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Assessment of inflammatory markers in Acne vulgaris patients: serum IL-17, IL22, and IL-10

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Abstract

One of the four main factors in the pathophysiology of acne vulgaris (AV) is inflammation, which may be a primary or secondary process caused by Propionibacterium acnes. To counteract inflammatory mediators, the immune system possesses a variety of anti-inflammatory mechanisms. The data supporting the early involvement of the inflammatory pathway in the etiology of AV, a chronic, complex, inflammatory skin condition, has become more clear. This cell line's activators, Th17 cells, and the cytokines that follow are all probably important in initiating and maintaining the disease. The aim of this study is to determine the degree to which acne vulgaris is influenced by the interleukins IL-10, IL-17, and IL-22. Individualized patients in our study, sex-matched controls were included, and there were 42 male and 58 female AV patients, aged 12 to 30. An enzyme-linked immunosorbent assay (ELISA) was used to measure the serum levels of IL-10, IL-17, and IL-22. The levels were then connected with the severity of acne. In cases, the serum levels of IL-10, IL-17, and IL-22 were 0.34±0.080, 0.13, 0.14±0.0035, 0.136-0.233, and 0.24±0.0142, 0.126-0.243 pg/ml, respectively, while in controls, they were 0.13±0.028, 0.10±0.0085, 0.14±0.0035, 0.117-0.126, and 0.22±0.0089, 0.114-0.127 pg/ml. For IL-10 and IL-17, there was a substantial difference in levels between patients and controls; however, for IL-22, the difference was negligible. The levels of IL-10 and IL-17 showed an extremely strong positive connection. This study came to the conclusion that IL-10 and IL-17 are important effectors in the pathophysiology of AV. In acne lesions, Th17 cells activated by IL-22 are probably the main source of IL-37.

Keywords: Acne vulgaris, IL-10**,** IL-17, IL-22

Introduction

Sever chronic inflammatory disease are accompanied by the appearance of symptoms of pilosebecous as in acne vulgaris and it is represented by non-inflamatory lesion (comedons black and white head) the symptoms of inflammatory lesions which is (papules, pustules and noudles) [1]. The final result was sebum production and the inflammatory process ans also the

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androgen excess states, the fatness and the multiplication of Propionibacterium acnes [2]. On the basis of severity, we will classify the Acni to the three type mild, moderate and severe that doing by the using the Global Acne Assessment Scale [3]. Seborrheic (oily) skin, which affects the face, neck, back, and chest, is where it mostly starts when we sew [4]. Immune system cells release cytokines, which are nonstructural, highly active, short soluble polypeptides (8–20 kilodalton). They mediate intricate cell interactions and are primarily produced by monocytes, macrophages, lymphocytes, granulocytes, epithelial cells, keratinocytes, and fibroblasts [5].

They have a limited range of function and are produced in little numbers in the normal state. Infection triggers cause them to create cytokines, which include natural inhibitors of these cytokines and anti-inflammatory cytokines. Thus, the most important cytokines are IL-10 and IL-17 [6]. Eleven cytokines were implicated in the interleukin- (IL) group. As well. It is also an important component of the immune system. One of the most well-known cytokines in the pathophysiology of acne is interleukin-10 (IL-10), which is also the most researched member of the group [7]. A naturally occurring receptor antagonist protein controls the biological activity of IL-10 [8]. The current study sought to measure the levels of IL-10 and IL-17 in the same patients in the province of Baghdad. Interleukin-22 (IL-22) is a cytokine that is functionally described by anti-inflammatory effects and, most importantly, in the immune response to infections; it is produced from helper cell type (Th 2), which inhibits cytokine production from Th 1 cells [9].

Materials and Methods

One hundred AV sufferers were enrolled in our study. The inclusion criteria included untreated (washout period for topical and systemic acne therapy: 2–4 weeks, respectively) cases of AV in individuals of either sex between the ages of 14 and 35. we carried out this study in the Al-Yarmouk Teaching Hospital in Baghdad between October 2021 and March of the 2022. In our earlier study, we categorized our patients into three groups: male, female and controls acne sufferers and Comparison theinterleukin (IL-10 and IL-17 and IL-22) with Mild, Moderate and Severe Acne vulgaris.

Twenty control subjects had five milliliters of venous blood drawn as a last resort with a sterile needle and aseptic technique on a minimum of 100 patients (39 men and 61 women) for blood testing. In this instance, three milliliters were immediately put into a sterile Gel tube, given time to settle, and the serum was then extracted using a 15-minute centrifugation at 4000 rpm. The serum was frozen and stock piling at -20°C. The interleukin (IL-10 and IL-17 and IL-22) grouping was measured in the 80 skin inflammation patients (35 men, 45 women), as well as the 20 controls (10 men, 10 women).

