Journal of Cardiovascular Disease Research
ISSN: 0975-3583,0976-2833 VOL 13, ISSUE 4, 2022

A Study on Biochemical Profile and Oxidative Stress in Patients of Diabetic Chronic Kidney Disease at a Tertiary Care Centre of West Bengal: A Cross-Sectional Study.

Dr.Hare ram singh[1], Dr. Sunil Kumar[2] , Dr. Naresh Kumar Munda[3].

[1]Associate Professor, Department of Pharmacology, Faculty of Gouri Devi Institute of Medical sciences & hospital, Durgapur
[2]Associate Professor, Department of General Surgery, Faculty of Gouri Devi Institute of Medical sciences & hospital, Durgapur.
[3]Assistant Professor, Department of Community Medicine, Faculty of Icare Institute of Medical Sciences and Research and Dr. B C Roy Hospital, Haldia, India.

Corresponding Author: Dr. Naresh Kumar Munda, **E mail**: drnaresh2k@gmail.com.

Received-9.08.2022, Accepted-5.09.2022, published-4.10.2022

ABSTRACT

Background: Diabetic chronic kidney disease (CKD) is one of the most prevalent and debilitating complications of type 2 diabetes mellitus. Oxidative stress plays a pivotal role in the progression of renal damage in diabetic patients. **Objective:** To study and evaluate the biochemical profile and markers of oxidative stress in patients diagnosed with diabetic CKD attending a tertiary care centre in West Bengal. **Methods:** A hospital-based cross-sectional study was conducted on 66 patients diagnosed with diabetic CKD (Stages 3–5). Biochemical parameters including serum creatinine, blood urea, eGFR, HbA1c, fasting blood glucose, lipid profile, and oxidative stress markers such as malondialdehyde (MDA), superoxide dismutase (SOD), and catalase were measured. **Results:** The mean age of participants was 54.8 ± 9.6 years with a male predominance (63.6%). Significantly elevated levels of MDA (6.82 ± 1.74 nmol/mL) and markedly reduced levels of SOD (1.24 ± 0.38 U/mL) and catalase (18.6 ± 4.9 U/mL) were observed. Renal and glycaemic parameters were markedly deranged across all CKD stages. **Conclusion:** Diabetic CKD patients exhibit significant oxidative stress alongside deranged biochemical parameters. Routine assessment of oxidative stress markers may aid in early identification of progressive renal injury and help guide therapeutic strategies.

Keywords*: Diabetic CKD, oxidative stress, MDA, SOD, catalase, biochemical profile, West Bengal.*

1. INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder of global significance, and its burden in India continues to grow at an alarming pace. With an estimated 101 million diabetics in India as of recent surveys, the country holds the unenviable distinction of being the 'diabetes capital' of the world. Chronic kidney disease (CKD) resulting from diabetes — termed diabetic nephropathy or diabetic CKD — is one of the leading causes of end-stage renal disease (ESRD) worldwide, accounting for nearly 40–50% of all dialysis-dependent patients[1].

In West Bengal, a densely populated state of eastern India, the dual burden of diabetes and CKD poses a significant public health challenge. Patients in rural and peri-urban areas often present late to tertiary care facilities, by which time considerable renal dysfunction has already set in. The biochemical derangements seen in diabetic CKD are multifactorial and involve alterations in glycaemic control, renal function markers, lipid metabolism, and inflammatory pathways[2].

Of particular scientific interest in recent years has been the role of oxidative stress in the pathogenesis and progression of diabetic CKD. Oxidative stress refers to an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defence mechanisms. In diabetic CKD, chronic hyperglycaemia leads to excessive generation of free radicals, which in turn cause lipid peroxidation, protein modification, and DNA damage — all of which accelerate glomerular and tubular injury. Malondialdehyde (MDA), a product of lipid peroxidation, serves as a reliable marker of oxidative stress, while enzymatic antioxidants such as superoxide dismutase (SOD) and catalase represent the body's natural defence mechanisms[3].

Despite a growing body of literature on diabetic CKD at the national level, region-specific data from West Bengal, particularly at the tertiary care level, remain sparse. This study was therefore designed to evaluate the biochemical profile and oxidative stress markers in patients with diabetic CKD attending a tertiary care centre in West Bengal and to explore their clinical correlations[4].

