaluate the levels of selected inflammatory markers and hematological parameters in young adult females presenting with striae distensae at Imo State University, Owerri. A total of one hundred (100) participants were recruited for the study, comprising fifty (50) young adult females with confirmed SD and fifty (50) apparently healthy age-matched females without SD serving as controls. Informed consent was obtained from all participants. S EVEN milliliters (7ml) of venous blood were collected aseptically from each participant. 2ml was dispensed into EDTA container and mixed with inversion. The anticoagulant samples were used for determination of heamatological parameters. The remaining 5ml was dispensed into the vaccutainer tube with minimal stasis, allowed to clot, and centrifuged at 3000 rpm for 5 minutes to obtain serum. Inflammatory markers (CRP, IL-6, and TNF-α) were analyzed using enzyme-linked immunosorbent assay (ELISA) methods. Data collected were analyzed using the Statistical Package for Social Sciences (SPSS) version 27.0 and expressed as mean ± standard deviation (SD). A P-value less than 0.05 ($P < 0.05$) was considered statistically significant. Results showed that Patients with Striae Distensae exhibited significantly elevated inflammatory markers compared to controls. The mean CRP level in the test group was 45.86 ± 0.85 mg/L versus 31.78 ± 1.74 mg/L in controls ($P < 0.05$). IL-6 was 8.87 ± 0.15 pg/mL in patients and 6.72 ± 0.13 pg/mL in controls ($P < 0.05$), while TNF-α measured 150.66 ± 2.84 in patients versus 121.69 ± 1.46 in controls ($P < 0.05$). This study has demonstrated significant elevations in inflammatory markers alterations in a cohort of patients with chronic inflammation. The cytokine dysregulation observed particularly involving CRP, IL-6, and TNF-α is reflective of active systemic inflammation and provides a plausible mechanistic link to dermal tissue remodeling, which is relevant to SD.

Key words: inflammatory markers; females; striae distensae

Introduction:

Stretch marks, also known as Striae Distensae (SD), are a common dermatological disorder that is characterised by linear skin scars. The usual architecture of the skin is disrupted by dermal ripping and skin overstretching, which results in these scars. Depending on the developmental stage, Striae Distensae (SD) manifest as streaks that differ in colour, texture, and appearance. In their early stages, they frequently appear as reddish or purplish lines (striae rubrae), and as they mature, they change to a hypopigmented or white phase (striae albae). The abdomen, thighs, buttocks, breasts, and hips are among the body parts that are most commonly affected by SD because they are frequently stretched [1]. Despite not being fatal, the disorder can have a substantial negative influence on a person's quality of life, particularly for young adult females who may suffer from psychological anguish as a result of the aesthetic and cosmetic consequences [2].

Between 50 and 80 percent of people will experience SD at some point in their life, making it extremely common across a wide range of demographics. Due to physiological changes brought on by puberty, weight fluctuations, and pregnancy, young adult females are especially at risk. People experiencing quick growth spurts, substantial weight gain, or hormonal therapy are also commonly observed to have the syndrome. Genetic predisposition, hormonal changes, mechanical stress, and lifestyle variables are some of the risk factors that lead to the development of SD. The chance of getting SD is increased if there is a family history of the disorder, suggesting a possible genetic foundation for susceptibility [3]. Furthermore, increased oestrogen and corticosteroids during pregnancy and puberty hinder the production of collagen and elastin, which increases the likelihood of skin tears.

One of the main causes of SD is the mechanical stretching of the skin that occurs during pregnancy, muscle hypertrophy, or rapid weight increase. Scarring results from the overstretching's disruption of the skin's collagen and elastin network. Furthermore, a high body mass index (BMI), inadequate diet, and inadequate hydration are aggravating lifestyle factors. A comprehensive approach to SD, which is sometimes dismissed as a purely cosmetic issue but may have deeper physiological ramifications, is made easier by an understanding of these interrelated causes [4].

