IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 2 Ser. 13 (February. 2019), PP 49-54
www.iosrjournals.org

Study of lipid profile, lipid peroxidase, glutathione peroxidase, superoxide dismutase and catalase inDiabetes and non-diabetic foot ulcer patients

Dr.Prasanta Kumar Bhattacharyya [1],PriyankaBiswas [2], Dr.Debarshi Jana [3], Dr.Jayanta Mukherjee [1], Dr.Madhusanta De[2]

[1] *KPC Medical College & Hospital, Kolkata*
[2] *Ramakrishna Mission SevaPratishthan, Kolkata*
[3]*Institute of Post-Graduate Medical Education and Research, A.J.C. Bose Road, Kolkata-700020, West Bengal, India*
Corresponding Authors: Dr.Debarshi Jana

Abstract
Oxidative stress (OS) and lipid peroxidation has been associated in the development of diabetic complications including diabetic foot ulcer. In this study, the levels of lipid peroxides (LPO), catalase and lipid profile as well as the enzymatic antioxidant activities of superoxide dismutase (SOD) and glutathione peroxidase (GPx) in type 2 diabetes mellitus and diabetic foot ulcer subjects were assessed and compared with apparently healthy normal subjects to understand the involvement of OS in the subjects.
It was found that level of mean TC, TC and LDL had decreased in type 2 DM with foot ulcer, type2 DM without foot ulcer, non-diabetic foot ulcer and healthy control respectively and this difference was statistically significant. Level of mean HDL had significantly lower in type 2 DM with foot ulcer.Mean lipid peroxidase level had significantly higher in type2 DM with foot ulcer and non-diabetic foot ulcer ($p<0.0001$). Mean SOD level had significantly higher in type2 DM with foot ulcer and non-diabetic foot ulcer ($p<0.0001$). Mean Catalase level had significantly higher in type2 DM with foot ulcer and non-diabetic foot ulcer ($p=0.0135$).
Increased lipid profile subsequent to diabetic conditions of foot ulcer induces an over-expression of lipid peroxidase, SOD and Catalase activity suggesting a compensatory mechanism by the body to prevent further tissue damage in the subjects.
Key Words: *Oxidative stress, Lipid peroxidation, lipid profile, Diabetes, non-diabetic foot ulcer.*

Date of Submission: 13-02-2019 Date of acceptance: 28-02-2019

I. Introduction

Diabetes and Diabetic foot ulcer (DFU) pose a major threat to the public health throughout the world.[1] India is the (a) country with the largest number of diabetic patients in the world. Diabetic foot ulcer is a major disabling complication of diabetes which often precedes amputation of the limb.[2] According to the Global Lower Extremity Amputation Study Group, 25-90% of all amputations were associated with diabetes. [3] Considering the large population and high occurrence of diabetes in India, the burden of its difficulty would become enormous. Diabetic foot ulcer is one of the common causes of hospital admissions among diabetics in India. [1]

The DFU requires a long healing time and a multidisciplinary therapy, such as control of blood sugar levels, daily treatment of wounds, proper antibiotic therapy, and surgical revascularization. The ulcer can worsen and lead to amputation of affected lower extremity, estimated 85% cases in Unites States.[4,5] In addition; patients require a considerable cost to treat DFU, disturbance of daily activities, psychological, social, and quality of life. Optimal therapy management can accelerate wound healing and reduce other complications of diabetes mellitus.[4] The standard treatments of DFU were blood glucose regulation, use of antibiotics, ulcer debridement, wound care, offloading (no load or pressure), and improved blood flow or revascularization.[5]

The therapy can repair the hypoxic tissue, increase perfusion, reduce edema, decrease inflammatory cytokines, increase fibroblast proliferation, increase collagen production, and promote angiogenesis by the activity of reactive oxygen species (ROS). [5,6] Increase of ROS will improve the regulation of antioxidant enzyme activity of tissue.[5] Obesity animal model showed Hyperbaric oxygen therapy (HBOT)could effect of profile lipid, by increasing low-density lipoprotein (LDL) and triglyceride cholesterol and decreasing high-density lipoprotein (HDL) and total cholesterol.[7] This study was aimed to various biochemical parameters like triglyceride cholesterol (TG), Total cholesterol (TC), HDL and LDL at admission of diabetic foot ulcer patients

who were admitted in our hospital in Kolkata. We had also correlated with Lipid peroxidase, Glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) activities in Diabetes and Non-Diabetic foot ulcer patients.