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Similar to serological assay, each type of interleukin was measured using an IL-10 and IL-17 and IL-22 focus kit, with the control set and chosen contaminated group using the kit with the original number 1. using a specific kit made for each interleukin that we required for use, in accordance with the (ELISA) protocols. Measurable analysis: information was shared through information investigations using ± SD. The associations between every group of patients with skin inflammation and a control group were then examined using a T-test and an automated Minitab program. The result of that correlation was explained by a variance analysis using the computer program Minitab 14 and the statistical tests Chi-square and Chances Proportion. (P< 0.01) was thought to be.

Results

Serum Interleukins (IL-10 and IL-17 and IL-22) fixation

There were 42 (42%) men and 58 (58%) women in the study group. There were 11 (34.32%) males and 19 (61.37%) females in the control group. According to the distribution of age and gender, the groups were comparable (P values of 0.898 and 0.875, respectively). The sickness lasted an average of 7.18 years, with a range of 12 to 15 years. While 33 (53.57%) and 6 (9.23%) patients had moderate and severe acne, respectively, 25 (41%) individuals had mild acne. The male and female cases had significantly higher serum levels of IL-10 were (0.34±0.080, 0.13 pg/ml) and IL-17 were (0.18±0.0085, 0.136-0.233 pg/ml) than the controls for IL-10 were (0.13±0.028, 0.10±0.0085 pg/ml) and and IL-17 were (0.14±0.0035, 0.117-0.126 pg/ml) as well the ***P. Value*** (P < 0.0001) respectively, while the cases had insignificantly higher levels of IL-22 were (0.15 ± 0.0174 and 0.23 ± 0.0152 pg/ml) than the control (0.13 ± 0.0095, 0.21 ± 0.0099 pg/ml). Additionally, there was a noteworthy positive connection between rising acne severity and IL-10 levels [Table 2]. Additionally, a substantial positive connection (P < 0.0001) with IL-17 was demonstrated.

Table .1. Serum (IL-1α) concentration (pg/ml) of patient groups compared to control group examination.

Parametars		Sample size	**Mean ± SD**	Median	Min-max	***P.Value***
IL- 10	Male control	10	0.13±0.028	0.08	0.07-0.79	<0.0001
	Male patient	35	0.34±0.080	0.12	0.09-2.89	
	Female control	10	0.10±0.0085	0.14±0.0184	0.05-0.26	
	Female patient	45	0.13	0.13	0.07-0.84	

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Table. 2. Correlation of serum(IL-17) focus (pg/ml) of the patient gatherings with control group.

Parametars		Sample size	**Mean ± SD**	Median	Min-max	***P.Value***
IL- 17	Male control	10	0.14±0.0035	0.12	0.09-0.19	<0.0001
	Male patient	35	0.18±0.0085	0.18	0.12-0.37	
	Female control	10	0.117-0.126	0.13±0.0174	0.15-0.62	
	Female patient	45	0.136-0.233	0.14	0.17-0.8	

Table. 3. Correlation of serum(IL-17) focus (pg/ml) of the patient gatherings with control group.

Parametars		Sample size	Mean ± SD	Median	Min-max	*P.Value*
IL- 22	Male control	10	0.22±0.0089	0.15	0.15-0.5	0.706
	Male patient	35	0.24±0.0142	0.17	0.14-0.92	
	Female control	10	0.114-0.127	0.12±0.0184	0.13-0.72	
	Female patient	45	0.126-0.243	0.13	0.18-0.91	

Table. 4. Comparison between serum interleukin (IL-10) Levels of acne vulgaris severity

IL- 10						
group of AV	Sample size	Mean ± SD	Median	Min-max	P	R
Mild	29	0.12±0.0284	0.11	0.07-0.69	0.046	0.338
Moderate	41	0.15±0.0229	0.14	0.08-0.73		
Severe	10	0.17±0.0467	0.13	0.11-0.45		

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Table. 5. Comparison between serum interleukin (IL-10) Levels of acne vulgaris severity

IL- 17						
group of AV	Sample size	Mean ± SD	Median	Min-max	P	R
Mild	29	0.18±0.0135	0.17	0.14-0.34		
Moderate	41	0.19±0.0117	0.18	0.12-0.35	0.702	0.017
Severe	10	0.2±0.0216	0.22	0.14-0.26		

Table. 6. Comparison between serum interleukin (IL-10) Levels of acne vulgaris severity