Mechanical, hormonal, and molecular disturbances are all part of the pathophysiology of SD. Tensile pressures that exceed the skin's capacity cause the dermis to weaken and elastic fibres to rupture, which is the main pathological alteration. The cells called fibroblasts, which produce collagen and elastin, are less active in skin afflicted by SD. The production of extracellular matrix proteins necessary for skin integrity is disrupted by hormonal changes, especially high cortisol levels, which also affect fibroblast function [5]. Inflammatory reactions cause erythema and vascular alterations in the early stages of SD. The mature SD appears atrophic and hypopigmented as the inflammation gradually goes down.

Inflammatory mediators play a critical role in the early stages of SD formation, according to recent developments in dermatological research. Immune cell infiltration, particularly that of macrophages and lymphocytes, has been noted in the dermal layers during the early rubrae phase. These immune cells release pro-inflammatory cytokines including interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α), which cause matrix metalloproteinases (MMPs) to break down the extracellular matrix. The skin structure is weakened by this enzymatic breakdown of collagen and elastin fibres, which also starts the development of noticeable stretch marks [6].

Additionally, it is becoming more widely acknowledged that C-reactive protein (CRP), a systemic acute-phase reactant produced in the liver in response to IL-6, may be a diagnostic of subclinical inflammation in skin conditions like SD. Particularly in inflammatory skin disorders, elevated CRP levels may be a reflection of systemic immune activation and ongoing tissue remodelling. Investigating these indicators presents a viable way to learn more about the pathophysiological underpinnings of SD and could aid in early detection and individualised treatment plans [7].

One of the most common dermatological disorders, especially in young adult females, is Striae distensae (SD). Even though SD is regarded as benign and non-life-threatening, those who are affected bear a significant psychological and cosmetic cost. It frequently results in negative body image, low self-esteem, and emotional suffering, particularly in a group that is already heavily influenced by society and culture to preserve physical attractiveness. However, the majority of current research focusses mostly on cosmetic treatment techniques rather than its biological basis, indicating that the scientific community is still struggling with an imperfect grasp of its pathophysiological roots [8].

Clinical observations clearly show that SD progresses from striae rubrae (early inflammatory phase) to striae albae (chronic scar phase) [9]. The precise cellular and molecular processes causing this evolution are still unknown, though. According to new research, fibroblast dysfunction, extracellular matrix breakdown, and local inflammation are key factors in the development of lesions [10]. Numerous inflammatory skin conditions have been linked to systemic indicators including C-reactive protein (CRP) and inflammatory cytokines like interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α). Nevertheless, little is known about their precise function and therapeutic importance in relation to striae distensae. [11]

Despite being medically innocuous, stretch marks, or striae distensae (SD), can be very problematic from a cosmetic and psychological standpoint, especially for young adult women. Even though it affects up to 90% of pregnant women and a significant portion of female adolescents and young adults, little is known about the condition's molecular and systemic causes.

Important gaps in our understanding of its pathogenesis, development, and possible systemic implications have been created by the overemphasis on cosmetic therapy at the expense of scientific research. Therefore, this study is warranted on a number of important clinical, scientific, psychological, and public health fronts [12].

Due to hormonal changes throughout puberty, weight fluctuations, genetic predispositions, and lifestyle variables, SD is very common in young adult females. Even though striae are frequently written off as a cosmetic annoyance, they can cause serious emotional anguish, anxiety, social shame, and low self-esteem. Especially for young women navigating formative educational, professional, and interpersonal situations, these psychological repercussions might impede social connections and quality of life. SD rarely receives clinical attention despite this burden unless patients actively seek dermatological advice. Examining the molecular causes of SD can improve clinical identification and de-stigmatization, redefining it as a disease deserving of study and treatment [13].

The precise pathophysiological mechanisms are still unclear, despite the fact that several histological studies have shown collagen disintegration and decreased elastin in SD lesions. Although not well investigated, the significance of inflammatory mediators such C-reactive protein (CRP), tumour necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) in skin remodelling and dermal alterations linked to SD has been hypothesised. By examining these indicators, the study hopes to shed light on the inflammatory aspect of SD and provide a possible molecular explanation for the clinically observed skin deterioration. Beyond topical cosmetics that may target inflammatory pathways, establishing a connection between inflammation and SD could direct future therapies [14].

