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Determination Superoxide Dismutase and Glutathione Levels in Serum of Patients with Diabetic Foot Infection in Karbala City

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تحديد مستويات سوبراوكسايد-دسميوتايز و الجلوتاثيون في مصل مرضى التهاب القدم السكري في مدينة كربلاء

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ABSTRACT

Background: Diabetic foot infections (DFIs) are identified as localized purulence or inflammation that appears at the site beneath the malleoli in a diabetic patient. The goal of this study was to measure the levels of glutathione (GSH) and superoxide dismutase (SOD) in healthy people in Karbala city as well as in patients with type 2 diabetes mellitus who had or did not have diabetic foot ulcers.

Materials and Methods: This study involved 120 participants ranging in age from 35 to 75 years, and was conducted at the Imam Al-Hassan Center for Endocrinology and Diabetes in Karbala, Iraq. To seperate the serum, five milliliters of the participants' blood were collected, put within gel tubes, and spun at 4000 xg in a cooled centrifuge. The serum was stored at -20˚C in Eppendorf tubes_prior to utilizing it in determination of SOD and GSH levels.

Results: Superoxide dismutase (SOD) activity mean levels in DFI group where the result from patient were significantly lower (241.4±35.61) (U / L) ($p \leq 0.001$) than in diabetic patients and healthy individuals. Similarly, glutathione (GSH) mean levels in DFI group patients were lower than in diabetic patients and healthy individuals (26.586±2.77) µmole/ml ($p \leq 0.001$).

Conclusion: These results imply that diabetic foot infections may be influenced by decreased antioxidant levels. This suggests that these antioxidants may be used as possible markers to diabetic those who are more vulnerable.

Key words :Diabetic foot infection; Diabetes mellitus; SOD; GSH; antioxidants

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INTRODUCTION

Clinically, DFI is characterized as the existence of signs of an inflammatory process in any tissue located underneath the malleoli in a diabetic patient. Peripheral neuropathy, peripheral artery disease, or immune dysfunction can be used as screening markers for inflammation in people with diabetic foot problems. DFIs normally start when the protective cutaneous envelope is disrupted, usually at the site of an ulceration or trauma. This is frequently seen in patients with peripheral neuropathy and peripheral artery disease [1].

Nowadays, oxidative stress is believed to be a key factor in diabetic wound healing. The body produces too many reactive oxygen species (ROS) as a result of an imbalance between free radicals and antioxidants, which harms cells and tissues and decelerates the healing of wounds. Consequently, dropping ROS levels with ant oxidative systems may lessen damage brought on by oxidative stress and enhance healing [2]. Insulin resistance, β-cell dysfunction, and late diabetes complications can all be brought on by oxidative stress [3]. Reactive oxygen species, or ROS, play a crucial role in controlling several phases of wound healing. Low ROS levels are definitely required to protect against external harm [4]. However, severe oxidative stress on tissues and a decrease in antioxidant capacity result in redox imbalance, which is the main reason diabetic wounds don't heal [5]. Research has shown that people with long-term type 2 diabetes have significant declines in antioxidant enzyme activity, and that highly oxidizing conditions linked to hyperglycemia and tissue hypoxia penetrate non-healing diabetic wounds, delaying wound repair [6]. In order to mitigate the impact of free radicals, the body produces additional substances known as antioxidants, which can come from external sources or be produced internally. These substances include vitamins A, C, and E, minerals like Se, Mn, Cu, and Zn, and enzymes like catalase, glutathione peroxidase, and glutathione reductase. Pro-oxidants and antioxidants maintain a balance in a healthy body, and oxidative stress results from a shift in this balance in favor of pro-oxidants. In order to shield biological systems from oxidative stress, human antioxidant defenses have evolved [7]. One of the key metallo-enzymes in the body's antioxidant defense against oxidative stress is superoxide dismutase (SOD). Therefore, SOD supplements may be employed in a variety of pathological conditions and boost the body's natural antioxidant mechanism to neutralize excessive free radicals [8]. Usually, ROS and the superoxide anion O2 •− have two roles to play. They are a byproduct of O2 reduction and necessary for cell signaling in a healthy equilibrium situation. However, in a pathological environment, they are deemed hazardous since they can promote disease and the death of cells through autophagy, necrosis, and apoptosis. Superoxide dismutase is an enzyme that plays the important role in excluding the majority of superoxide produced by living things. Furthermore, the toxicity caused by superoxide and its derivative. Additionally, superoxide and its byproducts' toxicity play a crucial role in the oxidative elimination of microbial pathogens, which leads to their overabundance by immune cells with specific functions, such neutrophils or macrophages,