2. OBJECTIVES

The primary objective of this study was to assess the biochemical profile — including renal function tests, glycaemic indices, and lipid parameters — in patients of diabetic CKD across CKD Stages 3 to 5. The secondary objectives were to evaluate oxidative stress markers (MDA, SOD, and catalase) in the study population, to correlate these markers with the severity of CKD staging, and to describe the sociodemographic characteristics of the study participants attending a tertiary care centre of West Bengal.

3. METHODOLOGY

3.1 Study Design and Setting

This was a hospital-based, cross-sectional observational study conducted at the Department of Medicine and Biochemistry of a tertiary care medical college in West Bengal, India. The study period extended over twelve months.

3.2 Sample Size Calculation

The sample size was calculated using the standard formula for estimating a population proportion:

$$\mathbf{n = Z^2 \times p \times q / d^2}$$

Where: Z = 1.96 (for 95% confidence interval), p = 0.40 (assumed prevalence of oxidative stress abnormality in diabetic CKD based on prior literature), q = 1 – p = 0.60, d = 12% (allowable error, i.e., 0.12).

Calculation: $n = (1.96)^2 \times 0.40 \times 0.60 / (0.12)^2 = 3.84 \times 0.24 / 0.0144 = 0.9216 / 0.0144 \approx 64$. Adjusting for 3% non-response, the final sample size was rounded up to 66 patients.

3.3 Sampling Method

A non-probability purposive sampling technique was employed. Patients attending the outpatient and inpatient departments who satisfied the inclusion criteria were consecutively enrolled until the required sample size was achieved. This method was chosen given the specific clinical diagnosis required and the finite availability of eligible patients within the study period.

3.4 Inclusion and Exclusion Criteria

Inclusion criteria: Patients aged 30 years and above with an established diagnosis of type 2 diabetes mellitus (as per ADA guidelines) and CKD Stages 3 to 5 (eGFR < 60 mL/min/1.73m²) were included. Both sexes were eligible. Exclusion criteria: Patients with non-diabetic CKD, acute kidney injury, active malignancy, severe hepatic dysfunction, pregnancy, or those already on haemodialysis or peritoneal dialysis were excluded. Patients who had received antioxidant supplementation within the past three months were also excluded to avoid confounding.

3.5 Data Collection and Biochemical Analysis

After obtaining written informed consent from each participant, a detailed clinical history and physical examination were recorded on a structured proforma. Venous blood samples (10 mL fasting) were collected. Biochemical analyses — including serum creatinine, blood urea, eGFR (calculated using CKD-EPI equation), HbA1c, fasting blood glucose, total cholesterol, triglycerides, LDL, HDL, serum albumin, and haemoglobin — were performed on a fully automated biochemistry analyser. Oxidative stress markers (MDA by thiobarbituric acid reactive substances [TBARS] method; SOD and catalase by spectrophotometric methods) were measured in the research biochemistry laboratory. Ethical clearance was obtained from the Institutional Ethics Committee prior to commencement.

3.6 Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 22.0 (IBM Corp.). Continuous variables were expressed as mean ± standard deviation (SD). Categorical variables were expressed as frequencies and percentages. Correlation between oxidative stress markers and CKD stages was assessed using Spearman's rank correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

4. RESULTS

4.1 Sociodemographic Profile

A total of 66 patients with diabetic CKD were enrolled in the study. The sociodemographic characteristics of the study participants are presented in Table 1. The majority of participants (47.0%) were in the 45–59 year age group, and the mean age was 54.8 ± 9.6 years. Male patients constituted 63.6% of the study population. A slight majority of participants (56.1%) hailed from rural areas. With regard to educational attainment, 18.2% of participants were illiterate, while 33.3% had completed secondary level education. A significant proportion (40.9%) were engaged in farming or manual labour. Regarding the duration of diabetes, the largest group (43.9%) had been living with diabetes for 5–10 years. In terms of CKD staging, 42.4% were in Stage 4, making it the most prevalent stage in the study group.

