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RESEARCH ARTICLE

INFLUENCE OF SUPEROXIDE DISMUTASE ACTIVITY ON DIABETIC NEUROPATHY PATIENTS

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ABSTRACT

Diabetic neuropathy (DN) is one of the most common troublesome microvascular complications and it has also got some oxidative stress mechanism associated with nerve damage, followed by structural and functional change. The present study aimed in assessing the antioxidant superoxide dismutase activity in type 2 diabetes patients with neuropathy. The study was conducted with 109 diabetic neuropathy patients from the out patients endocrinology department of Ramakrishna Mission Seva Pratishthan, Kolkata. 30 healthy controls were also taken in this study. All the procedures were done with the consent of the participants. After confirmed positive sensory / polyneuropathy problem by nerve conduction velocity (NCV) test, the patients have been selected in this study. The present result shows that SOD activity in DN was much higher than that of control group. Furthermore, a considerable difference was observed in male and female patients when different duration of the disease was considered.

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INTRODUCTION

Diabetes is known to be one of the foremost causes of mortality and morbidity in the world (Oyenihi, 2015) and also affects the quality of life (Algaidi, 2011). It is a complex and progressive disease that results in multiple complications which include retinopathy, nephropathy, cardio -myopathy, hepatopathy, and neuropathy (Aljabri 2010). Diabetic neuropathy (DN) is a microvascular complication that affects up to 50% of these diabetic patients and is a major cause of mortality and morbidity in this population. The complex etiology of DN is still not clear (Bulent *et al.,* 2016, Boulton 2005, Liu 2015). Hyperglycemia-induced overproduction of free radicals is widely recognized as the link between diabetes and diabetes complications. Considering the epidemic of diabetes throughout the world and the fact that diabetic neuropathy is one of the most common long-term complications of diabetes, it is important to look into details of its pathophysiology. Oxidative stress rooting from long term hyperglycemia has been implicated in the pathogenesis of diabetic neuropathy (Negi *et al.,* 2011, Vincent 2008). Oxidative stress are measured through certain observable biomarkers, several enzymatic biomarkers, of which Superoxide dismutase (SOD) plays an important role as antioxidant defense in nearly all cells exposed to oxidative stress.

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MATERIALS AND METHODS

This study was conducted in the cytogenetic unit of Vivekananda Institute of Medical Sciences, Ramakrishna Mission Seva Pratishthan, Kolkata and was pre-approved by the institutional Ethics Committee. The type 2 diabetic 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. The study involved 139 individuals and consisted of 109 patients with type 2 DM along with neuropathy and 30 patients without a history of diabetes as a control group. The included patients were nonsmokers, nonpregnant and nonlactating, without alcohol consumption.

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) (Woolliams 1983).

RESULTS

Nerve conduction velocity (NCV) was done in all the 109 diabetic neuropathy patients. The patients having positive sensory/polyneuropathy problem have been selected in the present study. The average age of the selected patients in the study was 51.5± 11.2 year and the average duration of the disease was 8.6± 6.8 year. It was observed from the data that fasting glucose levels were significantly higher in DN than the healthy control group (Table 1). The result shows that the enzyme activity (SOD) in diabetic neuropathy is significantly higher (201.45±36.61 u/gm Hb) than control group (92 ± 52 u/gm Hb). When the patients were grouped based on duration of the disease, it was observed that no noticeable change was revealed in SOD activity when duration of the disease is less (<10 years) Figure 1. Whereas significant changes in SOD activity observed, with the increased duration of the disease (>10 years).

Table 1. Biochemical and general feature of the participant

	Control	DN patients
No. of subject	30	109
Age mean ± SD (year)	35± 9.2	51.57 ± 11.20
Gender Male /Female	20/10	70/39
Duration of Diabetic mean ± SD (year)	--	8.6 ± 6.8
Impaired fasting glucose test mean ± SD (mg/dl)	--	169 ±73.52
SOD Activity ± SD (unit /mg Hb)	92 ± 52	201.45 ±36.61

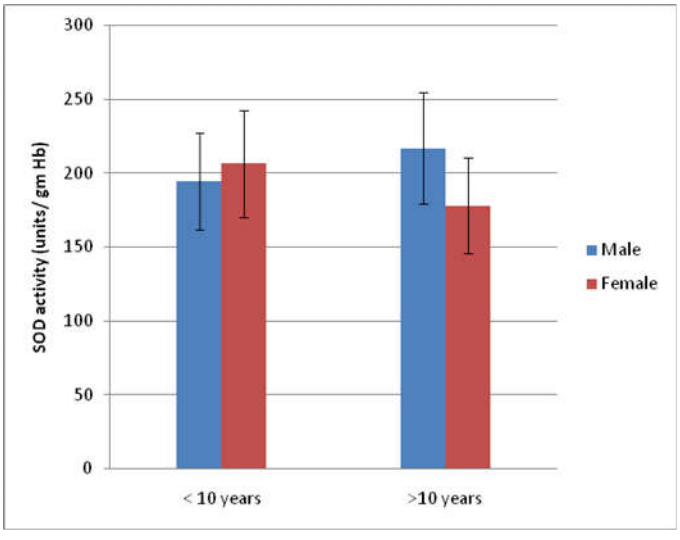

Figure 1. SOD activity with respect to duration of the disease between male and female diabetic neuropathy patients

DISCUSSION

Hyperglycaemia-induced oxidative and nitrosative stress has been singled out as one of the major links between diabetes and diabetic complications (Vincent 2008). Hyperglycaemia leads to generation of free radicals due to autoxidation of glucose and glycosylation of proteins (Negi *et al.,* 2011). The persistent increase in reactive oxygen species (ROS) and reactive nitrogen species (RNS) accompanied by a decrease in antioxidant activity leads to the occurrence of oxidative and nitrosative stress which can cause endothelial dysfunction, insulin resistance, and alterations in number and functions of pancreatic β cells and eventually leads to diabetic microvascular and macrovascular complications (Al-Faris 2010). Increased free-radical formation and/or a defect in antioxidant defenses which result in oxidative stress have been implicated in the pathogenesis of diabetic neuropathy (Bandeira *et al.,* 2013). Diabetic neuropathies are heterogeneous and affect different parts of the nervous system with various clinical manifestations (Ziegler 2004). Antioxidants are available endogenously as a normal defense mechanism of the cell or obtained exogenously from diet. The superoxide dismutase activities in patients with *diabetes mellitus* (DM) were assayed in the present study in order to evaluate its usefulness for monitoring and also evaluate the relation between SOD activities and neuropathy. The data shows a positive correlation between glucose concentration and super oxide dismutase. The results of the present study is in tune with the observation of Kharazi-Nejad *et al.,* (2014) where in it is mentioned that this increased SOD activity culminated as a result of stress produced by high concentration of free radicals due to hyperglycemia. The results are also in agreement with the others where in they demonstrated a relationship between the glucose concentration and enzyme activity (Thomas, 2014). The present study shows that the enzyme activity is significantly higher in the patients having diabetic neuropathy than the control group. Moreover, a significant difference was reveled in male and female patients when duration of the disease (>10 years) was considered. Future studies are warranted to further understand the relationships and mechanism involved in the duration or modulation of diabetic neuropathy patients.

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