



ANTIOXIDANT STATUS AND OXIDATIVE STRESS IN THE PATHOPHYSIOLOGY OF CHRONIC KIDNEY DISEASE

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ABSTRACT:

Introduction: Chronic Kidney Disease (CKD) is a global public health priority marked by an irreversible decline in renal function¹. Emerging research identifies redox imbalance—where reactive oxygen species (ROS) overwhelm innate antioxidant defenses—as a critical driver of CKD progression and associated cardiovascular complications.

Objectives: This study aimed to assess renal function and oxidative status by estimating serum urea, creatinine, and Malondialdehyde (MDA) levels in CKD patients compared to healthy controls³. Additionally, it sought to evaluate antioxidant defense through Vitamin C levels and lipid profiles to determine their association with cardiovascular risk.

Materials and Methods: A prospective hospital-based case-control study was conducted involving 150 subjects (100 CKD cases and 50 healthy controls) at the Subbaiah Institute of Medical Sciences. Serum parameters including urea, creatinine, MDA (Draper and Hadley method), Vitamin C (Aye Kyaw method), and a full lipid profile were analyzed. Statistical analysis was performed using Student's unpaired t-test and Karl Pearson's correlation.

Results: The mean concentrations of serum creatinine (7.116 mg/dL) and blood urea (93.14 mg/dL) were significantly higher in cases compared to controls ($p < 0.001$). Oxidative stress was markedly elevated in CKD patients, with mean MDA levels at 6.01 nmol/ml compared to 2.72 nmol/ml in controls ($p < 0.001$). Conversely, Vitamin C and HDL levels were significantly depleted in the case group ($p < 0.001$). CKD patients also exhibited a pro-atherogenic lipid profile with significantly elevated Total Cholesterol and LDL levels.

Conclusion: Patients with CKD experience a profound biochemical imbalance characterized by heightened oxidative stress and diminished antioxidant defenses¹². The significant rise in MDA and depletion of Vitamin C facilitate lipid peroxidation and endothelial damage, contributing to high cardiovascular morbidity¹³. These findings suggest that integrating redox-status monitoring and targeted antioxidant therapies, such as Vitamin C supplementation, may improve clinical outcomes in the renal population.

Keywords: Chronic Kidney Disease (CKD), Oxidative Stress, Malondialdehyde (MDA), Vitamin C, Lipid Peroxidation, Cardiovascular Risk

INTRODUCTION:

