Journal of Academy of Biomedical Sciences (JABS)
An official Journal of **Indian Academy of Biomedical Sciences (IABS)**
www.iabsjournal.com

Original Article
Published : 10th October 2025
Volume 2 | Issue 1

Assessment of Oxidative Stress Related Parameters in the Development of Diabetes and Diabetic Nephropathy

Sanchit Tiwari[1], Israr Ahmad[2,3,] Priyanka Agrahari[1], Satyendra Kumar Sonkar[4], Gyanendra Kumar Sonkar[2], Abbas Ali Mahdi[2], Mohammad Kaleem Ahmad[2]

[1]*Department of Biochemistry, Maharshi Vishwamitra Autonomous State Medical College Ghazipur, UP, India.*
[2]*Department of Biochemistry, King George's Medical University, Lucknow, UP, India.*
[3]*Deapartment of Biotechnology, Era University, Lucknow, UP, India.*
[4]*Department of Medicine, King George's Medical University, Lucknow, UP, India.*

Abstract

Background: The primary cause of end-stage renal disease is diabetic nephropathy (DN). Type 2 diabetes mellitus (T2DM) and the development of diabetic nephropathy are significantly influenced by reactive oxygen species (ROS). Assessing the levels of oxidative and antioxidative markers in T2DM and DN patients in the North Indian community is the aim of our current investigation. Type 2 diabetes patients and those with nephropathy were used as controls to measure the levels of Catalase (CAT), lipid peroxides (LPO), superoxide dismutase (SOD), reduced glutathione (GSH), glutathione peroxidase (GPx), glutathione reductase (GR), and protein carbonyl content.

Results: SOD and CAT activity was considerably lower in DN than in controls. Patients with diabetes and nephropathy had considerably higher levels of lipid peroxidation and protein carbonyl content than controls. Compared to the control group, type 2 diabetes patients with and without nephropathy showed significantly lower levels of GSH, GPx, and GR activity.

Conclusion: Our research revealed that higher LPO levels might be causing cellular antioxidant enzyme levels to drop, which would contribute to worse OS in T2DM patients with nephropathy as opposed to those with simple T2DM. As a result, OS might be an essential mediator in the pathophysiology of diabetic nephropathy development.

***CORRESPONDENCE:**
Mohammad Kaleem Ahmad
(mohdkaleemahmad@kgmcindia.edu)

KEYWORDS:
Diabetic nephropathy, Lipid peroxidation, Superoxide dismutase, Type 2 diabetes.

HOW TO CITE:
Tiwari S, Ahmad I, Agrahari P, Sonkar SK, Sonkar GK, Mahdi AA, Ahmad MK. Assessment of Oxidative Stress Related Parameters in the Development of Diabetes and Diabetic Nephropathy. Journal of Academy of Biomedical Sciences. 2025;2(1):10-15

doi: 10.61081/jabs/2v1i103

INTRODUCTION

In modern India, type 2 diabetes mellitus (T2DM) is one of the most serious public health issues, currently affecting approximately, 62.4 million individuals. Projections by the International Diabetes Federation (IDF) estimate this figure will rise to nearly 100 million by 2030.[1] Chronic kidney disease (CKD) is one of the main side effects of type 2 diabetes, and in 20–30% of diabetics, diabetic nephropathy (DN) is the main cause of end-stage renal disease (ESRD).[2,3] Genetic predisposition, persistent hyperglycemia, activation of the polyol and protein kinase C pathways, stimulation of the renin-angiotensin system, increased formation of advanced glycation end products (AGEs), glomerular hyperfiltration, and elevated reactive oxygen species (ROS) are some of the factors that contribute to the onset and progression of diabetic nephropathy (DN).[4]

Oxidative stress parts a pivotal character in mediating many of the molecular mechanisms underlying DN pathogenesis. Chronic hyperglycemia and increased AGE accumulation contribute to redox imbalance, impacting both the renin-angiotensin mechanism and transforming growth factor-beta (TGF-β) signaling. This dysregulation promotes chronic inflammation, as well as structural changes such as glomerular and tubular hypertrophy. Mesangial cell growth, excessive extracellular matrix (ECM) deposition, thickening of glomerular and tubular membranes, podocyte dysfunction, and apoptosis are the main causes of renal fibrosis in DN. These pathological features culminate in clinical manifestations such as albuminuria, proteinuria, glomerulosclerosis, and tubulointerstitial fibrosis, all of which are driven by redox disturbances.[5]

