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Relationship between Pro-Inflammatory Cytokines and Growth Factors in Patients with Diabetic Foot Ulcers

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ABSTRACT

Aims Several factors are involved in the healing process of diabetic foot ulcers. Thus, the present study was conducted to evaluate the relationship between pro-inflammatory cytokines and growth factors in patients with diabetic foot ulcers.

Instrument & Methods This study was conducted on three groups of subjects. The first group was men and women (n=30) with diabetes and diabetic foot ulcers referring clinical centers in Iran. Another group was including diabetic women and men patients (n=30) without ulcers. The third group included healthy men and women (n=30). Blood samples were collected and assessed for the serum concentrations of TNF-α, IL-1β, IL-6, bFGF, IFN-γ, selenoprotein, PDGF, VEGF and GM-CSF with the help of specific kits.

Findings The results showed significant differences between groups for the serum concentrations of TNF-α (p=0.001), IL-1β (p=0.001), IL-6 (p=0.001), bFGF (p=0.001), IFN-γ (p=0.001), selenoprotein (p=0.001), PDGF (p=0.001), VEGF (p=0.001) and GM-CSF (p=0.001). The results showed the serum concentrations of TNF-α, IL-1β, IL-6, IFN-γ, PDGF, and GM-CSF were significantly higher in the patients with diabetes and foot ulcers, diabetes, and healthy subjects, respectively. The results showed a positive correlation between pro-inflammatory cytokines, while a negative correlation was observed between pro-inflammatory cytokines and other variables.

Conclusion Pro-inflammatory and inflammatory factors are higher in diabetic patients, especially those with diabetic ulcers. The concentration of factors can be considered for the treatment and as markers for the treatment of foot ulcers.

Keywords Diabetes; Ulcer; Inflammation; Cytokines

CITATION LINKS

[1] Accelerative effect of nanohydrogels based on chitosan/ZnO incorporated... [2] Biological fabrication and electrostatic attractions of new layered... [3] Fabrication, characterization and application of novel nanoemulsion polyvinyl alcohol/chitosan... [4] Fabrication of novel polysaccharide hybrid nanoliposomes containing citral for... [5] Mechanisms of obesity-and diabetes mellitus-related pancreatic carcinogenesis... [6] The impact of metabolic syndrome and type 2 diabetes mellitus on... [7] Predictors of lower extremity amputation in patients with diabetic foot ulcer: Findings... [8] Contribution of platelets, the coagulation and fibrinolytic systems to... [9] Diabetic wound healing in soft... [10] Evolving spectrum of diabetic wound: Mechanistic... [11] Prolonged growth hormone/insulin/insulin-like growth factor... [12] Effectiveness of topical caraway essential oil loaded into... [13] Chronic hyperglycemia mediated physiological alteration and... [14] Association of glycemic indices (hyperglycemia, glucose variability, and hypoglycemia)... [15] Pathophysiology diabetic foot... [16] Chronic non-healing diabetic foot ulcer treated by... [17] In situ sprayed NIR-responsive, analgesic black... [18] Diabetic foot infections... [19] Interleukins (from IL-1 to IL-38), interferons... [20] Curcumin accelerates cutaneous wound healing via... [21] Cytokines, chemokines and growth factors in... [22] Mediators of neuropathic pain; focus on... [23] Epigallocatechin-3-gallate suppresses the global... [24] The systemic-level repercussions of cancer-associated inflammation mediators produced in... [25] Inflammation and atherosclerosis: a review of the role of interleukin-6 in the... [26] Regulation of immune cells in atherosclerosis and its... [27] Regulation of interferon-γ during innate and adaptive... [28] VEGF and bFGF induction by nitric oxide is associated with... [29] The roles of vascular endothelial growth factor... [30] The role of vascular endothelial growth factor... [31] VEGF, PF4 and PDGF are elevated in platelets of colorectal... [32] ELISA for quantification of granulocyte macrophage-colony stimulating factor...

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Introduction

A wound is defined as a disruption in the defense function of the skin [1, 2]. It is also defined as the loss of the integrity of the covering tissue, which can be accompanied by or without damage to the connective tissue, resulting from physical or thermal damage to the skin [3, 4]. Diabetes is a type of metabolic disease related to the body's metabolism. It is one of the most common glandular diseases associated with high blood glucose levels caused by defects in insulin secretion, function, or both [5, 6].