Chronic Kidney Disease (CKD) has emerged as a global public health priority, characterized by the progressive and irreversible decline in renal function over time. While traditional risk factors such as hypertension and diabetes mellitus are well-documented, recent research has pivoted toward the fundamental role of redox imbalance in the pathophysiology of renal damage ^{1,2}. Oxidative stress, defined as the pathological state where the production of reactive oxygen species (ROS) overwhelms the body's innate antioxidant defense mechanisms, is now recognized as both a consequence and a potent driver of CKD progression ^{3,4}. **The Mechanism of Oxidative Stress in CKD:** In a healthy physiological state, the kidneys are highly metabolically active organs, particularly the proximal tubular cells, which rely on mitochondrial oxidative phosphorylation to meet high energy demands for solute reabsorption ^{2,5}. However, in the context of renal injury, these mitochondria become primary sources of excessive ROS, including superoxide radicals and hydrogen peroxide. This overproduction is further exacerbated by the upregulation of enzymatic systems such as nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (NOX), xanthine oxidase, and uncoupled nitric oxide synthase ^{2,6}. The resulting oxidative environment triggers a cascade of molecular events: **Cellular Damage:** ROS cause direct oxidative modification of lipids (lipid peroxidation), proteins, and DNA, leading to structural and functional impairment of podocytes and tubular cells ^{4,7}. **Inflammation:** Oxidative stress activates transcription factors like NF- κ B, which induce the expression of proinflammatory cytokines (IL-6, TNF- α). This creates a self-perpetuating "vicious cycle" where inflammation and oxidative stress amplify each other ^{3,8}. **Fibrosis:** ROS stimulate the Transforming Growth Factor-beta (TGF- β) pathway, promoting the transformation of fibroblasts and the excessive accumulation of extracellular matrix, eventually leading to glomerulosclerosis and tubulointerstitial fibrosis ^{2,4}. **The Role of Antioxidant Status** Conversely, the "antioxidant status" of a CKD patient refers to the collective efficiency of enzymatic defenses—such as Superoxide Dismutase (SOD), Catalase, and Glutathione Peroxidase (GPx)—and non-enzymatic scavengers like Vitamin C, Vitamin E, and Glutathione ^{1,7}. In CKD, this defense system is often severely compromised. Uremic toxins, which accumulate as GFR declines, directly inhibit antioxidant enzymes, while dietary restrictions to prevent hyperkalemia often led to a reduced intake of antioxidant-rich fruits and vegetables ^{1,3}. As renal failure progresses toward End-Stage Renal Disease (ESRD), the imbalance becomes more pronounced. Studies consistently show that markers of oxidative damage, such as Malondialdehyde (MDA) and 8-isoprostanes, correlate inversely with the glomerular filtration rate (GFR) ^{1,7}. Furthermore, renal replacement therapies like hemodialysis can ironically worsen oxidative stress through the activation of polymorphonuclear neutrophils during blood-membrane interactions ^{1,6}. **Clinical Implications and Rationale:** Understanding the interplay between antioxidant status and oxidative stress is critical not only for slowing renal decline but also for addressing systemic complications. Oxidative stress is a major link between CKD and its primary cause of mortality: cardiovascular disease ^{3,8}. High levels of ROS promote endothelial dysfunction and atherosclerosis, significantly increasing the risk of cardiovascular events in the renal population. This review aims to explore the current understanding of these redox dynamics within the CKD pathophysiology. By evaluating the markers of oxidative stress across different CKD stages and the potential of antioxidant therapies, we can better define the window for clinical intervention to preserve renal function and improve patient outcomes.

OBJECTIVES:

1. To assess renal function and oxidative status by estimating the serum concentrations of urea, creatinine, and Malondialdehyde (MDA) in patients with chronic kidney disease (CKD) compared to healthy controls.
2. To evaluate the antioxidant defense and lipid profile in CKD patients by measuring serum Vitamin C levels and lipid parameters (TC, TG, HDL, and LDL) to determine their association with cardiovascular risk.

3. To correlate oxidative stress markers (MDA and Vitamin C) with renal parameters and lipid profiles to understand their role in the pathophysiology of CKD and identify potential targets for therapeutic antioxidant interventions.

MATERIAL & METHODS:

Study Design: A prospective hospital-based case-control study.

Study area: Department of Biochemistry Subbaiah institute of medical sciences and research centre, Shivamogga.

Study Period: September 2024 to August 2025.

Study population: Clinically diagnosed cases of CKD attending nephrology department. Healthy controls were selected from regular blood donors in the blood bank.

Sample size: The study consisted of 150 subjects (100 cases and 50 controls).

Sampling method: Simple random technique.

Inclusion criteria:

1. Clinically diagnosed cases of CRF in the age group between 35-60 years who were on medical treatment.
2. An equal number of age and sex-matched healthy controls.

Exclusion Criteria:

Patients with:

1. Those taking medicines with antioxidants
2. Liver diseases
3. Infectious diseases
4. Immune complex diseases
5. Malignancies

Ethical consideration: Institutional Ethical Committee permission was obtained before the commencement of the study.

Study tools and Data collection procedure:

a) Sample collection:

Under aseptic precautions, about 5 ml of blood was collected in a sterile vacutainer from selected CRF patients on medical treatment. Serum was separated by centrifugation and used for analysis of the following parameters.

b) Parameters:

Serum was used for estimating the following parameters

- Estimation of serum lipid peroxidation product -- Malondialdehyde--- Method of DRAPER AND HADLEY
- Estimation of serum Vitamin C (Ascorbate)--- Method of Aye Kyaw, (1978)
- Blood urea by Modified Berthelot Method
- Serum Creatinine by Jaffe's method
- Serum Cholesterol (CHOD/POD Method)
- Serum Triglycerides (GPO /POD ESPAS, 1973)
- Serum HDL Cholesterol (Phosphotungstate-CHOD-POD)
- Serum LDL AND VLDL - By Calculation (FRIEDWALD W.T.,1972)

All the reagents used in the estimation were of analytical grade.