Table 1: Sociodemographic Profile of Study Participants (n = 66)

Sociodemographic Variable	Category	n = 66 (%)
Age Group (Years)	30–44	14 (21.2%)
	45–59	31 (47.0%)
	60–74	18 (27.3%)
	≥75	03 (4.5%)
Gender	Male	42 (63.6%)
	Female	24 (36.4%)
Residence	Urban	29 (43.9%)
	Rural	37 (56.1%)
Educational Status	Illiterate	12 (18.2%)
	Primary	21 (31.8%)
	Secondary	22 (33.3%)
	Graduate & above	11 (16.7%)
Occupation	Farmer/Labour	27 (40.9%)
	Service/Business	18 (27.3%)

Sociodemographic Variable	Category	n = 66 (%)
	Housewife	14 (21.2%)
	Retired/Unemployed	07 (10.6%)
Duration of Diabetes (years)	< 5 years	16 (24.2%)
	5–10 years	29 (43.9%)
	> 10 years	21 (31.9%)
CKD Stage	Stage 3	22 (33.3%)
	Stage 4	28 (42.4%)
	Stage 5	16 (24.3%)

4.2 Biochemical and Oxidative Stress Parameters

The mean values of all biochemical and oxidative stress parameters are summarised in Table 2. The mean serum creatinine was 4.72 ± 1.93 mg/dL, and mean blood urea was 87.6 ± 26.4 mg/dL — both markedly elevated above the normal reference range — reflecting advanced renal dysfunction in the study cohort. The mean eGFR was 22.4 ± 11.8 mL/min/1.73m², consistent with CKD Stage 4. Glycaemic control was poor across the group, with a mean HbA1c of 9.1 ± 1.6% and mean fasting blood glucose of 187.4 ± 48.7 mg/dL. Lipid parameters showed hypercholesterolaemia (total cholesterol 218.6 ± 42.1 mg/dL) and hypertriglyceridaemia (176.9 ± 55.3 mg/dL), indicating a dyslipidaemic state.

Regarding oxidative stress markers, MDA was significantly elevated at 6.82 ± 1.74 nmol/mL — nearly double the upper limit of the normal range — indicating heightened lipid peroxidation. In contrast, the antioxidant enzymes SOD (1.24 ± 0.38 U/mL) and catalase (18.6 ± 4.9 U/mL) were markedly reduced compared to their respective reference values, signifying a depletion of the antioxidant defence system. Serum albumin was low at 3.2 ± 0.6 g/dL, indicating nutritional compromise, and mean haemoglobin was 9.4 ± 1.9 g/dL, consistent with the anaemia of CKD.

A statistically significant positive correlation was observed between MDA levels and CKD stage ($r = 0.68$, $p < 0.001$). Conversely, SOD and catalase showed a significant negative correlation with advancing CKD stage ($r = -0.61$ and -0.57 respectively, $p < 0.001$). HbA1c showed a significant positive correlation with MDA ($r = 0.52$, $p < 0.01$), further supporting the link between poor glycaemic control and increased oxidative damage.

Table 2: Biochemical and Oxidative Stress Parameters in Study Participants (n = 66)

Biochemical Parameter	Mean ± SD	Reference Range
Serum Creatinine (mg/dL)	4.72 ± 1.93	0.7–1.2
Blood Urea (mg/dL)	87.6 ± 26.4	15–45

Biochemical Parameter	Mean ± SD	Reference Range
eGFR (mL/min/1.73m²)	22.4 ± 11.8	> 60
HbA1c (%)	9.1 ± 1.6	< 5.7
Fasting Blood Glucose (mg/dL)	187.4 ± 48.7	70–100
Total Cholesterol (mg/dL)	218.6 ± 42.1	< 200
Triglycerides (mg/dL)	176.9 ± 55.3	< 150
MDA (nmol/mL)	6.82 ± 1.74	1.5–3.5
SOD (U/mL)	1.24 ± 0.38	2.5–5.0
Catalase (U/mL)	18.6 ± 4.9	30–65
Serum Albumin (g/dL)	3.2 ± 0.6	3.5–5.0
Haemoglobin (g/dL)	9.4 ± 1.9	12–17

5. DISCUSSION

The findings of the present study provide a comprehensive picture of the biochemical derangements and oxidative stress burden in patients with diabetic CKD at a tertiary care centre in West Bengal. The predominance of male patients (63.6%) and the middle-aged demographic (mean age 54.8 years) are consistent with published literature from similar settings in India, where men tend to seek hospital care more readily and diabetes tends to manifest clinically in the fourth to sixth decade of life[5].

