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Estimation of IL-17A and IFN-y in the Burn of Patients that Afflicted by Different Bacterial Types

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Abstract--- *The objective from this study was to detected the level of IL-17A and IFN-y in the burn patients that afflicted by different types of bacteria compare with the control group of patients that not afflicted by bacteria. The samples collection was carried out from 15/10/2017 to 22/1/2018 at the burns hospital in Baghdad. Samples were collected from patients sera. collection samples were from (88) burn patients, 58 of them that infected by deferent types of bacteria, 29 patients were males, and 29 patients were females. The other 30 patients that not infected by bacteria, 15 patients were males, and 15 patients were females, this group used as control group. The investigation of the level of the IL-17A and IFN-y were carried out by ELISA assay in the teaching Laboratories in Baghdad. The results showed decrease in the level of IL-17A in the patients were 43.43 pg+5.426 compare to the control group were 55.77pg +5.418. and its showed decrease in IFN-y levels of patients were 36.95pg+5.808 compare to the control group were 46.63pg+5.816. The current study reported highly significant difference of IL-17A and IFN-y levels between the patients and control group at the level (P < 0.01).*

Keywords--- *Burn Patients, Interleukin – 17 and Interferon – Gamma.*

I. INTRODUCTION

Burns are one of the major common destruct types of trauma (1).

Burns disturb skin integrity and skin immune system that protective against the activity of pathogenic organisms (2).

The majar challenge for burn patients is nosocomial infection (3). Infection is important cause of morbidity and mortality hospitalized burn patients (4).

Nosocomial infection in the burns is higher due to immunocompomised status and nature of injury (5). The immunological organization of the body controlled by numerous parameters, and any change in the normal regulation of immune coordination may lead to diseases (6).

The immune response toward the infection in both lines the first and second are inhibition. Skin becomes damage, allowing invasion of pathogen, the damaged skin and exudates of patients create the conditions for microbial growth (7).

The pathophysiology of injury burns with its major immune alteration and metabolic status (8).

The current evidence suggests that the T-helper (Th17) lymphocytes is important in the regulation of the immunity of mucosal surface. Th17 help to maintain immune homeostasis (9). The effecter of T-cells were

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described: inflammatory cells (Th_1) which produce interleukin –2 (IL-2), interferon gamma (IFN-y) and other cytokines (10).

The named of interleukin was in (1979) to replace different various names (T-cell replacing factor III, lymphocyte activating factor...etc.) (11).

Interleukin-17A is inflammatory cytokine produced by activated T-cells. Molecules newly discovered that associated to IL-17A are forming a group of cytokines. IL-17A group protype member has nominated IL-17A. (12). have documented that five members were described and cloned due to advances in the genome of human sequencing and proteomes: (IL-17F, IL-17B, IL-17D, IL-17C, IL-17E).

IL-17A produces by Th17 cells recruits neutrophils into infected tissue and increase the antimicrobial function of neutrophils (13).

Also IL-17A stimulates of endothelial, epithelial cells, and macrophage cells to produce chemokines and proin flammatory cytokines, which are factors induce " //systemic inflammatory response syndrome" (14).

The IL-17A secretion by (CD_4^+) Th17 cells and its specific functions are stimulating the epithelial, endothelial cells, fibroblastic and macrophage cells to generate (IL-6, IL-8, G-CSF, M-CSF and I – COM_1) (15).

These soluble factors contributing to local rooting of cells that infected because of antimicrobial peptide that produce by endothelial cells stimulated by IL-17A (16).

IL-17A billons to the family of IL-17 in which IL-17F has analogous functions. IL-17A bind to the receptor of IL-17A: IL-17RA and IL-17RC of human (17).

(18). reported that Interferon –gamma (IFN-y) are proteins produced by the vertebral cells as reaction to pathogenic agents such as bacteria, tumor cells, viruses and parasites.

IFN- y is critical cytokine for adaptive and innate immunity against intracellular bacteria. IFN-y promotes T and B cells differentiation, stimulates cytotoxic T-cell and activates microbicidal function of macrophages (19).

Interferons were describe first in (1957). The discover was a result of works on the interference of viral, interference refers to inhibition growth of virus(20). IFN-y is adimenized soluble factor it’s a number of type II classes of interferons. Interferons were named for their ability to interference with replication of viral (21).

IFN-y produced by T-cell, NK cell and its functions are: antiviral, immunregulatory, augment macrophage activation, MHC class I,II and anti proliferative (22). Cellular response to IFN-y is stimulates by its interaction with ahetro dimeric receptor. Receptors are: $IFNGR_1$, $IFNGR_2$ (23).