The possible discovery of inflammatory biomarkers that could be used as prognostic, diagnostic, or treatment-monitoring instruments for SD provides a significant rationale for this investigation. Clinicians may be able to determine the risk of SD progression or evaluate disease activity if specific inflammatory markers or blood indices are regularly increased in afflicted individuals. Additionally, this would open the door for more individualised treatment plans that target patients according to their biomarker profiles rather than just their appearance.

Materials And Method:

Study Area

The area was Imo State University (IMSU) a public university located in Owerri, the capital city of Imo State, Nigeria.

Advocacy, Mobilization and Pre-survey Contact.

Informed consent was obtained from study participants who are eligible for the study. With the help of a structured questionnaire, data of individual participants were collected.

Study Population

The sample size for the study was calculated using the Fisher's formula below. The assumed prevalence rate of Striae Distensae in females in Imo State University is 3%.

Therefore, calculating the sample size using

$$n = \frac{Z^2 \cdot P\ (1-P)}{E^2}$$

Where :

- n = required sample size =?
- Z = Z-value corresponding to the desired confidence level = 1.96
- P = Expected prevalence or proportion 3% (P = 0.03)
- E = Margin of error 5% (E = 0.05)

$$n = \frac{(1.96)^2 \times (0.03) \times (1- 0.03)}{0.05^2}$$

$$n = \frac{3.8416 \times 0.03 \times 0.97}{0.0025} \qquad n = 44.7$$

Therefore, a total of 50 test subject would be used, and the same number of controls will be used also.

Subject Selection:

Inclusion Criteria:

1. Young adult females aged 18 to 35 years.
2. Participants with clinically visible and confirmed Striae Distensae (striae rubrae or striae albae).
3. Participants who provide informed consent and agree to comply with the study protocols.
4. Participants with no acute or chronic illnesses that might independently affect inflammatory markers and Hematological parameters.
5. Participants who are not pregnant at the time of the study.

Exclusion Criteria:

1. Participants below 18years and above 35years of age.
2. Individuals with systemic diseases or dermatological conditions such as diabetes mellitus, autoimmune disorders, eczema, or psoriasis.
3. Participants with a history of systemic or topical corticosteroid use within the past six months, as these may affect skin integrity and immune response.
4. Pregnant women or those who have given birth within the past year, as hormonal changes during pregnancy can independently influence SD development.
5. Individuals who have undergone recent surgical or cosmetic procedures targeting SD (e.g., laser treatments, microneedling).
6. Participants who are unwilling to follow the study protocol or withdraw consent at any stage.
7. Individuals with a history of smoking, alcohol dependence, or substance abuse, as these factors may confound the study results.

Study Design

This research was a comparative cross-sectional study aimed at evaluating and comparing specific inflammatory markers between young adult females with striae distensae and apparently healthy females without the condition. The target population included 50 females aged 18 to 35 years with clinically confirmed SD, alongside an age-matched control group of 50 females without SD for comparative purposes. Participants were recruited through outreach, and referrals, with eligibility assessed based on pre-defined inclusion and exclusion criteria.

Data Collection

Study Parameters

The parameters determined included; inflammatory markers (CRP, IL-6, TNF- α),

Sample Collection

Tourniquet was applied to upper forearm of the subjects after assuming a comfortable sitting position. The site chosen for vene-puncture was wiped with 70% alcohol for sterilization. 5 milliliters (7 ml) of blood was then collected. The 5ml was dispensed into the vaccutainer tube with minimal stasis. The tube was properly labeled with the subject's name, sample number and date of collection. The blood was allowed to clot at room temperature, and serum separated and harvested into clean dry well labelled sample bottles following centrifugation at 3000 rpm for 5 minutes. The sample was stored in a freezer at -20˚C.

Laboratory Procedures

All reagents were commercially prepared and the manufacturer's standard operating procedures (SOP) strictly adhered to.

The determination of C-reactive Protein Interleukin 6 (IL-6) and Tumour necrosis factor α (TNF – α) were done Using ELISA

Statistical Analysis

All statistical analysis was conducted using SPSS version 21.0, with a significance level set at $p<0.05$. Data was expressed as mean ± standard deviation. Comparison of groups was done using Student t-test at $p< 0.05$.