II. Materials And Methods

This prospective study was conducted at Hospital based K.P.C. Medical College & Hospital, Jadavpur and Ramakrishna Mission SevaPratishthan, Kolkata from Jan 2015- Dec 2018. We had selected 50 patients with Type2 DM with foot ulcer, 50 patients with non-diabetic foot ulcer, 50 patients with type2 DM without foot ulcer and 50 patients with healthy control in definite Inclusion and exclusion criteria.

Estimation of Lipid profiles and Enzymes: All tests were done by semi auto analyzer by ERBA Chem - 5 Plus V2 by TRANSASIA using spectrophotometry principle. Lipid profile includes flowing parameters estimation of serum Total Cholesterol (TC), Triglyceride (TG), High density lipoprotein cholesterol (HDL), Low density lipoprotein cholesterol (LDL) and Very low density lipoprotein cholesterol (VLDL). Cholesterol and its esters are released from lipoproteins by detergents. Cholesterol esterase hydrolyses the esters. In the subsequent oxidation by cholesterol oxidase, hydrogen peroxide (H2O2) is liberated. The colorimetric indicator is quinoneimine is generated from 4-aminoantipyrine and phenol by H2O2 under the catalytic action of peroxidase (Trinder's reaction). TG reacts with water to form glycerol and fatty acid in the presence of lipoprotein lipase. The glycerol formed reacts with adenosine tri-phosphate (ATP) in the presence of glycerol kinase to form glycerol-3-P and adenosine di-phosphate (ADP). The glycerol-3-P reacts with oxygen to form dihydroxyacetone phosphate and hydrogen peroxide in the presence of glycerol-3-P oxidase. Hydrogen peroxide reacts with 4-aminoantipyrine to form red quinine and water in the presence of enzyme peroxidase. The intensity of purple coloured complex formed during the reaction is directly proportional to the triglyceride concentration in the sample and is measured at 546 nm. HDL and LDL were estimated by apospecific antibody mediated immunoturbidimetry.

Lipid peroxidase, Glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) had measured by standard procedure.

Statistical Analysis: For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS (version 24.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. One-way analysis of variance (one-way ANOVA) was a technique used to compare means of three or more samples for numerical data (using the F distribution). Unpaired proportions were compared by Chi-square test or Fischer's exact test, as appropriate.p-value ≤ 0.05 was considered for statistically significant.

III. Results And Analysis

We found that in type2 DM with foot ulcer, the mean age (mean±s.d.) of patients was 54.6200± 10.8438 years. In non-diabetic foot ulcer, the mean age (mean±s.d.) of patients was 42.3600± 13.4661 years. In type2 DM without foot ulcer, the mean age (mean±s.d.) of patients was 50.5000± 11.3986 years. In healthy control, the mean age (mean±s.d.) of patients was 45.1800± 15.2472 years. Distribution of mean age vs. group was statistically significant ($p<0.0001$).

It was found that association of sex vs. group was statistically significant ($p<0.0001$). We found that in type2 DM with foot ulcer, higher number of patients 16(32.0%) had house wife. In non-diabetic foot ulcer, higher number of patients 28(56.0%) had house wife. In type2 DM without foot ulcer, higher number of patients 26(52.0%) had house wife. In healthy control, higher number of patients 29(58.0%) had house wife. Association of occupation vs. group was not statistically significant ($p=0.0002$).