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when innate immunity is activated [9]. Free radicals are eliminated by glutathione (GSH), which also lowers hydrogen peroxide levels in cases of oxidative stress by turning hydrogen peroxide into water. Diabetes patients' erythrocytes have been shown to have a decrease in their lowered GSH level. Increased oxidative stress (increased ratio of NADH/NAD) and competition between aldose reductase and glutathione reductase for NADPH, a cofactor, cause a decrease in the level of GSH [10].

MATERIALS AND METHODS

Study design and Samples collection:

The study was designed to be a case control. 120 blood samples were drawn from 40 patients with diabetic foot ulcers patients with type 2 diabetic foot ulcers who met the age requirement of 35 and who had received a previous diagnosis from a clinical physician at the Imam Al-Hasan Center for Endocrinology and Diabetes between October 2022 and January 2023 were included in the study. Additionally, forty blood samples from patients with type 2 diabetes and forty blood samples from healthy people were taken. The blood samples were stored in gel tubes until they were seperated in a cooling centrifuge. The serum was then stored in a deep freezer until it was needed to determine SOD and GSH levels. Individuals under 35 years old, those with foot ulcers who are not diabetics, those with Type 1 Diabetes Mellitus, and pregnant women were excluded from this study.

Determination of antioxidants concentration in serum:

Evaluation of Superoxide dismutase (SOD) activity:

According to Ref [11], the autoxidation of pyragallol was used to measure the activity of superoxide dismutase.
1. Tris buffer (pH 8.0) was made by dissolving 0.111 g of EDTA and 0.258 g of tris in dH2O, then adding water to reach a volume of 100 ml.
2. To make pyragallol solution (0.2 mM), 0.0252 gm of the substance was dissolved in 10 ml of HCl, and the volume was then increased to 100 ml with dH2O.

Procedure

The reaction mix, according to Ref [11], consists of 50 µl serum, 2 ml of tris buffer, and 0.5 ml of pyragallol (0.2 mM), which is a light-absorbing substance that absorbs light at 420 nm. The components in the control solution are the same, except dH2O has been used in place of the enzyme extract. We utilized dH2O as a blank. The quantity of enzyme needed to prevent 50% of

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pyragallol oxidation is known as one unit of enzyme. The following formula was used to determine SOD activity [11].

$$SOD\ Activity\ (unit) = \frac{\frac{\%P}{50\%} \times R}{T}$$

Where:

• %P: the percentage of inhibition of pyragallol reduction

*Note: Δabs of each sample (X%) are compared to Δabs of the control (100%) to determine the %P of each sample.

• R: Volume of the entire reaction (2.55 ml)
• T: Reaction time in minutes (two minutes)

Evaluation of Glutathione (GSH) concentration:

- **Principle:**

Glutathione molecules readily degrade Ellman's reagent 5,5 Dithio bis (2-Nitro benzoic acid) (DTNB), resulting in a yellowish product with a high optical density and increased absorbance at 412 nm that is directly proportional to glutathione content [12]. Methodology outlined by Moron et al. (1979) for assessing the GSH level in serum, whereas the subsequent remedies have been made:

1. The first step in making the sodium phosphate buffer (0.2M) was to dissolve 2.4 grams of sodium phosphate in 100 milliliters of distilled water. The pH was then adjusted to 8. 2. Dithionitrobenzene solution (0.6 mM) (DTNB): This was made by dissolving 0.023 gm of DTNB in the 0.2 mM sodium phosphate buffer that was previously prepared. Once the dissolution was complete, the volume of the buffer was increased to 100 ml. The preparation of trichloroacetic acid (TCA) 5% involved dissolving 5 grams of TCA in 100 milliliters of distilled water.

