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Antioxidant activity of superoxide dismutase in relation to the neurological complications in diabetes mellitus

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Abstract

Aim: The antioxidant activity of superoxide dismutase (SOD) was evaluated with the duration of neurological problems like poly/sensory neuropathy due to diabetes.

Material and Method: The study was conducted with 55 diabetes patients with poly/sensory neuropathy from the out patients endocrinology department of Ramakrishna Mission Sevapratishthan, Kolkata, along with 25 healthy controls. All the procedure of the study was followed with the consent of the participants. In this study, the patients have been selected after nerve conduction velocity (NCV) test for identifying the poly/sensory nerve dysfunction. The SOD activity of the RBC lysate was determined.

Result: In our study, the SOD activity was increased with the duration of neurological dysfunction in relation to diabetes.

Conclusion: The increase in activity with duration of the diabetic neuropathy may be a result of protective mechanism against oxidative stress developing in the tissue.

Keywords: diabetes mellitus, neurological dysfunction, superoxide dismutase, antioxidant, oxidative stress

1. Introduction

India faces a grave health care burden due to the high prevalence of type 2diabetes and its sequel, as a result of societal influences and changing lifestyles [1]. It is a complex and progressive disease that results in multiple complications which include nerve damage resulting poly/sensory neuropathy. Considering the epidemic of diabetes throughout the world and the fact that diabetic neuropathy (DN) is one of the most common long term complications of diabetes, it is important to look into details of its pathology. Oxidative stress resulting from enhanced free radical formation has been implicated in the pathogenesis of neurological complications in diabetes. Oxidative stress rooting from long term hyperglycemia has been established as a link that provides a unified mechanism of tissue damage [2]. International researchers observed that antioxidants commonly promoted as being good for our health may speed up early onset of type 2diabetes by mopping up free radicals i.e. Reactive Oxygen Species (ROS) that may play a protective role in the early stages of type 2 diabetes by enhancing insulin action. [3]. In diabetic patients an altered balance between reactive oxygen species production and antioxidant levels has been reported but there is still lack of data regarding the actual status of antioxidant enzymes in diabetic patients. [4]. In healthy individuals, oxidative damage to tissue is prevented by a system of defence which includes antioxidant enzymes and small molecules with scavenging ability such as antioxidant, vitamins [5]. So the present study aimed in evaluating the antioxidant activity of SOD which plays an important role in antioxidant defence mechanism in relation to the neurological complications which developed with the duration of the disease.

2. Materials and Methods

The type 2 diabetic neuropathy (DN) patients were taken from the outpatient Department of Endocrinology, Ramakrishna Mission Seva Pratishthan, Kolkata. A questionnaire was used in the assessment of DN presence according to clinical symptom, duration, medication and socioeconomic condition. All procedures were done with the consent of participants. This study was conducted in the Genetic unit of Vivekananda Institute of Medical Sciences, Ramakrishna Mission Seva Pratishthan, Kolkata and was pre-approved by the institutional Ethics Committee. The study involved 80 individuals and consisted of 55 patients with type 2 Diabetes mellitus (DM) along with neuropathy and 25 patients without a history of diabetes as a control group. Patients were included in the study after confirmation gained by impaired fasting glucose test (>126 mg/dl) and positive nerve conduction velocity report.

For enzyme SOD activity 3 ml of venous blood was collected from cubital vein in EDTA (k2) containing vial. After collection, the blood was kept 30 minutes for settling down the cells. Then the whole blood was centrifuged at 3000 rpm for 10 minutes to collect the RBC (aspirate off the plasma). Then collected RBC was washed with 9% NaCl solution and centrifuged for 10 minutes at 3000 rpm after each wash. The

lysate was diluted with 0.01 mol/l Phosphate buffer (pH 7.0) for SOD assay. The activity of Superoxide dismutase was evaluated by spectrophotomatric method (505 nm) [SpectraMax UV-Visible spectrophotometer (SPECORD 50 PLUS)] using RANSOD kit (Randox Laboratories, Ltd) [6].

3. Result

The subject of this study consisted of 80 individual, aged 53.6 ± 11.6 years, with 55 type 2 with neuropathy and 25 controls. The average time from diagnosis of diabetes was 10.2 ± 7.1 years. Table 1 present the characterization of study sample, according to socioeconomic, clinical and biochemical profiles. Nerve conduction velocity (NCV) was done in all the 55 diabetic neuropathy patients. The patients having positive sensory/polyneuropathy problem have been selected in the present study.

When the patients were grouped in relation to the duration of disease, an interesting observation was revealed. It was observed that SOD activity significantly increased from 0 years to >16 years of the disease. Conversely, no difference in activity was revealed during the mid period from 0 - >16 years though they are grouped according to the duration of diabetes neuropathy.

Table1: Genaral and biochemical characters of the studied group.