It was found that in type 2 DM with foot ulcer, the mean TC (mean±s.d.) of patients was 274.7400 ± 7.6740.In non-diabetic foot ulcer, the mean TC (mean±s.d.) of patients was 224.9600 ± 5.4845.In type2 DM without foot ulcer, the mean TC (mean±s.d.) of patients was 243.8400 ± 18.4207.In healthy control, the mean TC (mean±s.d.) of patients was 178.9800 ± 5.0689.Distribution of mean TC vs. group was statistically significant ($p<0.0001$). We found that in type2 DM with foot ulcer, the mean TG (mean±s.d.) of patients was 247.6600 ± 21.9894.In non-diabetic foot ulcer, the mean TG (mean±s.d.) of patients was 197.2600 ± 21.9432.In type2 DM without foot ulcer, the mean TG (mean±s.d.) of patients was 209.0800 ± 21.7423.In healthy control, the mean TG (mean±s.d.) of patients was 128.8600 ± 4.9693.Distribution of mean TG vs. group was statistically significant ($p<0.0001$).It was found that in type2 DM with foot ulcer, the mean HDL (mean±s.d.) of patients was 27.3400 ± 4.2166.In non-diabetic foot ulcer, the mean HDL (mean±s.d.) of patients was 37.5600 ± 5.6176.In type2 DM without foot ulcer, the mean HDL (mean±s.d.) of patients was 31.0000 ± 5.8519.In healthy control, the mean HDL (mean±s.d.) of patients was 63.6200 ± 8.3076.Distribution of mean HDL vs. group was statistically significant ($p<0.0001$).It was found that in type2 DM with foot ulcer, the mean LDL (mean±s.d.) of patients was 178.6600 ± 4.0839.In non-diabetic foot ulcer, the mean LDL (mean±s.d.) of patients was 147.3800 ± 24.7789.In type2 DM without foot ulcer, the mean LDL (mean±s.d.) of patients was 162.6200 ± 17.6749.In

healthy control, the mean LDL (mean±s.d.) of patients was 81.2600 ± 6.7122.Distribution of mean LDL vs. group was statistically significant (p<0.0001).

It was found that in type2 DM with foot ulcer, the mean lipid peroxidase (mean±s.d.) of patients was 10.0962 ± 5.8684 nM/ml. In non-diabetic foot ulcer, the mean lipid peroxidase (mean±s.d.) of patients was 7.8310 ± 2.0075 nM/ml.In type2 DM without foot ulcer, the mean lipid peroxidase (mean±s.d.) of patients was 5.1203 ± 3.2438 nM/ml.In healthy control, the mean lipid peroxidase (mean±s.d.) of patients was 3.0972 ± 1.3517 nM/ml.Distribution of mean lipid peroxidase vs. group was statistically significant (p<0.0001).We found that in type2 DM with foot ulcer, the mean GPx (mean±s.d.) of patients was 2665.1124 ± 738.6879 U/ml.In non-diabetic foot ulcer, the mean GPx (mean±s.d.) of patients was 2668.3600 ± 752.4394 U/ml.In type2 DM without foot ulcer, the mean GPx (mean±s.d.) of patients was 2517.9236 ± 411.6788 U/ml.In healthy control, the mean GPx (mean±s.d.) of patients was 2413.2708 ± 29.5731 U/ml.Distribution of mean GPx vs. group was not statistically significant (p=0.0703).It was found that in type2 DM with foot ulcer, the mean SOD (mean±s.d.) of patients was 170.1680 ± 32.0413 U/ml.Innon diabetic foot ulcer, the mean SOD (mean±s.d.) of patients was 158.1754 ± 51.7318 U/ml.In type2 DM without foot ulcer, the mean SOD (mean±s.d.) of patients was 142.3636 ± 47.9840 U/ml.In healthy control, the mean SOD (mean±s.d.) of patients was 133.6424 ± 22.3237 U/ml.Distribution of mean SOD vs. group was statistically significant (p<0.0001).We found that in type2 DM with foot ulcer, the mean Catalase (mean±s.d.) of patients was 5507.2318 ± 109.8146 U/min/ml.In non-diabetic foot ulcer, the mean Catalase (mean±s.d.) of patients was 5498.9928 ± 140.4437 U/min/ml.In type2 DM without foot ulcer, the mean Catalase (mean±s.d.) of patients was 5489.8886 ± 132.4173 U/min/ml.In healthy control, the mean Catalase (mean±s.d.) of patients was 5435.1132 ± 93.3506 U/min/ml. Distribution of mean Catalase vs. group was statistically significant (p=0.0135).