4. Tris buffer solution (1.4 M) was made by dissolving 4.82 ml of Tris-base in 10 ml of EDTA-Na2 (0.4 M), which was made by dissolving 1.489 gm of EDTA-Na2 in 10 ml of distilled water, then adding 0.1N of hydrochloric acid to get the pH to 8.9.

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• Setting up the glutathione standard curve:

The methods employed:

1. EDTA-Na2 (0.2M): 100 milliliters of distilled water are used to dissolve 0.477 grams of EDTA-Na2.

2. The preparation of the standard glutathione solution (3 mg/ml) involves dissolving 0.0307 g of glutathione in 10 ml of EDTA-Na2 (0.2 M).

The subsequent volumes of a standard glutathione solution were added in test tubes (two tubes for each volume) after the graduated concentrations of glutathione standard solutions were created. Next, the appropriate amount of EDTA-NA2 was applied. The tubes were then well mixed for ten to fifteen minutes, centrifuged at 300 xg for fifteen minutes, and the absorbance was measured using a spectrophotometer by using the blank solution to read the zero absorption at 412 nm. The sample absorbance was then read five minutes after the addition of Ellman's reagent. Next, 0.02 ml of DTNB and 0.8 ml of Tris buffer were added to each tube. The calibration curve was used to determine the GSH concentration, with concentrations ranging from (0.0, 5, 10, 20, 30, 40, 50 , 60 , 80, and 100) µmol/L that mentioned in Figure No. 1

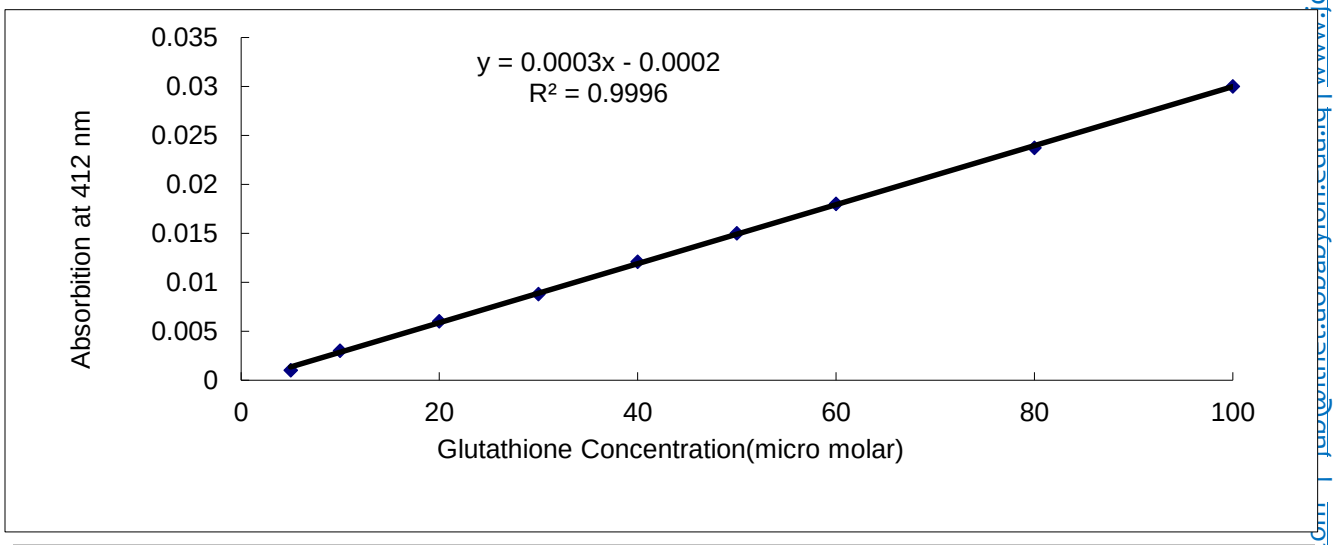

Fig. No. 1 : Standard curve of GSH

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Ethical consideration:

The Ethics Committee at the University of Karbala's College of Science approved this work. Before any samples were collected, verbal consent was obtained from each participant and all subjects included in this study were informed.