IL- 22						
group of AV	Sample size	Mean ± SD	Median	Min-max	P	R
Mild	29	0.22±0.0163	0.18	0.15-0.39		
Moderate	41	0.23±0.0276	0.17	0.17-0.92	0.585	0.034
Severe	10	0.21±0.0195	0.16	0.18-0.37		

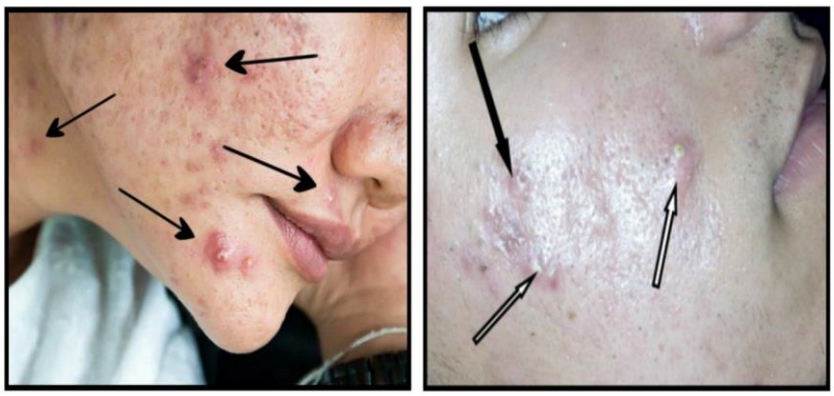

Figure .1. Comedones (white bolts) and pustule (dark bolt) facial skin in the patient with .Skin inflammation sores

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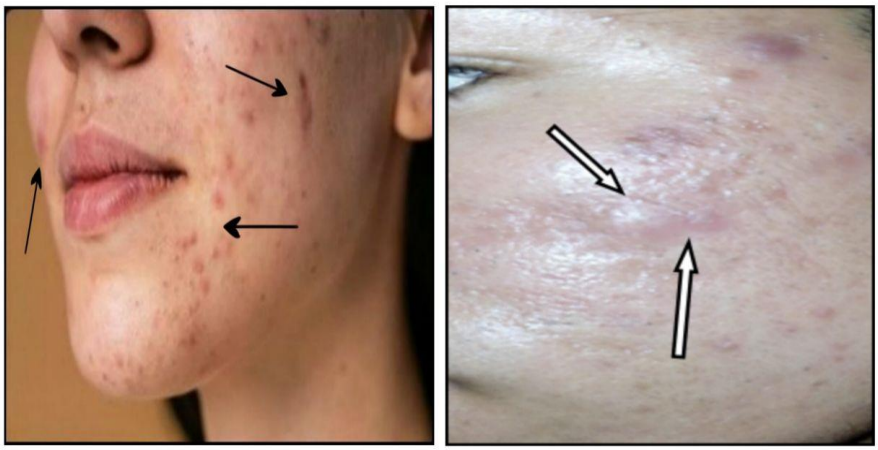

Figure .2. Papule on the facial skin in patient with Acne lesions.

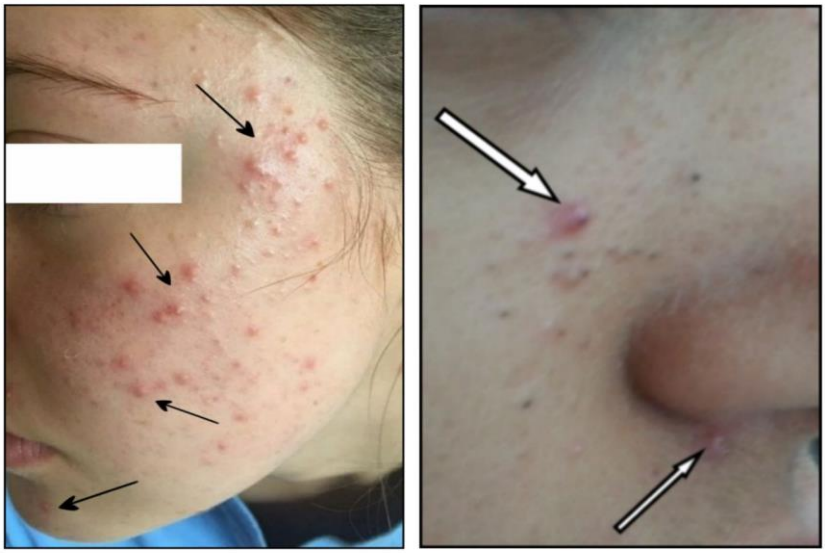

Figure .3. Comedones (clogged pores) on the facial skin in quiet with Skin break out sore

Discussion

It is still unknown what exactly happened in the etiopathogenesis of AV. Keratinocytes and sebocytes function as immune cells in the immunocompetent pilosebaceous unit [10]. Both pathogen recognition and the production of abnormal lipids are capabilities of these cells. According to earlier research, IL-1α is the primary cytokine linked to the pathophysiology of AV, primarily in the synthesis of microcomedones [11].