IV. Discussion

Diabetic foot ulcer is the most common complication of diabetes mellitus. The lifetime prevalence of foot ulceration is about 15%. [8] Macro and microvascular involvement and neuropathy plays a major role in the pathophysiology of diabetic foot ulcers. [9] According to the Diabetes Atlas 2013 published by the International Diabetes Federation, the number of people with diabetes in India currently is 65.1 million, which is expected to rise to 142.7 million by 2035. [10] Mean age of the study population was 51 years, which is in par with the previous studies in India. [1,11]

Regarding lipid profile, the level of serum cholesterol, triglycerides and LDL are higher in this group of patients. This finding can be correlated to the fact that being a metabolic disorder diabetes mellitus causes altered protein and lipid metabolism and thereby favors the disease progression. Mean serum cholesterol level in our patients was higher as compared to the data from a recent multicentric study from India. [11] These findings demand the need of education among diabetics regarding the risk factors in our area. Some studiesshow that majority of the patients with diabetic foot ulcer are males and with age of more than 40years.This finding is also similar to that in the previous literature. [1,11]

We found that in mean age was higher in type2 DM with foot ulcer patients than others and that had statistically significant (p<0.0001).Present study found that male had more prevalence in Type2 DM with Foot Ulcer and it was statistically significant (p<0.0001). In type2 DM with foot ulcer, higher number of patients 16(32.0%) had house wife. In non-diabetic foot ulcer, higher number of patients 28(56.0%) had house wife. In type2 DM without foot ulcer, higher number of patients 26(52.0%) had house wife. In healthy control, higher number of patients 29(58.0%) had house wife. Association of occupation vs. group was not statistically significant (p=0.0002).

Semadi IN et al [12] found that there was significant increase of LDL cholesterol levels (p = 0.009), HDL cholesterol levels (p = 0.002), and total cholesterol levels (p = 0.023) after therapy in Hyperbaric oxygen therapy group, but not in control group. The difference of all blood glucose parameters was not significant (p > 0.05). However, the difference in LDL cholesterol, HDL cholesterol, and total cholesterol was significantly different (p < 0.05).

It was found that level of mean TC had decreased in type 2 DM with foot ulcer, type2 DM without foot ulcer, non-diabetic foot ulcer and healthy control respectively and this difference was statistically significant (p<0.0001).We found that level of mean TG had decreased in type 2 DM with foot ulcer, type2 DM without foot ulcer, non-diabetic foot ulcer and healthy control respectively and this difference was statistically significant (p<0.0001). It was found that level of mean HDL had increased in type 2 DM with foot ulcer, type2 DM without foot ulcer, non-diabetic foot ulcer and healthy control respectively and this difference was statistically significant (p<0.0001). Level of mean HDL had significantly lower in type 2 DM with foot ulcer.It was found that level of mean LDL had decreased in type 2 DM with foot ulcer, type2 DM without foot ulcer, non-diabetic foot ulcer and healthy control respectively and this difference was statistically significant (p<0.0001). Level of mean LDL had significantly higher in type 2 DM with foot ulcer.

A study by Tsuneyama et al [13] in animal model obtained increased in triglyceride, increased in LDL cholesterol, decreased in HDL, and decreased in total cholesterol in HBOT group when compared with non-HBOT group, but these results were not significant. The results were in contrast with one study; we found significant increase of LDL cholesterol levels (p = 0.009), significant increase of HDL cholesterol levels (p = 0.002), and significant increase of total cholesterol levels (p = 0.023), but not significant decreased of triglyceride cholesterol. [12]The lipid may be migrated into the blood and stored in hepatocellular, which can cause organ damage.[13] Patient with metabolic syndrome had a complex reaction with HBOT, since the increase in oxygen may improve tissue oxygen, increasing proliferation, higher wound healing, and kill anaerobic bacteria. However, the side effect of an increase in oxygen was higher oxidative stress in tissue. Further investigation must be conducted to reduce the adverse effect of oxidative stress.

Bolajoko EB et al [14] found that significant elevated values of LPO (39.86%) and 8-OHdG (45.53%) were found in DM subjects compared with the NC subjects. This increase in both parameters was greater for DF subjects: 80.23% and 53.91% respectively. SOD activities were significantly reduced in DM (14.82%) and DF (4.09%) subjects in contrast with elevated activities of GPx observed in DM (21.87%) and DF (20.94%) subjects. Glycated haemoglobin/fasting plasma glucose (HbA1c/FPG) correlated positively with LPO, 8-OHdG and GPx, whereas a negative correlation was observed for SOD.