High blood glucose levels (hyperglycemia) can lead to serious health problems. It causes ulcers in diabetic patients. About 25% of people who suffer from diabetes show foot ulcers during their lifetime. Ulcers and other complications are responsible for the hospitalization of 20% of the approximately three million people who are referred to hospitals for diabetes treatment every year [7]. Diabetes delays cell infiltration, angiogenesis, coagulation, and wound closure [8]. The increase in blood glucose causes an increase in oxidative stress and a decrease in the expression of insulin-like growth factor-1, which in turn affects the proliferation of fibroblasts and keratinocytes and the regeneration of epithelial tissue and the wound healing process [9, 10]. The decrease in endothelial insulin/insulin-like growth factor-1 signaling is a key factor that delays the wound healing process [10, 11]. Chronic diabetic wounds are closely related to the state of permanent inflammation, increased pro-inflammatory cytokines, and defects in the expression of growth factors [12]. Indeed, in diabetic patients, due to impaired insulin secretion, the cells of the body are not able to use the glucose inside the blood vessels in such a way that the inside of the cells is free of glucose and glucosemia, while the amount of glucose in the blood vessels and capillaries is high [13, 14]. Over time, high glucose can cause damage in the capillaries and nerves in such a way that improper blood supply and nerve damage in the lower limbs make them prone to diabetic ulcers [15]. One of the first symptoms of diabetic foot ulcers is abnormal inflammation, burning, redness, and bad smell [16]. Black tissue (dark) around the wound, which is caused by insufficient blood supply in the area, is one of the visible signs of diabetic foot ulcer, which indicates the death of cellular tissue around the wound due to infection [17]. Diabetic foot ulcer symptoms are not always visible. Sometimes the ulcer is not visible until an infection occurs [13, 18].

Several factors are involved in the healing process of diabetic foot ulcers. In the recent study, we aimed to investigate pro-inflammatory cytokines and growth factors in patients with diabetic foot ulcers. Thus, the present study was conducted to evaluate the relationship between pro-inflammatory cytokines and growth factors in patients with diabetic foot ulcers.

Instrument and Methods

This cross-sectional study was conducted on diabetic patients referred to Iranian clinical centers in Tehran from January to December, 2022. 90 samples including men and women were selected and divided into three groups of 30 samples including diabetic patients with foot ulcers (Ulc), diabetic patients without foot ulcers (Diab), healthy control (Cont).

Blood samples were collected and assessed using specific kits for the serum concentrations of TNF-α, IL-1β, IL-6, bFGF, IFN-γ, selenoprotein, PDGF, VEGF, and GM-CSF. The levels of these biomarkers were evaluated using ELISA kits (Hangzhou Eastbiopharm Co., LTP) according to the manufacturer's recommendations. All the subjects signed written consent forms and were aware of the details of the current study. An equal ratio of both genders (15 men and 15 women) was selected in each group with equal ratios for age, BMI, etc.

The data were analyzed for normality, and they were normal. Since ANOVA was used to analyze them. Pearson correlation was used to investigate the correlation between variables.

Findings

The mean age was 41.23±7.85 years in Ulc, 44.56±10.11 years in Diab, and 42.10±12.10 years in Cont groups (p>0.05).

Table 1. Comparing the mean serum concentrations of studied factors between the Ulc (n=30), Diab (n=30), and Cont (n=30) groups (all significant at 0.001)

Factors	Ulc	Diab	Cont
TNF-α (ng/mL)	7.21±1.23	4.23±1.23	1.56±0.23
IL-1β (ng/mL)	17.23±3.21	12.35±1.56	4.23±0.69
IL-6 (ng/mL)	10.25±1.26	6.78±0.78	2.36±0.57
bFGF-2 (pg/mL)	221.2±15.6	251.8±15.2	321.2±14.6
VEGF (ng/mL)	0.56±0.21	1.21±0.23	3.21±0.21
IFN-γ (pg/mL)	3.21±0.23	1.89±0.42	0.45±0.21
Selenoprotein (µg/mL)	2.21±0.25	2.89±0.12	5.12±0.21
PDGF (ng/mL)	7.21±1.25	5.63±0.21	2.54±0.15
GM-CSF (pg/mL)	2.85±0.31	1.75±0.25	0.95±0.25

There were significant differences between groups for the serum concentrations of TNF-α (p=0.001), IL-1β (p=0.001), IL-6 (p=0.001), bFGF (p=0.001), IFN-γ (p=0.001), selenoprotein (p=0.001), PDGF (p=0.001), VEGF (p=0.001) and GM-CSF (p=0.001). The results showed the serum concentrations of TNF-α, IL-1β, IL-6, IFN-γ, PDGF, and GM-CSF were significantly higher in the patients of Ulc, Diab, and Cont groups, respectively. The results also showed that serum concentrations of bFGF, selenoprotein, and VEGF were significantly higher in Cont, Diab, and Ulc groups, respectively.