Oxidative stress, characterized by the overproduction of free radicals and peroxides, disrupts cellular homeostasis and damages critical biomolecules including lipids, proteins, and DNA. This oxidative imbalance is attributed to either increased generation of oxidizing agents or a compromised

antioxidant defense system, notably involving glutathione (GSH).[6,7] Experimental data have shown that diabetic rats exhibit elevated nitric oxide levels and depleted GSH, alongside reduced actions of key antioxidant enzymes such as glutathione peroxidase (GPx) and glutathione-S-transferase.[8] According to Singh and Singh,[9] diminished GSH levels in diabetic patients result in reduced antioxidant enzyme function, thereby promoting oxidative injury and contributing to the pathogenesis of DN.

The current study search for to elucidate the role of oxidative stress in the progression of DN among Indian patients with diabetes, emphasizing the redox alterations that accompany disease advancement.

MATERIALS AND METHODS

Study population and study design: The study included 300 volunteer subjects in all, divided into three groups: 100 subjects with T2DM with nephropathy, 100 subjects with T2DM without nephropathy and 100 healthy subjects as healthy control. The institutional ethics committee gave its approval to the study protocol.

Type 2 Diabetes Mellitus Screening

Patients with T2DM were defined as having a HbA1c > 7%, a fasting blood sugar (FBS) level ≥ 125 mg/dL, and a postprandial blood sugar (PPBS) level ≥ 200 mg/dL.

Diabetic Nephropathy Screening

Serum creatinine, blood urea, and the amount of albumin excreted in a 24-hour urine sample have all been used to measure the presence of DN. Urinary excretion of albumin ≥30 mg/dL albumin in a 24-hour collection is considered significant albuminuria, and both micro and macroalbuminuria were present in our study participants. In a 24-hour collection, participants with macroalbuminuria had urinary excretion of ≥300 mg/dL of urinary albumin, whereas those with microalbuminuria had albumin 30–299 mg/dL.

Inclusion and Exclusion Criteria

The study excluded participants with autoimmune illnesses, liver and heart failure, allergies, eczema, thyroid abnormalities, infectious disease, and alcohol misuse. The study included participants with diabetes, diabetic patients with pre-renal failure, and diabetic patients with kidney failure.

Sample Collection

Following a 12-hour overnight fast, venous blood samples were taken from each participant. After putting the samples on ice, they were centrifuged for an hour at 3,500 rpm and 4°C for 15 minutes. The supernatants were then stored for later analysis at –80°C.

Biochemical Assay

Important variables include age, body mass index (BMI), HbA1c, urea level, serum creatinine, lipid profiles, uric acid, total protein, albumin protein, urine albumin test, and FBS and PPBS levels. All biochemical analyses were carried out using the Vitros 250 Autoanalyzer (Johnson & Johnson, Germany) using kits.

OXIDATIVE STRESS PARAMETERS

Estimation of lipid peroxides (LPO): by the procedure of Ohkawa *et al.*[10]

Catalase (CAT) measurement: by the procedure of Aebi and Suter.[11]

Estimation of Superoxide Dismutase (SOD): by the procedure of McCord and Fridovich[12]

Assay of GPx: by the procedure of Rotruck *et al.*, 1973.[13]

Estimation of GSH: by the procedure of Kuo *et al.*, 1983.[14]

Estimation of Glutathione Reductase (GR): by the procedure of Hazelton and Lang.[15]

Protein carbonyl group (PC): by method of Levine and Williams, 1994.[16]

Analysis of Statistics

Version 29.0 of IBM-SPSS was used for statistical analysis. The mean ± standard deviation (SD) was used to express the results. One-way analysis of variance (ANOVA) and post-hoc Tukey's test were used to compare the subject groups' demographic profiles and clinical characteristics. Pearson's correlation analysis was used to find the correlation between the various parameters. A *p-value* of less than 0.05 was deemed significant.

RESULTS

The baseline aspects of T2DM, DN and healthy controls are provided in table 1.

In this investigation, we found that DN patients were considerably older than controls. Whencompared to controls, T2DM and DN patients had noticeably higher levels of FBS, PPBS, HbA1C, TG, and uric acid. Additionally, DN patients had a significantly higher level of creatinine than controls, while T2DM patients had a much lower level. In contrast, DN patients had significantly lower levels of LDL, HDL, and albumin than controls. T2DM patients also had lower amounts of these substances, though not to a significant degree.

The results of oxidative stress markers estimation in T2DM, DN and controls are represented in table 2.