It was found that in type2 DM with foot ulcer, the mean lipid peroxidase (mean±s.d.) of patients was 10.0962 ± 5.8684 nM/ml. In non-diabetic foot ulcer, the mean lipid peroxidase (mean±s.d.) of patients was 7.8310 ± 2.0075 nM/ml.In type2 DM without foot ulcer, the mean lipid peroxidase (mean±s.d.) of patients was 5.1203 ± 3.2438 nM/ml.In healthy control, the mean lipid peroxidase (mean±s.d.) of patients was 3.0972 ± 1.3517 nM/ml. Mean lipid peroxidase level had significantly higher in type2 DM with foot ulcer and non-diabetic foot ulcer ($p<0.0001$).Level of GPx had higher in type2 DM and non-diabetic foot ulcer patients but this association was not statistically significant ($p=0.0703$). It was found that in type2 DM with foot ulcer, the mean SOD (mean±s.d.) of patients was 170.1680 ± 32.0413 U/ml.In non-diabetic foot ulcer, the mean SOD (mean±s.d.) of patients was 158.1754 ± 51.7318 U/ml.In type2 DM without foot ulcer, the mean SOD (mean±s.d.) of patients was 142.3636 ± 47.9840 U/ml.In healthy control, the mean SOD (mean±s.d.) of patients was 133.6424 ± 22.3237 U/ml. Mean SOD level had significantly higher in type2 DM with foot ulcer and non-diabetic foot ulcer ($p<0.0001$).We found that in type2 DM with foot ulcer, the mean Catalase (mean±s.d.) of patients was 5507.2318 ± 109.8146 U/min/ml.In non-diabetic foot ulcer, the mean Catalase (mean±s.d.) of patients was 5498.9928 ± 140.4437 U/min/ml.In type2 DM without foot ulcer, the mean Catalase (mean±s.d.) of patients was 5489.8886 ± 132.4173 U/min/ml.In healthy control, the mean Catalase (mean±s.d.) of patients was 5435.1132 ± 93.3506 U/min/ml. Mean Catalase level had significantly higher in type2 DM with foot ulcer and non-diabetic foot ulcer ($p=0.0135$).

Lipid peroxidation had a relationship with high blood glucose and high oxidative stress in diabetic patients. In a clinical study, there was a significant association of higher lipid peroxidation with high fasting blood glucose and high HbA1c levels.[15]

V. Conclusion

Increased lipid oxidation subsequent to diabetic conditions of foot ulcer induces an over-expression of lipid peroxidase, SOD and Catalase activity suggesting a compensatory mechanism by the body to prevent further tissue damage in the subjects. We concluded that significant increase of lipid profile may be caused by the reaction of lipid peroxidation. Increased of lipid profile can be the side effect of diabetic foot ulcer and further multicentre research to study disadvantages of increased in lipid profile is required.

Reference

[1]. Vijay V, Snehalatha C, Ramachandran A. Socio-cultural practices that may affect the development of the diabetic foot. IDF BULLETIN. 1997;42:10-13.
[2]. Assal JP, Groop L. Definition, diagnosis and classification of diabetes mellitus and its complications. World Health Organization. 1999:1-65.
[3]. Li X, Xiao T, Wang Y, Gu H, Liu Z, Jiang Y, Liu Y, Lu Z, Yang X, Lan Y, Xu Z. Incidence, risk factors for amputation among patients with diabetic foot ulcer in a Chinese tertiary hospital. Diabetes research and clinical practice. 2011 Jul 31;93(1):26-30.
[4]. Wounds International. International Best Practice Guidelines: Wound Management in Diabetic Foot Ulcers. London: Wounds International A division of Schofield Healthcare Media Limited Enterprise House; 2013. p. 1-23.
[5]. Irawan H, Kartika. Terapioksigenhiperbariksebagaiterapiadjuvan kaki diabetik.CerminDunia Kedokteran-245. 2016;43:782-5.
[6]. Bhutani S, Vishwanath G. Hyperbaric oxygen and wound healing. Indian J Plast Surg. 2012;45:316-24.
[7]. Yasuda K, Adachi T, Gu N, Matsumoto A, Matsunaga T, Tsujimoto G, et al. Effects of hyperbaric exposure with high oxygen concentration on glucose and insulin levels and skeletal muscle-fiber properties in diabetic rats. Muscle Nerve. 2007;35:337-43.
[8]. Reiber GE, Boyko EJ, Smith DG. Lower extremity foot ulcers and amputations in diabetes.Diabetes in America. 1995 Jul 1;2:40927.
[9]. Stockl K, Vanderplas A, Tafesse E, Chang E. Costs of lower-extremity ulcers among patients with diabetes. Diabetes Care. 2004 Sep 1;27(9):2129-34.
[10]. Whiting DR, Guariguata L, Weil C, Shaw J. IDF diabetes atlas: 6.global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res Clin Pr. 2013;94(3):311-21