The severely elevated renal function markers — serum creatinine (4.72 mg/dL) and blood urea (87.6 mg/dL) — reflect the advanced stage of CKD in a significant proportion of study participants at the time of enrolment. This is not an uncommon observation in tertiary care centres of eastern India, where late presentation due to limited healthcare access and poor disease awareness remains a pressing challenge. The mean eGFR of 22.4 mL/min/1.73m² placing most patients in Stage 4 underscores the urgent need for timely nephrology referral in diabetic patients[6].

Poor glycaemic control, as evidenced by mean HbA1c of 9.1%, is a cardinal finding in this study and is in alignment with data from comparable Indian studies. Persistent hyperglycaemia activates multiple pathways — including the polyol pathway, advanced glycation end product (AGE) formation, and protein kinase C activation — all of which converge to generate reactive oxygen species (ROS) and propagate oxidative stress[7]. The dyslipidaemia observed in this cohort, particularly elevated triglycerides and total cholesterol, further compounds the oxidative burden on the already-injured kidneys[8].

The most clinically significant finding of this study is the marked elevation in MDA and concomitant reduction in SOD and catalase levels. MDA, a terminal product of lipid peroxidation, is widely accepted as a reliable biomarker of systemic oxidative stress. The near-doubling of MDA levels beyond the normal reference range in our study population reflects an overwhelming

oxidative challenge at the cellular level. This finding is consistent with studies by Bhatt et al. (2013) and Suryakar et al. (2006), who similarly reported elevated MDA in Indian patients with diabetic nephropathy[9].

The depletion of SOD and catalase — the two most important enzymatic antioxidant shields — indicates that the body's natural defence mechanisms are unable to neutralise the surge of free radicals generated in diabetic CKD. This antioxidant insufficiency may accelerate glomerular hypertrophy, mesangial expansion, and tubular epithelial damage, hastening the progression toward ESRD. The strong positive correlation between MDA and CKD stage ($r = 0.68$, $p < 0.001$) and the inverse correlation of SOD and catalase with disease severity further strengthen this pathophysiological hypothesis.

The low serum albumin and anaemia of CKD noted in the study group are consistent with the malnutrition-inflammation complex that is well recognised in advanced CKD. Hypoalbuminaemia in itself can impair the transport and activity of endogenous antioxidants, thereby amplifying oxidative stress. The rural predominance in this sample (56.1%) and the low educational status of a significant proportion of participants point toward socioeconomic and literacy barriers as contributing factors to late presentation and inadequate glycaemic control[10].

These findings collectively suggest that routine assessment of oxidative stress markers alongside conventional biochemical parameters should be considered an integral part of the evaluation of diabetic CKD patients. Therapeutic strategies targeting oxidative stress — such as optimisation of glycaemic control, use of renin-angiotensin-aldosterone system (RAAS) blockers, dietary antioxidant supplementation, and possibly novel antioxidant pharmacotherapy — may offer additional renoprotection in this high-risk group.

6. CONCLUSION

This cross-sectional study conclusively demonstrates that patients with diabetic CKD at a tertiary care centre in West Bengal carry a substantially elevated oxidative stress burden, characterised by increased MDA and depleted antioxidant enzyme activity, alongside significantly deranged biochemical parameters. The severity of oxidative stress correlates meaningfully with advancing CKD stage and poor glycaemic control. These findings highlight the importance of integrating oxidative stress assessment into routine clinical monitoring of diabetic CKD patients. Public health efforts must also focus on early diabetes detection, education, and timely nephrology referral to prevent the progression of renal disease in this vulnerable population.

7.Declaration

Conflict of Interest: The authors declare no conflict of interest.

Source of Funding: The study received no external funding. It was conducted as a departmental academic research project.

Acknowledgements: The authors express their sincere gratitude to the nursing staff, physiotherapy team, and the patients of the Department of . General Medicine and department of Biochemistry, IIMSAR Haldia Purba Medinipur, for their co-operation and support in the conduct of this study.

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