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Oxidative Stress and Related Consequences in Patients with Type 2 Diabetic Mellitus

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ABSTRACT

Background: An imbalance between biochemical pathways that increase ROS generation and the body's antioxidant defense system causes oxidative stress (OS), a metabolic disorder. It's linked to heart disease, cancer, neurological issues, aging, and respiratory infections.

Objective: This study compared persons with and without complications from type 2 diabetes (T2D) in terms of their total antioxidant capacity and total oxidative stress in the blood.

Methods: The sample consisted of 80 T2D patients, either with or without subsequent complications. Two groups of participants were formed: those with diabetes (Diab) and those with secondary complications (DSC). To extract serum, blood samples were taken. The colorimetric approach was used to assess the serum samples' total antioxidant capacity (TAC) and total oxidative stress (TOS).SPSS 23.0 was used to analyze all data.

Results: Blood glucose and total oxidative stress were considerably ($p=0.05$) greater in the Diab group than in the DSC group. However, the DSC group's total antioxidant capacity was considerably ($p<0.003$) lower than the Diab group.Patients with T2D who experienced secondary problems had higher levels of oxidative stress.

Conclusion: The conclusion showed that total oxidative stress was higher and total antioxidant capacity was lower in type 2 diabetic patients compared to non-diabetic individuals; this, in turn, causes the development of difficulties related to the disease

Keywords: Diabetic, T2D, Complications, Oxidative stress, TAC.

1. INTRODUCTION

Type II diabetes mellitus (T2DM) is expected to become one of the leading causes of disability and premature mortality globally, with a prevalence of 700.2 million by 2045 [1]. By the same year, 8.3% of the global population will be living with prediabetes, the condition that leads to type 2 diabetes. A person has prediabetes if their blood sugar levels are neither too high nor too low. Pathogenic processes and effects associated with hyperglycemia can be observed even in the absence of symptoms [3].

Modern research has linked type 2 diabetes to oxidative stress and inflammation [4]. Insulin resistance is the hallmark of type 2 diabetes, and oxidative stress (OS) is an additional factor in both the development and advancement of the illness. Because their antioxidant defense mechanism is weakened, pancreatic β cells are more prone to damage and cell death caused by reactive oxygen species (ROS) [5]. Additionally, OS hinders the development and operation of β cells, which leads to a decrease in both the quantity and quality of insulin released. One more way OS might reduce insulin's effectiveness is by interfering with glucose signaling pathways. Similar to how prediabetes develops into diabetes, hyperglycemia develops when insulin secretion and function both decline at the same time [5,6].

A lot of theories have been put out over the last hundred years to try to explain how diabetic complications occur. For example, oxidative stress [8], hypoxia (both actual and artificial), carbonyl stress, advanced glycation end-products (AGEs),

the Maillard hypothesis [6,7], polyol pathway activation, increased protein kinase C activity, disruptions to lipoprotein metabolism, altered expression of cytokines or growth factors, and so on. One reason for this is that there are still many unanswered questions about the cause of diabetic complications. Possible explanations for these enigmas include a common pathogenic mechanism, the activation of stress response pathways specific to different tissues, or a combination of the two. The general public tends to see these ideologies as interconnected and overlapping. To be more specific, many people thought that oxidative stress could unite all the hypotheses. One way oxidative stress can speed up the synthesis of AGEs is by increasing free radical scavenging. The subsequent release of growth factors and inflammatory cytokines may occur as a result of these AGEs activating redox signaling pathways. Oxidative stress can result from a variety of sources, including the polyol pathway, hypoxia (which initiates glycolysis and inflammatory maintenance through ROS formation), carbonyl stress, and others. A unified paradigm about the pathobiology of diabetes complications was therefore developed at the beginning of the century. There are several important pathways that have been linked to the development of diabetes problems, and ROS is thought to be the main initiator in many of these pathways [9]. The unifying theory has rapidly become a cornerstone of diabetes research, attracting nearly all researchers who have investigated diabetic complications thus far. Disappointingly, human trials of a number of antioxidants that had shown promise in preclinical animal models only provided limited protection against diabetic sequelae. However, new experimental data shows that diabetes-related problems can develop even without elevated ROS production.