[11]. Viswanathan V, Thomas N, Tandon N, Asirvatham A, Rajasekar S. Profile of diabetic foot complications and its associated complications-a multicentric study from India. JAPI. 2005 Nov 7;53:933-6.
[12]. Semadi IN, Irawan H. Blood glucose and lipid profile in patients with diabetic foot ulcer that underwent hyperbaric oxygen therapy. BALI MEDICAL JOURNAL. 2017 Jan 1;6(2):405-8.
[13]. Tsuneyama K, Chen Y-C, Fujimoto M, Sasaki Y, Suzuki W, Shimada T, et al. Advantages and disadvantages of hyperbaric oxygen treatment in mice with obesity hyperlipidemia and steatohepatitis. TheScientificWorldJOURNAL. 2011;11:2124-35.
[14]. Bolajoko EB, Mossanda KS, Adeniyi F, Akinosun O, Fasanmade A, Morapane M. Antioxidant and oxidative stress status in type 2 diabetes and diabetic foot ulcer. South African Medical Journal. 2008;98(8):614-7.
[15]. Tiwari BK, Pandey KB, Abidi AB, Rizvi SI. Markers of oxidative stress during diabetes mellitus.Journal of Biomarkers. 2013;2013:1-8.

Table: Distribution of mean lipid profile in four groups

		Number	Mean	SD	Minimum	Maximum	Median	p-value
TC	Type2 DM with foot ulcer	50	274.7400	7.6740	260.0000	289.0000	276.0000	<0.0001
	Non diabetic foot ulcer	50	224.9600	5.4845	206.0000	231.0000	226.0000	
	Type2 DM without foot ulcer	50	243.8400	18.4207	220.0000	271.0000	240.0000	
	Healthy Control	50	178.9800	5.0689	163.0000	190.0000	177.5000	
TG	Type2 DM with foot ulcer	50	247.6600	21.9894	220.0000	289.0000	246.0000	<0.0001
	Non diabetic foot ulcer	50	197.2600	21.9432	174.0000	231.0000	182.0000	
	Type2 DM without foot ulcer	50	209.0800	21.7423	174.0000	231.0000	220.0000	
	Healthy Control	50	128.8600	4.9693	120.0000	139.0000	129.5000	
HDL	Type2 DM with foot ulcer	50	27.3400	4.2166	21.0000	36.0000	27.0000	<0.0001
	Non diabetic foot ulcer	50	37.5600	5.6176	25.0000	46.0000	38.0000	
	Type2 DM without foot ulcer	50	31.0000	5.8519	21.0000	45.0000	31.0000	
	Healthy Control	50	63.6200	8.3076	51.0000	79.0000	64.0000	
LDL	Type2 DM with foot ulcer	50	178.6600	4.0839	174.0000	190.0000	177.0000	<0.0001
	Non diabetic foot ulcer	50	147.3800	24.7789	120.0000	190.0000	132.0000	
	Type2 DM without foot ulcer	50	162.6200	17.6749	130.0000	182.0000	174.0000	
	Healthy Control	50	81.2600	6.7122	70.0000	92.0000	81.0000	

Table: Distribution of mean Lipid peroxidase, Glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT)in four groups