Results:

Results obtained was expressed in tables as illustrated below

Variables	Test Subject (N = 50)	Control Subject (N = 50)	t-Value	P-value
CRP (mg/L)	45.86 ± 0.85	31.78 ± 1.74	14.5692	0.000
IL-6 (pg/mL)	8.87 ± 0.15	6.72 ± 0.13	21.5498	0.000
TNF- α (pg/mL)	150.66 ± 2.84	121.69 ± 1.46	18.2558	0.000

Table 1: The *mean ± standard deviation values of inflammation makers in young adult females with striae distensae (Test Subjects) compared to controls*

Keywords:

P < 0.05 = Statistically Significant

P > 0.05 = Not Statistically Significant

CRP: C-reactive Protein

IL-6: Interleukin 6

TNF- α: Tumour necrosis factor α

Table 1 shows the mean values of inflammatory markers C-reactive protein (CRP), Interleukin-6 (IL-6), and Tumor Necrosis Factor-alpha (TNF-α) in patients with Striae Distensae (Test group) compared to the control group. The mean value of CRP in the test subjects was 45.86 ± 0.85 mg/L, which was significantly higher than that of the control subjects (31.78 ± 1.74 mg/L) at P < 0.05. Similarly, the mean value of IL-6 was significantly elevated in test subjects (8.87 ± 0.15 pg/mL) compared to controls (6.72 ± 0.13 pg/mL) at P < 0.05.

Furthermore, the mean value of TNF-α was also significantly increased in patients with Striae Distensae (150.66 ± 2.84 pg/mL) compared to the control group (121.69 ± 1.46 pg/mL) at P < 0.05.

Discussion:

In order to draw conclusions that may be pertinent to the pathophysiology of Striae Distensae (SD), this study sought to assess the levels of specific inflammatory markers in young adult females with the disease using a reference model: C-reactive protein (CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α) [15]. In order to provide light on the systemic effects of inflammation and its function in tissue remodelling and vascular changes—both of which are probably implicated in the development of SD—the study additionally evaluated the association between these indicators [16].

The results showed that SD patients had significantly higher levels of the inflammatory markers TNF-α, IL-6, and CRP than did healthy controls. One important acute-phase protein that is generated in response to cytokine stimulation, especially IL-6, is CRP. Chronic inflammatory diseases like SD are known to have elevated CRP levels [17]. Psoriasis and atopic dermatitis patients have also been found to have elevated CRP, indicating that systemic inflammation may affect the skin and may play a role in the extracellular matrix remodelling observed in SD [18].

Test respondents' levels of IL-6, a cytokine essential to both acute and chronic inflammatory responses, were noticeably greater than those of controls. This conclusion is consistent with that of [19], who demonstrated that IL-6 mediates chronic inflammation by preventing erythropoiesis and causing vascular dysfunction, in addition to inducing hepatic synthesis of acute-phase proteins. Additionally, the overexpression of IL-6 has been linked to the breakdown of dermal matrix proteins by inducing matrix metalloproteinases (MMPs), which are enzymes that break down collagen and elastin, a crucial aspect of the pathophysiology of [20].

The condition's inflammatory nature is further supported by test subjects' higher TNF-α levels as compared to controls. Strong pro-inflammatory cytokine TNF-α promotes a persistent inflammatory state that hinders tissue healing by inducing leukocyte recruitment and endothelial activation [21]. TNF-α also promotes fibroblast dysfunction and increases adhesion molecule expression. According to skin physiology, this might promote dermal tearing and collagen disruption, which would aid in the development of SD.

Our results are corroborated by a study by [22] that found that those with striae distensae had higher levels of serum pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α), suggesting an inflammatory

foundation for the disorder. [23,24]

Conclusion:

In a sample of individuals with persistent inflammation, this study has shown notable increases in inflammatory marker changes. Particularly including CRP, IL-6, and TNF-α, the cytokine dysregulation seen here is indicative of ongoing systemic inflammation and offers a tenable molecular explanation for dermal tissue remodelling, which is pertinent to SD.

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