Lipid peroxidation levels were substantially greater in T2DM patients with DN (p <0.001) and T2DM patients with DN (p <0.001) than in controls. Both T2DN and DN patients had considerably lower SOD and CAT activity (p <0.001) than the healthy control. While GR levels were lower in diabetic patients and DN patients (p <0.01), the differences between the two groups and the healthy controls were not statistically significant. However, GSH, GPx level were significantly decrease in T2DM subjects and DN patient (p <0.001). In

Table 1: Clinical and anthropometric characteristics of T2DM and DN Subjects.

Variables	Control (*N* = 100)	Type 2 Diabetes (*N* = 100)	Diabetic Nephropathy (*N* = 100)
Duration Onset		10.02 ± 3.01	11.22 ± 6.4
Age	50.94 ± 10.18	53.48 ± 9.39	57.84 ± 9.5
BMI	26.05 ± 4.39	24.11 ± 5.69**	22.28 ± 4.03***
HbA1C	5.41 ± 0.59	9.72 ± 3.08***	8.22 ± 2.32***
FBS	94.66 ± 9.00	176.22 ± 85.25***	179.66 ± 67.65***
PPBS	127.81 ± 15.05	264.80 ± 106.27***	247.57 ± 82.41***
HDL	43.49 ± 13.94	39.30 ± 25.74	36.78 ± 23.19**
LDL	88.73 ± 32.51	79.39 ± 36.51	68.77 ± 40.40***
VLDL	26.51 ± 8.86	35.86 ± 28.56**	32.29 ± 22.06*
TG	132.41 ± 44.59	202.29 ± 149.37***	163.16 ± 109. 99**
Cholesterol	148.68 ± 29.86	147.38 ± 47.24	139.18 ± 44.86
Urea	28.92 ± 6.42	34.16 ± 23.29*	118.58 ± 63.39***
Creatinine	0.81 ± 0.12	0.72 ± 0.18***	4.69 ± 3.57***
ALP	93.07 ± 16.82	236.28 ± 213.97***	258.94 ± 187.89***
Uric Acid	4.5 ± 0.98	4.83 ± 1.05*	7.34 ± 2.27***
Total Protein	7.21 ± 0.55	6.60 ± 1.00***	6.40 ± 0.84***
Albumin Protein	4.01 ± 0.56	3.57 ± 0.68***	3.03 ± 0.65***
Urine Protein	3.42 ± 2.78	8.89 ± 5.71**	152.20 ± 83.55***

All data are shown as mean ± SD. $p < 0.05$ is considered statistically significant

Table 2: Comparisons of oxidative stress markers in T2DM, DM subjects and control.

S.No.	Variables	Healthy Control (*N* = 100)	Type 2 Diabetes (*N* = 100)	Diabetic Nephropathy (*N* = 100)
1	LPO (nmol MDA/ml)	2.13 ± 1.13	4.25 ± 1.039 ***	6.91 ± 1.16 ***
2	SOD (Unit/mg protein)	6.02 ± 2.92	4.18 ± 1.5 ***	3.08 ± 2.73 ***
3	Catalase (Unit/mg protein)	12.93 ± 5.53	11.80 ± 4.75	10.91 ± 4.36**
4	Glutathione reductase (Unit/min/ mg protein)	1.11 ± 0.51	1.07 ± 0.27	0.98 ± 0.14 **
5	Glutathione (GSH) ug/ml	3.46 ± 0.97	2.11 ± 0.53 ***	1.08 ± 0.46 ***
6	GPx (unit/min/mg protein)	47.46 ± 12.51	30.85 ± 6.13 ***	25.24 ± 6.03 ***
7	Protein carbonyl (nmol/mg protein)	0.08 ± 0.02	0.16 ± 0.07***	0.30 ± 0.05***

All data are shown as mean ± SD. p< 0.05 is considered statistically significant

addition, PC was considerably higher in DN and diabetic patients than in the control group (p <0.001).

In table 3, we analysed matrix correlation between, the different parameters of oxidative stress, and we found that GSH significantly positively correlated with GPx (R2 = 0.235148, $p = 0.025$) and PC level was positively related with GSH level (R2 = 0.208369, $p = 0.046$) in DN patients.

The correlations between clinical and oxidative stress parameters by Person's correlation analysis are presented in table 4.