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Lipid peroxidase (nM/ml)	Type2 DM with foot ulcer	50	10.0962	5.8684	0.2280	25.0150	10.2175	<0.0001
	Non diabetic foot ulcer	50	7.8310	2.0075	1.7190	10.4650	8.1530	
	Type2 DM without foot ulcer	50	5.1203	3.2438	1.9230	20.0000	4.4600	
	Healthy Control	50	3.0972	1.3517	0.8330	6.9240	2.9450	
GPX (U/ml)	Type2 DM with foot ulcer	50	2665.1124	738.6879	2355.0000	6452.0000	6452.0000	0.0703
	Non diabetic foot ulcer	50	2668.3600	752.4394	2000.0000	6450.0000	6450.0000	
	Type2 DM without foot ulcer	50	2517.9236	411.6788	2000.0000	5015.0000	5015.0000	
	Healthy Control	50	2413.2708	29.5731	2352.0000	2446.0000	2446.0000	
SOD (U/ml)	Type2 DM with foot ulcer	50	170.1680	32.0413	106.7000	254.0000	168.0000	<0.0001
	Non diabetic foot ulcer	50	158.1754	51.7318	89.8800	279.4000	152.8800	
	Type2 DM without foot ulcer	50	142.3636	47.9840	81.2000	279.4000	141.1200	
	Healthy Control	50	133.6424	22.3237	89.9000	166.3200	137.6600	
Catalase (U/min/ml)	Type2 DM with foot ulcer	50	5507.2318	109.8146	5185.0100	5725.2200	5493.3400	0.0135
	Non diabetic foot ulcer	50	5498.9928	140.4437	5069.9300	5779.4100	5488.9850	
	Type2 DM without foot ulcer	50	5489.8886	132.4173	5069.9300	5779.4100	5492.2550	
	Healthy Control	50	5435.1132	93.3506	5185.0100	5580.1900	5460.7800	

Table: Association of Sex and occupationin four groups

		Type2 DM with Foot Ulcer	NON DIABETIC FOOT ULCER	Type2 DM without Foot Ulcer	HEALTHY CONTROL	Chi-square value	p-value
Sex	**Female**	17	38	35	34	22.9202	<0.0001
	Row %	13.7	30.6	28.2	27.4		
	Col %	34.0	76.0	70.0	68.0		
	Male	33	12	15	16		
	Row %	43.4	15.8	19.7	21.1		
	Col %	66.0	24.0	30.0	32.0		
Occupation	**Business**	9	0	5	1	61.7590	0.0002
	Row %	60.0	0.0	33.3	6.7		
	Col %	18.0	0.0	10.0	2.0		
	Farmer	4	8	7	7		
	Row %	15.4	30.8	26.9	26.9		
	Col %	8.0	16.0	14.0	14.0		
	House wife	16	28	26	29		
	Row %	16.2	28.3	26.3	29.3		
	Col %	32.0	56.0	52.0	58.0		
	Mechanic	0	2	1	3		
	Row %	0.0	33.3	16.7	50.0		
	Col %	0.0	4.0	2.0	6.0		
	office worker	2	0	0	0		
	Row %	100.0	0.0	0.0	0.0		
	Col %	4.0	0.0	0.0	0.0		
	Private company worker	3	2	0	1		
	Row %	50.0	33.3	0.0	16.7		
	Col %	6.0	4.0	0.0	2.0		
	Retd	10	0	1	2		
	Row %	76.9	0.0	7.7	15.4		
	Col %	20.0	0.0	2.0	4.0		
	Service	3	4	6	0		
	Row %	23.1	30.8	46.2	0.0		
	Col %	6.0	8.0	12.0	0.0		
	Tailoring	0	2	0	1		
	Row %	0.0	66.7	0.0	33.3		
	Col %	0.0	4.0	0.0	2.0		
	Works in field	3	4	4	6		
	Row %	17.6	23.5	23.5	35.3		
	Col %	6.0	8.0	8.0	12.0		

Dr.Debarshi Jana. “Study of lipid profile, lipid peroxidase, glutathione peroxidase, superoxide dismutase and catalase inDiabetes and non-diabetic foot ulcer patients.” IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), Vol. 18, no. 02, 2019, pp. 49-54.