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E-selectin, vascular cell adhesion molecules 1 (VCAM-1), and intercellular cell adhesion molecules-1 (ICAM-1) are all expressed more when IL-10 is present. It also encourages keratinocyte proliferation in the pilosebaceous unit [12].

According to available data, inflammation begins early in the pathophysiology of acne and is primarily brought on by keratinocytes and sebocytes' identification of C. acnes via TLR-2 [13]. Moreover, even before follicular hyper-cornification and comedone development, CD4, CD68, T cells and macrophages have been seen in the location of early lesions [14]. Our results demonstrated that acne sufferers had considerably higher levels of IL-10 and IL-17 than controls. Numerous cells, including monocytes, macrophages, and keratinocytes, emit IL-8, a strong neutrophil chemotactic factor [15].

Inflammatory lesions are created when neutrophils degranulate at the site of inflammation, releasing reactive oxygen species (ROS) and proteolytic enzymes [16]. Acne pustular lesions contain a large number of neutrophils, and the quantity of neutrophils is correlated with the levels severity of acne [17]. When C. acnes activates monocytes through TLR-2, IL-8 is also generated in addition to IL-1β and TNF-α. [18]. According to Kim et al., TLR-2 proteins were increased as acne lesions grew, which therefore raised IL-8 levels.

Higher levels of circulating IL-10 and more severe acne have been linked to polymorphisms in the IL-10-251T > A gene. Consistent with our results, (12 and 13) found that the severity of acne was correlated with an increase in IL-10 levels [19]. Additionally, we discovered a good correlation between the acne groups and the serum level of IL-10 (R was 0.257 and P was 0.036). Additionally, Sahib et al. [20] found that acne patients had higher levels of IL-8 than healthy people. Furthermore, Stankowska et al. assessed serum IL-17 and IL-22 levels in acne patients and controls [21]. They found that while IL-6 levels were higher in acne patients, IL-8 levels were the same in both groups, and that IL-10 levels increase in proportion to the severity of the disease. We found a strong positive correlation between the two cytokines, indicating that Th17 cells may be the primary producers of this cytokine in acne. Another element that encourages neutrophil recruitment and the ensuing inflammation in acne is Th17 cells. According to recent studies, this cell line contributes to the pathogenesis of acne.

Through the action of TGF-β, IL-10, IL-1β, and IL-22, it has been demonstrated that C. acnes causes naïve CD4+ cells to differentiate into Th17 cells [22]. It has also been demonstrated that cytokines including IL-1β and IL-6, which trigger the Th17 pathway, are present in acne lesions. Cytokines such as IL-17A, IL-17F, and IL-22 are produced by stimulated Th17 cells [22]. The primary cytokines that attract and activate neutrophils are IL-17A and IL-17F, but they can also target fibroblasts, monocytes, endothelial cells, and keratinocytes. Furthermore, they can trigger the production of pro-inflammatory mediators such as matrix metalloproteinases, IL-10, TNF, IL-1β, and PGE2, which may worsen lesional sites [20]. Previous research found that the severity of acne was positively correlated with IL-17 levels in serum and tissue [19]. Even while we did not find a significant change in the cytokine with increasing acne severity, our serum level of IL-17 was considerably higher in cases than in controls, which is consistent with previous studies (R = 0.034, P = 0.585). Additionally,

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contrary to a number of previous in vitro studies, we did not detect higher levels of IL-22 in patients as compared to controls [23].

Conclusions

The most frequent age group of Acne patients was 14-35 years old. This study supports the role of the IL-10, IL-17 and IL-22 but has no involvement in the incidence of Acne in baghdad province. The high rate of IL-10 and IL-17 fixation in patients with Skin break out contrasted with the control group. This alludes to its part in skin break-out injuries. By contrast, IL-10 and IL-17 levels were significantly different between patients and controls, but not IL-22. The levels of IL-10 and IL-17 showed an extremely strong positive connection. The degree of illness was significantly positively correlated with IL-22. IL-10 and IL-17 are important effectors in the pathophysiology of AV. In acne lesions, Th17 cells activated by IL-22 are probably the main source of IL-8.

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