	Healthy control	Diabetic Neuropathy
No. of Subject	25	55
Age of the subject year ± Sd	35 ± 9	53.6 ± 11.6
Duration of the disease	=	10.2 ± 7.1
Impaired fasting glucose	_	170 ± 69
Sod activity Unit / gmHb	89 ± 30.5	226.9 ± 55.7

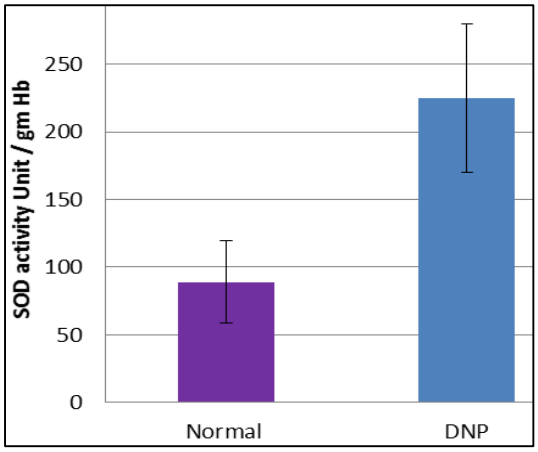

Fig 1: SOD activity of healthy control group and Diabetic Neuropathy Patients (DNP).

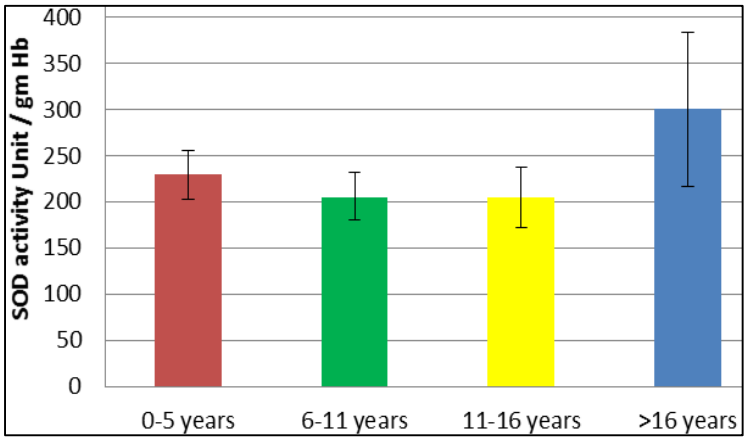

Fig 2: SOD activity in relation to the duration of Diabetic Neuropathy (DN)

4. Discussion

Neurological complication i.e. nerve damage is the common and most troublesome complication of diabetes resulting oxidative stress as unifying mechanism followed by structural and functional loss is already stated. Moreover large number of studies advocate the role reactive oxygen species in the developing and progression of the various disease affecting human being [2]. Considering the epidemic of diabetes throughout the world, diabetic neuropathy i.e. nerve damage is one of the most common complication of diabetes, hence it is important to look into details of its pathophysiology. Since oxidative stress leads to such a major role in the development of nerve damage, several antioxidants have been suggested which inhibit or impede the oxidative damage considering the

imperative role of oxidative stress in mediating nerve dysfunction in diabetes. A large number of antioxidant enzymes have been tested, of which SOD plays an important role in determining the risk of neuropathy in human diabetes patients. The results of the present study demonstrated a significantly higher enzyme activity in diabetic patients having damage and disfunction of nerve as revealed by nerve conduction velocity (NCV) test compared with the healthy control. This is in accordance to the study performed by Bandeira *et al*. which showed a increase in enzyme activity [7]. Increased SOD serum level in the diabetic group may appear as a result of a protective and adaptive mechanism developing in the tissue, and may also be an indicator of the increased generation of ROS in diabetes [8]. Some studies in patients with DM2 have revealed decrease in antioxidant defenses and an increase in oxidative damage markers especially against complication associated with DM2 [9, 10, 11]. Similar results to ours were found by Moussa [1], who observed the higher SOD activity in erythrocytes of type 1 and type 2 diabetes, though our data provided an additional information regarding the status of SOD activity which increased in relation to the severity of nerve damage as depicted by NCV test. Moreover there are several reports revealing a positive correlation between hyperglycemia and antioxidant capacity [1]. However it is worth mentioning that experimental evidences indicating that over expression of antioxidant enzymes can protect neurons against oxidative injury are still lacking [2]. Thus considering the expression, induction of enzyme that protect against oxidative stress induced damages, further in depth studies are warranted for better understanding of the pathogenesis of nerve dysfunction in diabetes and antioxidant activity.

5. Conclusion

Our results suggest that SOD activity in RBC lysate increases with the advancement of neurological complications along with diabetes mellitus compared with the healthy control groups. The increase in activity with duration of the disease may be a result of protective and adaptive mechanism against oxidative stress developing in the tissue. More multicentric, longitudinal studies with a large sample size are needed to have a better understanding of the interrelationship between oxidative imbalance and chronic neurological enhancement of diabetic patients.

6. References

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