The Impact of Oxidative Stress Levels in Individuals with Type 2 Diabetes Mellitus and Hypertension

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Description

Type 2 diabetes mellitus (T2DM) and hypertension are two common chronic diseases that often coexist. Oxidative stress, a condition where there is an imbalance between the production of reactive oxygen species and the body's antioxidant defense mechanisms, is known to play a role in the pathogenesis of both T2DM and hypertension [1, 2].

Research has shown that there is a correlation between the level of oxidative stress and the severity of these diseases. In T2DM patients with hypertension, a higher level of oxidative stress has been observed, which may contribute to the development and progression of complications such as cardiovascular disease and kidney damage [3- 5].

Therefore, it is important to manage oxidative stress levels in T2DM patients with hypertension through lifestyle modifications such as regular exercise, a healthy diet, and stress reduction techniques. Additionally, antioxidant supplements may be beneficial in reducing oxidative stress levels.

Overall, understanding the relationship between oxidative stress and T2DM with hypertension can lead to better management and prevention of complications associated with these chronic diseases. Oxidative stress plays a potential role in certain diseases such as diabetes mellitus [6-8]. Prolonged exposure to oxidative stress can lead to complications like hypertension and cardiac disease. The aim of the present study was to assess the state of oxidative stress in patients with type 2 diabetes mellitus (DM 2) and hypertension to understand the role of oxidative stress in diabetic complications [9]. The study measured levels of reactive oxygen species (ROS) and total antioxidant (TAO) in the serum of the patients.

The results indicated that hypertension was more prevalent in older age groups compared to other groups, although the difference was not statistically significant (P = 0.109) [10, 11]. Body mass index (BMI) showed slight variation between the groups, but again, the difference was not statistically significant (P = 0.574). There were no significant differences in fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) levels, with a slight decrease observed (P = 0.780 and 0.068, respectively).

Significant differences (P = 0.000) were observed for systolic (SYS) and diastolic (DIA) blood pressure. TAO levels showed a slight decrease in patients with both DM and hypertension (DM+HP), while ROS levels slightly increased in the same group, but these differences were not statistically significant (P = 0.676 and 0.736, respectively). In the DM group, ROS showed a weak non-significant correlation with SYS and a weak inverse correlation with DIA. In the DM+HP group, there was a weak non-significant inverse correlation with SYS and DIA. TAO showed a weak inverse correlation with SYS and a weak positive correlation with DIA in both the DM and DM+HP groups. Additionally, there was a significant inverse correlation between TAO and ROS in both groups [12, 13]. The study concluded that the elevation of ROS and the decrement of TAO were associated with hypertension in patients with DM.

The oxidative-redox system is responsible for maintaining the balance of free radicals and antioxidant molecules in the body. Prolonged imbalance in this system leads to oxidative stress, which contributes to the incidence and development of various diseases. The present study demonstrated that DM patients had higher levels of ROS and lower TAO levels, which is consistent with findings from other studies [14].

The pathophysiology and complications of DM involve the production of ROS through different mechanisms, including the polyol flux pathway, excessive formation of advanced glycation end products, increased expression of receptor for AGEs, activation of protein kinase C isoforms, overactivity of the hexosamine pathway, and inactivation of anti-atherosclerotic enzymes such as endothelial nitric oxide synthase and prostacyclin synthase. Oxidative stress contributes to the development of hypertension through mechanisms such as quenching of the vasodilator nitric oxide, generation of vasoconstrictor lipid peroxidation products, depletion of tetrahydrobiopterin, damage to endothelial cells and vascular smooth muscle cells, elevation of intracellular free calcium levels, increased endothelial permeability, and stimulation of inflammation and growth signaling events [15]. While oxidative stress can stimulate hypertension, it remains unclear whether ROS initiate the development of hypertension. Previous studies by Lassègue and Rhian have shown a strong association between hypertension and oxidative stress.

Considering the prolonged period of oxidative stress in DM patients and its contribution to complications, it is important to treat it with supplements, antioxidant-rich foods, and appropriate medications [16].

Acknowledgement

None

Conflict of Interest

None

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