Oxidative stress, which can result from an upsurge in reactive oxygen species (ROS) generation and a decline in the body's antioxidant defenses, is a known risk factor for chronic diabetic problems [10]. Superoxide dismutase (SOD), glutathione (GSH), bilirubin, ceruloplasmin, albumin, uric acid (UA), and ascorbic acid (CA) are antioxidants that can prevent macromolecular oxidation. To neutralize free radicals and shield the body from their toxic byproducts, antioxidants donate electrons to them [11]. Diabetes exacerbates oxidative stress because the body produces more free radicals, greater lipid peroxidation, and has weaker antioxidant defenses.[12] The purpose of this research was to examine the total oxidative stress and serum total antioxidant capacity in individuals who suffer from type 2 diabetes (T2D) and its complications

2. MATERIALS AND METHODS

This comparative study was conducted at Gulab Devi Hospital/ Al-Aleem Medical College, Lahore during December 2023 to August 2024 and comprised 80 patients. Presented al patients were males.

We took great care in selecting human participants based on their medical histories. The authenticity of the medical history and clinical profile of the diabetic group without subsequent problems was ensured. Patients' medical histories and current clinical profiles were used to determine the diabetic group with secondary problems. The research did not begin until written informed consent was obtained. Two groups were formed according to the participants' medical histories. Forty individuals made up each group. In the first group, known as DSC, were patients with diabetes and any problems that came with it; in the second, known as diab, were patients who had just been diagnosed with diabetes and had no other health issues. Between 11 a.m. and 12 p.m., we took blood samples from each participant and stored them in vacuum containers without anti-coagulant so we could extract serum. We measured total oxidative stress, total antioxidant capacity, and serum blood sugar in the serum. A colorimetric approach was employed to assess serum total oxidative stress (TOS), antioxidant capacity (TAC), and serum glucose level (Bioclin® Glucose Monoreagent detection kit), all of which are commercially available Elisa kits.

Statistical significance across groups was established using one way ANOVA followed by a post hoc test, and all datasets were reported as mean + SEM. If the p-value was less than 0.05, the result was deemed statistically significant.

3. RESULTS

Mean BMI in DSC group was 26.12±5.22 kg/m^2 and in Diab group mean BMI was 30.17±8.57 kg/m^2. The glucose level in group DSC was lower as compared to Diab group < 0.003. (Table 1).

Table 1: Comparison of glucose and BMI

Variables	DSC	Diab	P Value
Mean BMI (kg/m^2)	26.12±5.22	30.17±8.57	<0.005
Mean glucose level (mg/dl)	171.15±23.68	260.67±18.58	<0.003

However, the DSC group's total antioxidant capacity was considerably ($p<0.003$) lower than the Diab group.Patients with T2D who experienced secondary problems had higher levels of oxidative stress. (Table 2).

Table-2: Comparison of TAC and TOS

Variables	DSC	Diab	P Value
Mean TAC (mmol/L)	168.53 ±14.67	252.67±35.81	<0.001
Mean TOS (µmol/L)	47.37 ± 2.15	37.005 ± 3.1	<0.002

4. DISCUSSION

Compared to diabetic patients without secondary problems, those with diabetes (Diab) had a substantially higher serum glucose level (P≤0.05) in the present study. A prior study by Moussa (2008), [13] corroborated these findings, demonstrating that diabetic patients in the Egyptian community had substantially lower insulin levels and significantly higher blood glucose levels compared to healthy individuals. According to Evans et al. (2003), [14], who stated that elevated ROS generation could impair the activity of pancreatic β cells leading to reduced insulin secretion, the increased serum glucose level and decreased insulin level in newly diagnosed diabetic patients compared to healthy controls may be caused by increased oxidative stress. One of the main causes of type 2 diabetes is being overweight.

Compared to other groups, those who were overweight (measured by body mass index) in our study stood out, which went against what Farasat et al. (2009) found, which indicated that there were no significant variations in body mass index (BMI) between diabetes and healthy patients. Nevertheless, our findings are in line with those of Fawwad et al. (2006), [16] who found a substantial association between BMI and T2DM. They may have picked diabetic patients with a family history of the disease, which could explain this. Total antioxidant capacity was much lower in the DSC group compared to the Diab group in our study.

The findings of Zargari et al. (2018) and Varadhara et al. (2017)[17,18] corroborate these findings, showing that DSC patients had lower TAC compared to Diab patients. On the other hand, Verma et al. (2018) found that diabetes individuals had an enhanced total antioxidant capacity, which contradicts our results.The year 19 Similarly, our study indicated that total oxidative stress was considerably higher in the DSC groups compared to the Diab groups. Savu et al. (2012) reported that diabetic patients had higher levels of total oxidative stress compared to healthy individuals, which supports these findings.[20]

5. CONCLUSION

The conclusion showed that total oxidative stress was higher and total antioxidant capacity was lower in type 2 diabetic patients compared to non-diabetic individuals; this, in turn, causes the development of difficulties related to the disease.

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