Statistical Analysis:

We used statistical analysis software such as SPSS and Real Statistics to crunch the numbers for our investigation. Finding significant differences among the groups under study was our aim. If a difference met a rigorous threshold, it was considered statistically significant.

RESULTS AND DISCUSSION

A 120 people were included in the case-control study, 40 of them were T2DM patients with diabetic foot ulcers (DFI), 40 of them were T2DM patients without foot ulcers, and 40 of them appeared to be in the healthy control group. Samples were obtained at the Imam Al-Hassan Center for Endocrinology and Diabetes over the course of four months, from October 2022 to January 2023.

Compared to healthy controls, patients with diabetic foot infections had lower SOD range levels. There is a substantial difference in this biomarker level between the groups participating in the current study, according to table No. 1's results. Compared to DM and healthy individuals, SOD activity was considerably lower (241.4±35.61) (U/L) in patients in the DFI group ($p \leq 0.001$). In this investigation, the DFI group's glutathione concentration was significantly lower than that of the DM and control groups. Table No. 1's results showed that there was a substantial variation in this biomarker level between the groups that were enrolled in the current study. When compared to DM and healthy persons, the mean blood GSH levels in the DFI group patients reduced significantly (26.586±2.77) µmole/ml ($p \leq 0.001$).

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Tab. No. 1: Mean difference of antioxidants among the Three Studied Groups

Biomarker	DFI N=40 (mean±SD)	DM N=40 (mean±SD)	Control N=40 (mean±SD)	*P*-value
SOD (U/L)	241.4±35.61	160.44±66.78	307.90±45.71*	<0.001
GSH (µmole/L)	26.586±2.77	28.47±2.06	29.75±2.20*	<0.001
ANOVA was *: significant at p ≤ 0.05, Post hoc (LSD)				
SD: standard deviation; *: significant				

The current findings were in conflict with those of Bolajoko et al. (2017) in Nigeria, who found that when DFU patients and controls were examined, non-significant decreases in SOD activity levels were seen (P > 0.05) [13]. However, a local investigation by Moustafa and Omar (2017) discovered that individuals with T2DM had significantly lower serum levels of SOD than the control group (p< 0.001) [14]. The study's findings about the decline in SOD activity are similar to those of Bhatia et al. (2003), who reported a noteworthy decline in SOD activity in individuals with diabetes [15]. However, when evaluating the antioxidant enzymes, Bandeira et al. (2012) found that only SOD activity differed significantly between the groups, with diabetic patients having higher SOD activity [16].

Reactive oxygen species (ROS) are essential modulators of multiple stages in the healing process of wounds. Low ROS levels are definitely necessary for avoiding external damage [17]. However, redox imbalance is a key cause of diabetic wounds that do not heal due to high oxidative stress on tissues and a loss in antioxidant potential [18]. Clinical research revealed that the highly oxidizing environment that is linked to hyperglycemia and tissue hypoxia penetrates diabetic wounds that do not heal, delaying wound healing. Antioxidant enzyme activity is significantly reduced in those with long-term type 2 diabetes [19]. As a result, serum Oxidative Stress indicators were identified as possibly useful instruments for the DFI's diagnosis and treatment [20].

The results presented above contradicted those of a local study by Moustafa and Omar (2017), who found a significant negative fragile correlation between age and SOD in the patient group. They also reported that the serum level of SOD decreased with increasing age [14], while Hisalkar and his collaborates found that the SOD level decreased in the diabetic age group ≥50

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years compared to age groups (30–39) and (40–49) years. Accordingly, the findings of Hisalkar demonstrated that Oxidative Stress, which affects the aging process, can be brought on by a variety of things, such as an increase in the generation of free radicals, a drop in the antioxidant defense system, or a decrease in the removal or repair of old materials [21].