In correlation study, we observed that HbA1c and FBS level was significantly positive correlated with GPx level (R2 = 0.0506, $p = 0.02$, R2 = 0.1991, $p = 0.04$) and GSH level (R2 = 0.0501, $p = 0.04$, R2 = 0.2204, $p = 0.02$) in DN subjects. Level of uric acid was negatively related with GR level (R2 = -0.1981, $p = 0.04$) and positively related with PC (R2 = 0.1072, $p = 0.05$). Creatinine level was significantly positively correlated with CAT level (R2 = 0.2438, $p = 0.01$), and negatively with GPx level (R2 = -0.3176, $p = 0.001$) in DN subjects. LDL level was positively associated with CAT level (R2 = 0.2056, $p = 0.04$) in DN subjects.

Table 3: Matrix correlation between, the different parameters of oxidative stress in DN subjects.

Variables	Catalase	SOD	LPO	GR	GPx	GSH	PC
Catalase	1						
SOD	-0.14262	1					
LPO	0.024367	0.107152	1				
GR	0.039142	-0.06765	-0.06007	1			
GPx	-0.33836	0.15762	0.054556	-0.158	1		
GSH	0.072921	0.036716	-0.17568	0.110219	0.235148** ($p = 0.025$)	1	
PC	0.07463	0.067113	0.283521	0.059307	0.095554	0.208369* ($p = 0.046$)	1

$p < 0.05$ is considered statistically significant.

Table 4: Correlation between the clinical and oxidative stress parameters.

Variables	R2 – Value	*p*-value
HbA1c vs SOD	0.2165	0.03
FBS vs GPx	0.1991	0.04
FBS vs GSH	0.2204	0.02
Uric Acid vs GR	-0.1981	0.04
Uric Acid vs PC	0.1972	0.05
Creatinine vs Catalase	0.2438	0.01
Creatinine vs GPx	-0.3176	0.001
LDL vs Catalase	0.2056	0.04

Person's correlation. $p < 0.05$ is considered statistically significant.

DISCUSSION

The disruption of the equilibrium between the production of ROS and the defensive mechanisms of antioxidants has been identified as oxidative stress.[17] Several redo-sensitive cell-signaling molecules are activated, and cytotoxic materials are generated as a result of the increased ROS generation brought on by the oxidative stress caused by hyperglycemia. Cellular damage and malfunction follow, which ultimately leads to diabetic micro and macrovascular problems.[18,19] We assessed the oxidative stress state in DN and T2DM in this investigation. The current study demonstrates that diabetes mellitus, primarily in DN patients, is associated with significant oxidative stress.

Free radicals attacked membrane lipids to start lipid peroxidation, which resulted in large quantities of reactive species that have been linked to diabetes and its consequences.[20] A significant increase level of lipid peroxidation was found in T2DM and DN patients compared with healthy controls. Our finding is accordance of previous study.[21,22] This significant higher MDA levels possibly will be increased generation of free radicals in diabetes mellitus patients. MDA, a byproduct of lipid peroxidation, may be in charge of collagen's intermolecular cross-linking, which stabilizes and advances glycation. As glycated collagen triggers more lipid peroxidation and releases extra MDA, a vicious cycle is initiated.[23]

In-vivo, a number of enzyme antioxidants, including SOD, CAT, and GPx, are crucial for protecting against lipid peroxidation. LPO is largely protected by phospholipid hydroperoxide and GPx, an intrinsic renal cytoprotective antioxidant enzyme that is typically expressed in glomerular podocytes, parietal epithelial cells, and tubular epithelial cells.[24] The oxidized form of the enzyme GPx was reduced by GSH, which in turn reduced the dangerous intracellular reactive species H2O2. The lowering of GPx activity in DN patients is hampered by the increased production of LPO and H2O2, which leads to a decreased use of GSH.[25] Patients with DN have high aldolase reductase pathway activity, which consumes NADPH and causes an imbalance in GR activity. Therefore, reduced GPx antioxidant enzyme was seen in patients with DN.[23] In our results, we have found significant decrease levels of Gpx, GSH and GR in DN patients. Our results are accordance with previous studies.[26-28] They are observed decreased levels of GPx, GSH and GR in DN patients compared with healthy individuals.