IFN-y production occurs in a response to microbes such as: bacteria and viruses and their products: viral glycoprotein, viral RNA, endotoxin of bacteria, bacterial flagella. Also IFN-y production induced by: Mitogen and cytokines [IL-2, IL-1, IL-12,TNF] (24).

Present study aimed to evaluating the IL-17A and IFN-y in the burn patients compare to the control group.

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II. MATERIALS AND METHODS

The present work includes the collection of serum from (88) burn patients, (58) patients were infected by deferent types of bacteria, 29 patients were males and 29 patients were females the others 30 patient were not

Used the patients that not infected by bacteria as a control group. The collection of samples were from burns hospital in Baghdad from the period 15/10/2017 to 22/1/2018. This samples used to investigation the IL-17A and IFN-y in the patients and control group. The sera were collected brought to the teaching laboratories in Baghdad were tested. In this study used the IL-17A and IFN-y kits to made the assay. The company that accoutered the kits was cusabio in China.

The assay performed by ELISA instrument.

Methods:

Note (the same method of assay to the IL-17A and IFN-y)

1. Biotin antibody (1X) vial should be centrifuged before opening. Biotin antibody requires a 100 fold dilution was 10 micro letter of biotin antibody added to 990 micro letter of Biotin –antibody Diluent.
2. HRP- avidin (1X) the vial shoud be centrifuged before opening. HRP- avidin requires a 100 double dilution was 10 micro letter of HRP- avidin added to 990 micro letter 0f HRP-avid in diluent.
3. Wash Buffer (1X) must warm up to temperature of room and blend kindly until the crystals are absolutely dissolved. Diluted 20 ml of wash buffer (25 X) with 500 ml of distilled water.
4. Standard: the standard vial was centrifuged at 600 rpm for 30 second. dilute the standard with 1 ml of sample diluent. The product is formed a stock solution of 400 pg/ml. Pipette 250 micro letter of sample diluent into each tube (S0 – S6).Used the stock solution to produced a 2- double dilution sequence.

The dilution standard represent the high standard 400 pg/ml. Sample diluent stand for the zero standard 0 pg/ml

III. RESULTS

Table 1: Shows mean, standard deviation and T-test of IL-17A in the patients and control group

	groups	number	mean pg/ml	standard deviation	T-test	P-value	denotation
IL-17A	patients	58	43.43	5.426	10.114	(P<0.01)	highly significant Denotation
	control	30	55.77	5.418			

Table (1) showed the mean, standard deviation and T-test for IL-17A in the patients and control group sera.

In this table notice the decrease of the level of IL-17A in the patients sera were 43.43 pg+5.426. Compared with the control group sea were 55.77 pg+5.418, and notice highly significant deference between the two groups in the level (p<0.01).

Table 2: Mean, standard deviation and T-test of IFN-y in patients and control group

	groups	number	mean pg/ml	Standard deviation	T-test	P-value	denotation
INF- y	patients	58	36.95	5.808	7.411	(P<0.01)	Highly significant denotation
	control	30	46.63	5.816			

Table (2) showed the mean, standard deviation and T-test for IFN- y in the patients and control groups sera.

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In this table notice the decrease of the level of IFN- y in the patients sera, were 36.95 pg+5.808 compared with the control group sera, were 46.63 pg+5.816, and notice highly significant difference between the two groups in the level ($P<0.01$).

IV. DISCUSSION

Burns disturb skin integrity and skin immune system. The challenge for burn patients is nosocomial infection. Infections is an vital source of morbidity and mortality in the burns team.

The immunological response are inhibition in the burn patients. IL-17A proinflammatory interleukin produces by activated T-cells. In the current study notice the decrease of IL-17A for patients compared with the control group. This decrease may results from immunosuppression of the burn patients that afflicted by different types of bacteria. The suppression of immune system included the inhibition of Th17 cells which secretion of IL-17A (25).

The current study agree with (13). That reported the decrease of IL-17 in the burn patients, and agree with (26).

That reported the suppression of T-cells in the burn and septic injures. The current study disagree with (27). That reported the elevation of IL-17A in the sepsis patients.

IFN- y are proteins produced by the most cells of vertebrates. IFN- y is critical cytokine for adaptive and innate immunity. In the current study notice the decrease of the level of IFN- y in the patients compared with the control group. This decrease may result as a suppression of immune system in burn patients that afflicted by different bacterial types.

The immuneosuppression include the inhibition of NK cells that produced IFN- y (28). The current study agree with (29) that reported the decrease in IFN- y in the burn patients, and agree with (30). That reported immunedysfunction and the decrease of IFN- y burn patients. The current study disagree with (31). That reported no significant difference between burn patients and control group.

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