Olędzki and his colleagues' examined the SOD and CAT activity in erythrocytes obtained from young, healthy adults (aged 20–29) and elderly people (age > 60). They noted that the elderly patients' SOD activity was lower [22]. Bartosz found that glycation of SOD m and additional hydrogen peroxide can both inactivate enzymes, which explains why SOD activity declines in older women [23]. The aforementioned findings corroborated a local investigation by Muhanedalnajer and his colleagues, which found that GSH and total thiol levels were significantly lower in DFI as compared to the control group [24]. However, studies conducted in Algeria and Poland by Aouacheri and Gawlik found that GSH levels in diabetic patients were considerably greater than in healthy controls [25, 26]. Other studies that found that diabetes patients' plasma GSH levels are lower than those of controls also revealed decreased levels of GSH [27]. Furthermore, several of the metabolic abnormalities observed in diabetic patients and after the start of diabetic complications were assumed to be caused by a decreased GSH level [28]. Glutathione (GSH) protects the human body by lessening the effects of free radicals [29]. One major function of reduced glutathione (GSH) is to neutralize free radicals, protecting cells from oxidative damage. Diabetes mellitus is thought to be commonly caused by oxidative stress, which lowers extracellular and intercellular antioxidant levels. GSH is a non-enzymatic antioxidant that uses several strategies to either stop or prolong the oxidative process. Particularly susceptible to oxidative stress, antioxidant enzyme levels have been shown to both rise and fall in a variety of disease situations where elevated oxygen species are either a contributing factor to or the cause of diabetes mellitus [30].

Regarding age, table No. 2 illustrates our findings, which show that SOD activity was higher in patients aged 55–64 and thereafter decreased in older patients.

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Tab. No. 2: The effect of age on SOD activity according to the three studied groups

Groups	(35 – 44) Years (mean±SD)	(45 – 54) Years (mean±SD)	(55 – 64) Years (mean±SD)	(65 – 74) Years (mean±SD)	(≥75) Years (mean±SD)
	SOD activity (U/L)				
DFI	248.97±14.38	251.1±14.38	262.97±15.75	239.5±14.38	204.43±15.75
DM	190.85±14.38	191.75±14.38	189.76±15.75	125.45±15.75	104.39±14.38
Control	298.61±14.38*	338.6±14.38*	329.72±14.38	290.71±14.38*	281.85±14.38*
ANOVA was *: significant at p ≤ 0.05, Post hoc (LSD) SD: standard deviation; *: significant					

Table No. 3 illustrates how GSH levels decline with age in relation to age.

Tab. No. 3: The effect of age on GSH concentration according to the three studied groups

Groups	35 - 44 Years (mean±SD)	45 - 54 Years (mean±SD)	55 - 64 Years (mean±SD)	65 - 74 Years (mean±SD)	≥75 Years (mean±SD)
	Concentration (µmole/ml)				
DFI	27.03±0.89	27.16±0.89	26.62±0.98*	26.69±0.98	25.43±0.98
DM	29.87±0.89	29.65±0.89	28.54±0.98	27.5±0.98	26.82±0.89
Control	31.83±0.89	30.09±0.89	29.9±0.89	28.83±0.89	28.1±0.89*
ANOVA was *: significant at p ≤ 0.05, Post hoc (LSD) SD: standard deviation; *: significant					

In a Dutch study, It has been found that as people aged, both male and female GSH concentrations declined. GSH content was substantially lower in the age group of 60–80 years than in the age groups of 20–40 and 40–60 years for both sexes. These observations suggest that the detoxification capability of the GSH system in lymphocytes may decline with age in humans, since high GSH is a necessary component in the detoxification of numerous substances [31].

As the primary thiol-disulfide redox buffer in mammalian cells and a direct co-factor in enzyme reactions, glutathione is essential for numerous biological activities. Additionally, glutathione offers a vital defense mechanism that shields cells from a variety of stresses. Glutathione levels

seem to drop in a variety of tissues with aging, which increases the likelihood that cells will break under stress [32]. Redox imbalances may play a critical role in the onset and delayed healing of diabetic foot ulcers, according to a number of prior studies [33]. As a result, serum Oxidative Stress indicators were identified as possibly useful instruments in the DFI management and diagnosis [20].