The first line of defense against the damaging effects of oxygen radicals within cells is SOD, an enzyme that scavenges superoxide radicals (O2-). The occurrence of SOD in numerous compartments of the body enables it to neutralize O2 - radicals promptly and protects the cells from oxidative harm.[28] DN and T2DM patients have considerably lower SOD levels than healthy controls. Our results are in good agreement with previous findings.[20,23] In T2DM patients with microalbuminuria, a substantial reduction in SOD activity may lead to an elevated flux of O2-radicals, which in turn may indicate tissue damage or injury. Progressive glycation of enzyme proteins is linked to the decline in SOD levels in type 2 diabetes. Trace activity results from the glycation of about 50% of SOD in diabetic patients' erythrocytes. Further decreased SOD activity in DN patients may result from increased excretion from inflammatory kidney in nephropathy and increased production of O2-radicals, which

are used in the autoxidation process.[29] When compared to healthy controls, CAT activity is lower in T2DM and DN patients. In patients with T2DM who also nephropathy had, CAT activity was shown to be considerably lower than in those who did not.[30] This observation is consistent with our results. Increased endogenous production of O2-anion, as shown by elevated MDA, increased nitric oxide end products, and decreased SOD activity, may be the cause of the decline in CAT activity. Additionally, patients with DN may have lower CAT activity because they are experiencing higher levels of oxidative stress.[23] A widely used and established indicator of severe oxidative protein damage was PC content. Moreover, protein oxidation is a useful indicator for evaluating oxidative stress in vivo. An early and reliable indicator of protein oxidation is the production of carbonyl groups. The PC concentration was consistent, produced quantifiable findings, and seemed to biologically significantly represent illness outcomes. DN and T2DM were discovered to have elevated PC levels.[31]

CONCLUSION

In our study, correlation analysis reveals that the level of oxidative stress, i.e., alteration in the levels of oxidants and antioxidants, is thus related to the development of DN which justifies the aim of our study. Our current study's findings indicated that oxidative stress was out of balance in patients with DN and T2DM. Additionally, microalbuminuria raised oxidative stress in T2DM patients, which may be the cause of the pathophysiology of DN development. Furthermore, further extensive research must be done to confirm the aforementioned conclusions.

Acknowledgments: The Council of Science and Technology, U.P. (UPCST), No.CST/SERPD/D-293, provided assistance for this work.