There were no significant correlations between the studied factors in Cont group members (Table 2), but there were significant correlations between all factors in Diab group members (Table 3) and Ulc group members (Table 4) at p<0.0001.

Table 2. The correlations between the Cont group subjects (all insignificant).

Factor	9	8	7	6	5	4	3	2	1
1- TNF-α	0.058	0.052	0.065	0.057	0.052	0.045	0.078	0.089	1
2- IL-1β	0.075	0.081	0.063	0.025	0.101	0.081	0.58	1	
3- IL-6	0.041	0.069	0.057	0.052	0.061	0.032	1		
4- bFGF-2	0.063	0.052	0.048	0.063	0.025	1			
5- VEGF	0.041	0.032	0.026	0.081	1				
6- IFN-γ	0.056	0.032	0.032	1					
7- Selenoprotein	0.41	0.063	1						
8- PDGF	0.052	1							
9- GM-CSF	1								

Table 3. The correlations between the Diab group subjects (all significant at 0.0001).

Factor	9	8	7	6	5	4	3	2	1
1- TNF-α	0.412	-0.351	-0.441	0.551	-0.412	-0.341	0.354	0.356	1
2- IL-1β	0.362	-0.341	-0.365	0.375	-0.404	-0.331	0.312	1	
3- IL-6	0.381	-0.471	-0.414	0.632	-0.512	-0.44	1		
4- bFGF-2	-0.301	0.415	0.514	-0.412	0.512	1			
5- VEGF	-0.301	0.415	0.514	-0.412	1				
6- IFN-γ	0.389	-0.581	-0.412	1					
7- Selenoprotein	0.313	0.321	1						
8- PDGF	0.351	1							
9- GM-CSF	1								

Table 4. The correlations between the Ulc group subjects (all significant at 0.0001).

Factor	9	8	7	6	5	4	3	2	1
1- TNF-α	0.325	-0.326	-0.402	0.365	-0.521	-0.414	0.368	0.412	1
2- IL-1β	0.312	-0.371	-0.412	0.369	-0.415	-0.321	0.341	1	
3- IL-6	0.369	-0.441	-0.512	0.412	-0.369	-0.442	1		
4- bFGF-2	-0.369	0.442	0.441	-0.369	0.371	1			
5- VEGF	-0.371	0.443	0.552	-0.441	1				
6- IFN-γ	0.341	-0.512	-0.369	1					
7- Selenoprotein	0.321	0.341	1						
8- PDGF	0.341	1							
9- GM-CSF	1								

Discussion

This study was conducted to evaluate the relationship between pro-inflammatory cytokines and growth factors in patients with diabetic foot ulcers. The results showed a higher concentration of TNF-α in those with diabetes and foot ulcers. It shows that foot ulcers provoke TNF more than diabetes. TNF-α is a pleiotropic cytokine produced by cells such as keratinocytes, macrophages, and mast cells [19]. This factor acts in mechanisms such as the use of leukocytes, mainly neutrophils, the control of molecular adhesion, the production of chemokines and matrix metalloproteinases, and also as an inhibitor of metalloproteinases. TNF-α plays a beneficial role in the wound healing process and shows its mechanism by reducing the production of granulation tissue, but reducing the expression and concentration of this factor plays an important role in the production of collagen [20]. This factor regulates the activity of some fibroblasts, keratinocytes, and vascular endothelial cells and plays an important role in the production of metalloproteinases [21]. Thus, TNF increases in the response to the inflammation and exhibits its responses.