Odd ratio (OR):

To examine the relationship between GSH and SOD and instances of diabetic foot infection and diabetes, multinominal logistic regression analysis was used. As stated in table No. 4, it was discovered that the biomarkers GSH and SOD are protective factors that demonstrated a very significant in DFI (OR 0.616; 95% CI: (0.474-0.801) and OR: 0.961; 95% CI: (0.941-0.982)), respectively.

Tab. No. 4: the Associated of the analyzed factors in Diabetic foot infection disease two cases Compared to the control group

Variable	Groups	OR (CI:Lower – upper)	P value
GSH			
	Control	1[a]	**-**
	DF	0.616 (0.474-0.801)	**<0.001**
	DM	0.708 (0.555-0.902)	**0.005**
SOD			
	Control	1[a]	**-**
	DF	0.961 (0.941-0.982)	**<0.001**
	DM	0.955 (0.934-0.976)	**<0.001**
p<0.05 considered significantly different, 1[a] : reference category is Control			

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CONCLUSION

The Presence of significant lower levels of antioxidants in patients compared with healthy volunteers also the declined levels of superoxide dismutase (SOD) and glutathione (GSH) in patients with Diabetic Foot Infection (DFI) compared to Diabetic patients and healthy control may consider these markers as a protective factors.

Conflict of interests:

There are non-conflicts of interest.

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الخلاصة

المقدمة: عدوى القدم السكري (DFIs) تعرف بأنها علامات موضعية للالتهاب أو صديد يحدث في الموضع أسفل الكاحلين عند مريض السكري .هدفت هذه الدراسة إلى تحديد مستويات إنزيم سوبراوكسايددسميوتايز (SOD) والجلوتاثيون (GSH) عند مرضى السكري من النوع الثاني المصابين بقرح القدم السكري أو غير المصابين بها وكذلك الأفراد الأصحاء في مدينة كربلاء .أجريت هذه الدراسة في مركز الإمام الحسن للسكري والغدد الصماء في كربلاء بالعراق. **طرق العمل:** شملت الدراسة 120 شخصا" بداية من 35 سنة إلى 75 سنة فأكثر .تم سحب خمسة مل من الدم من المشاركين في الدراسة ثم وضعها داخل أنابيب جل وتم طردها بمعدل 4000 g × في جهاز طرد مبرد للحصول على المصل .ووضع المصل في أنابيب ابندروف وحفظ بدرجة حرارة (−20 درجة مئوية) حتى استخدامه لتقدير مستويات SOD و GSH. **النتائج**: أظهر متوسط مستوى نشاط إنزيم سوبراوكسايددسميوتايز (SOD) في مجموعة مرضى عدوى القدم السكري انخفاضًا ملحوظًا في القيم (241) (35.61 ± 4.وحدة / لتر) (قيمة الاحتمالية 0.001 ≥بالمقارنة مع مرضى السكري والأفراد الأصحاء وكذلك الجلوتاثيون (GSH) حيث انخفضت القيم المتوسطة لهذا المؤشر في مجموعة مرضى عدوى القدم السكري بشكل ملحوظ (26 (2.77 ± 586.ميكرومول / مل (قيمة الاحتمالية ≥ 0 (001.بالمقارنة مع مرضى السكري والأفراد الأصحاء.

الاستنتاجات :تشير دراستنا الحالية إلى أن انخفاض مستويات إنزيم سوبراوكسايددسميوتايز (SOD) والجلوتاثيون (GSH) بشكل ملحوظ لدى مرضى عدوى القدم السكري مقارنة بمرضى السكري والسيطرة الصحية قد يستخدم كمعلم للكشف عن المضاعفات للاشخاص الاكثر عرضة

الكلمات المفتاحية: قرحة القدم السكري ; داء السكري; سوبراوكسايددسميوتايز ; جلوتاثيون; مضادات الاكسدة

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