REFERENCES

1. Young BA, Maynard C, Boyko EJ (2003) Racial differences in diabetic nephropathy, cardiovascular disease, and mortality in a national population of veterans. Diabetes Care 26: 2392-2399.
2. Shahbazian H, Rezaii I. Diabetic kidney disease; review of the current knowledge. J Ren Inj Prev 2013;2(2):73e80.
3. Bouaziz A, Zidi I, Zidi N, Mnif W, Zinelabidine HT. Nephropathy following type 2 diabetes mellitus in Tunisian population. W Indian Med J 2012;61:881e9.
4. F. N. Ziyadeh, B. B. Hoffman, D. C. Han et al., "Long-term prevention of renal insufficiency, excess matrix gene expression, and glomerular mesangial matrix expansion by treatment with monoclonal antitransforming growth factor-β antibody in db/db diabetic mice," Proceedings of the National Academy of Sciences of the United States of America, vol. 97, no. 14, pp. 8015– 8020, 2000.
5. G. Manda, A.-I. Checherita, M. V. Comanescu, and M. E. Hinescu, "Redox signaling in diabetic nephropathy: hypertrophy versus death choices in mesangial cells and podocytes," Mediators of Inflammation, vol. 2015, Article ID 604208, 13 pages, 2015.
6. Schafer FQ, Buettner GR (2001) Redox environment of the cell as viewed through the redox state of the glutathione disulfide/ glutathione couple. Free Radic Biol Med 30:1191 – 212.
7. Waggiallah H, Alzohairy M (2011) The effect of oxidative stress on human red cells glutathione peroxidase, glutathione reductase level, and prevalence of anemia among diabetics. N Am J Med Sci 3:344–7.
8. Abdel-Moneim A, Abdel-Reheim ES, Abd El-Tawab SM, Yousef AI. Assessment of the ameliorative effect of p-coumaric acid and gallic acid on oxidative stress and hematological abnormalities in experimental type 2 diabetes. Gen Med Open 2018;2(6):5e6.
9. Singh K, Singh G. Alterations in some oxidative stress markers in diabetic nephropathy. J Cardiovasc Disease Res 2017;8(1):24e7.
10. Ohkawa H, Ohishi N, Yagi K (1979) Assay for lipid peroxides in animal tissue by thio-barbituric acid reaction. Anal Biochem 95: 258- 357.
11. Aebi H, Suter H (1974) Positive function of reduced glutathione (GSH) against the effect of peroxidative substances and of irradiation in the red cell. In: Flohe L, Benhar HC, editors. Glutathione. Stuttgart Georg Thieme 192- 9.
12. McCord JM, Fridovich I (1969) SOD enzyme function for erythrocuprein. J Biol Chem 224:6049 - 55.
13. Rotruck JT, Pope AL, Ganther HE, Swanson AB, Hafeman DG, Hoekstra WG (1973) Selenium: biochemical role as a component of glutathione peroxidase. Science 179(4073):588-90.
14. Kuo CH, Maita K, Sleight SD, Hook JB (1983) Lipid peroxidation: a possible mechanism of cephaloridine-induced nephrotoxicity. Toxicol Appl Pharmacol 67(1):78-88.
15. Hazelton GA, Lang CA (1980) GSH content of tissue in aging mouse. Biochem J 188:25- 30.
16. Levine RL, Williams JA, Stadtman ER, Shacter E (1994) Carbonyl assays for determination of oxidatively modified proteins. Methods Enzymol 233:346-57.
17. Snezhkina AV, Kudryavtseva AV, Kardymon OL, Savvateeva MV, Melnikova NV, Krasnov GS, Dmitriev AA. ROS generation and antioxidant defense systems in normal and malignant cells. Oxidative medicine and cellular longevity. 2019;2019(1):6175804.
18. Kachhawa K, Varma M, Kachhawa P, Agrawal D, Shaikh M, Kumar S (2016) Study of dyslipidemia and antioxidant status in chronic kidney disease patients at a hospital in South East Asia. J Health Res Rev 3:28-30.
19. Kannan K, Jain S K (2000) Oxidative Stress and apoptosis. Pathophysiology 7:153-63.
20. Engwa GA, Nweke FN, Nkeh-Chungag BN. Free radicals, oxidative stress-related diseases and antioxidant supplementation. Alternative Therapies in Health & Medicine. 2022 Jan 1;28(1).
21. Van der Jagt DJ, Harrison JM, Ratliff DM, Hunsaker LA, Van der Jagt DL (2001) Oxidative stress indices in IDDM subjects with and without long-term diabetic complications. Clin Biochem 34:265–70.
22. Inouye M, Mio T, Sumino K (1999) Glycated hemoglobin and lipid peroxidation in erythrocytes of diabetic patients. Metabolism 48: 205–9.
23. Bhatia S, Shukla R, Venkata Madhu S, Kaur Gambhir J, Madhava Prabhu K (2003) Antioxidant status, lipid peroxidation and nitric oxide end products in patients of type 2 diabetes mellitus with nephropathy. Clin Biochem 36(7):557-62.

24. Conz PA, Bevilacqua PA, LaGreca G, Danieli D, Rodighiero MP, Cavarretta L, et al (1993) Phospholipid hydroperoxidase glutathione peroxidase in the normal human kidney: A possible role in protecting cell membranes. Exp Nephrol 1:376-8.
25. Waggiallah H, Alzohairy M (2011) The effect of oxidative stress on human red cells glutathione peroxidase, glutathione reductase level, and prevalence of anemia among diabetics. N Am J Med Sci 3:344-7.
26. Kumawat M, Sharma TK, Singh I, Singh N, Ghalaut VS, Vardey SK, et al (2013) Antioxidant enzymes and lipid peroxidation in Type 2 diabetes mellitus patients with and without nephropathy. N Am J Med Sci 5:213-9.
27. Nagarajrao R, Alharbi SA (2018) Relationship between oxidant and antioxidant enzymes status in type 2 diabetic patients with nephropathy in Saudi population. Asian J Pharm Clin Res 11(1):363-368.
28. Kuldip Singh, Gurpreet Singh (2017) Alterations in some oxidative stress markers in diabetic nephropathy. J Cardiovasc Disease Res 8(1): 24-27.
29. Arai K, Lizuka S, Tada Y, Oilkawa K, Taniguelui N (1987) Increase in the glycosylated form of erythrocyte Cu Zn SOD diabetes and association of non-enzymatic glycosylation with enzyme activity. Biochem Biophys Acta 924:292-6.
30. Kedziora-Kornatowska KZ, Luciak M, Blaszczyk Pawlak W (1998) Lipid peroxidation and activities of antioxidant enzymes in erythrocytes of patients with non-insulin dependent diabetes mellitus with or without nephropathy. Nephrol Dial Transplant 13:2829–32 .
31. Cakatay U (2005) Protein oxidation parameters in type 2 diabetic patients with good and poor glycaemic control. Diabetes Metab 31:551–557.