Similar to the results for TNF-α, IL-1β concentration was significantly increased in response to ulcers and diabetes. It was significantly increased in diabetic patients with ulcer feet. Interleukin-1 beta is secreted from keratinocytes, fibroblasts, endothelial cells, the nervous system, immune cells such as macrophages and mast cells, and glial cells such as Schwann cells, microglia, and astrocytes [22]. The expression of interleukin-1beta in the wound area is related to the phenotype of pro-inflammatory macrophages and inhibits the interleukin-1beta pathway in the wound area [23]. Interleukin-1 causes various activities such as neurological, hematological, endocrinological, and metabolic system changes and has various effects on wound healing [24]. First, interleukin-1 induces capillary endothelial cells to produce some chemokines, such as MCP-1 and increases the synthesis of vascular adhesion molecules [24]. The activities cause mononuclear cells to seep into the injury points and control inflammatory responses. The increase of the factors is a result of the response to an ulcer. Seemingly, ulcers and diabetes cause major responses.

The results also showed that IL-6 concentration was significantly higher in those with diabetes ulcers, diabetic patients, and healthy subjects. Interleukin-6 plays an essential role in the inflammatory process, especially in the preliminary phase of inflammation [25]. In laboratory studies, it was shown that interleukin-6 has no chemotactic activity for

leukocytes, but mice lacking interleukin-6 have lower leukocyte infiltration. These mice showed lower fibrotic changes in liver fibrosis. Interleukin-6 may control the use of leukocytes in inflammatory points and fibrotic changes [26]. The increase of IL-6 in patients with diabetes could be attributed to disorders in metabolic responses. Excessive increase in diabetic patients with foot ulcers could be attributed to severe inflammation in the foot.

The inflammatory factor was IFN-γ. Interferon-gamma is a type II interferon. This interferon plays an essential role in innate immunity and adaptive immunity against viruses, some bacteria, and protozoa. It stimulates macrophages and the expression of major histocompatibility complex class II [27]. Thus, it also has a major role in immune responses, and its concentration increases in the response to ulcer inflammation.

The results showed a decrease in the concentration bFGF-2 in patients compared with healthy subjects. This factor's efficiency is known in the regenerated epidermis, newly created capillaries, and cells seeping into the flesh bud tissue. This factor, along with vascular endothelial growth factor, induces angiogenesis, supports cell nutrition, provides oxygen, and helps provide energy [28]. In inpatient subjects, this factor is degraded and/or consumed. Thus, its serum concentration is lower in patients compared with healthy subjects.

The results also showed a decrease in the concentration of VEGF in healthy subjects. Vascular endothelial growth factor is one of the desired genes that intervenes in the wound healing process and acts as an endothelial cell mitogen and chemotactic [29]. The mechanism of this factor is not only by stimulating angiogenesis but also by increasing the permeability of the vessel and facilitating wound healing. VEGF is a cytokine that is responsible for inducing angiogenesis, cell migration, proliferation, and synthesis of extracellular fluid proteins [30]. It was expected to be higher VEGF in diabetic patients and those with foot ulcers. However, the results showed lower concentrations in the patients. It could be attributed to selenoprotein concentration. Selenoprotein protects endothelial cells, and the decrease in selenoprotein decreases the concentration of VEGF.

The serum concentration of PDGF was significantly higher in patients compared with healthy subjects. Platelet-derived growth factor (PDGF) constitutes a family of dimeric isoforms that act on connective tissue cells and certain other cell types. PDGF was originally discovered as a constituent of platelets released into serum in conjunction with blood coagulation [31]. It shows that PDGF concentration increases in response to metabolic disorders of diabetes and also damages patients with foot ulcers.

GM-CSF was significantly higher in patients compared with those in the healthy group. It is a monomeric glycoprotein secreted by macrophages, T cells, mast cells, natural killer cells, endothelial cells, and fibroblasts and acts as a cytokine [32]. Thus, GM-CSF works parallel with pro-inflammatory cytokines and increases inflammatory responses.

The results showed a positive correlation between inflammatory cytokines. It is natural that inflammatory factors work in the same way and increase inflammatory responses. In addition, the increase in inflammatory responses modulates the expression and concentration of growth factors. It is essential to apply interventions to decrease inflammation and increase the concentration of growth factors. Higher responses in those with diabetes and ulcers could be attributed to bilateral disorders.

Conclusion

Pro-inflammatory and inflammatory factors are higher in those with diabetes, especially with diabetic ulcers. Higher inflammatory concentrations are a response to inflammatory status.

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