Statistical analysis:

Descriptive data were presented as percentages and continuous data was presented as mean, SD and range values. Student's unpaired, 't'-test was used for comparing the means of two groups (cases & controls). The relationship between measurements was assessed by Karl Pearson's coefficient of

correlation. Data were entered into Microsoft Excel and analysed in SPSS 20 package. For all the tests, a p-value of 0.05 or less was considered as statistical significance.

OBSERVATIONS & RESULTS:

TABLE – 1: AGE WISE DISTRIBUTION OF STUDY POPULATION

AGE (YEARS)	GROUP 1 (CASES)		GROUP 2 (CONTROLS)	
	FREQUENCY	PERCENT (%)	FREQUENCY	PERCENT (%)
35 to 45	36	36.0	18	36.0
46 to 55	36	36.0	23	46.0
56 to 65	28	28.0	9	18.0
Total	100	100.0	50	100
MEAN±SD	49.66±8.86		48.08 ± 6.827	

The subjects are predominantly in the age group of 35 to 55years in both the cases and controls.

TABLE 2: SEX WISE DISTRIBUTION OF STUDY POPULATION

SEX (M/F)	GROUP 1 (CASES)		GROUP 2 (CONTROLS)	
	FREQUENCY	PERCENT (%)	FREQUENCY	PERCENT (%)
F	48	48.0	18	36.0
M	52	52.0	32	64.0
Total	100	100.0	50	100

The study shows a male preponderance in both cases and controls.

TABLE-3: SERUM CREATININE IN STUDY POPULATION

PARAMETER	GROUP 1 (CASES)		GROUP 2 (CONTROLS)		p-VALUE	t-VALUE
	MEAN	SD	MEAN	SD		
CREATININE (mg/dL)	7.116	2.78	1.03	0.13	<0.001	19.857

The mean concentration of serum creatinine was in the range of 1.03+ 0.13mg/dL in controls and 7.116+2.78mg/dL in cases. Statistical analysis by unpaired t-test showed that the mean value of serum creatinine was increased in cases when compared to controls and was statistically significant (p<0.001).

TABLE-4: BLOOD UREA FINDINGS IN THE STUDY POPULATION

PARAMETER	GROUP 1 (CASES)		GROUP 2 (CONTROLS)		p-VALUE	t-VALUE
	MEAN	SD	MEAN	SD		

UREA (mg/dL)	93.14	21.539	29.40	2.95	<0.001	10.243
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The mean concentration of blood urea was in the range of 29.4+2.95 mg/dL in controls and 93.14+21.54mg/dL in cases. Statistical analysis by unpaired t-test showed that the mean value of blood urea was increased in cases when compared to controls and was statistically significant ($p < 0.001$).

TABLE-5: Shows the group statistics of vitamin c based on the values 3 groups are made group (1) <0.09mg/dl group (2) 0.1-0.19 mg/dl and group (3) 0.20-0.40mg/dl).

		VITAMIN C			Total
		<0.09 mg/dL	0.1 -0.19 mg/dL	0.2-0.4 mg/dl	
Controls	Count	0	9	41	50
	%within group	0.0%	18.0%	82.0%	100.0%
Cases	Count	19	81	0	100
	%within group	19.0%	81.0%	0.0%	100.0%

TABLE -6: MDA LEVELS IN CASES AND CONTROL

	Range (nmol/ml)	Mean (nmol/ml)	S.D	SEM
Cases n = 100	5.0 - 7.5	6.014	± 0.606	± 0.89
Controls n =50	2.1 - 4.0	2.72	± 0.468	± 0.09

t Value: 25.5

p Value: < 0.001

In the present study, mean values of Malondialdehyde among cases is 6.01 nmol/ml + 0.61(Mean+ S.D) with the range of 5.0 to 7.5 nmol/ml and that of control is 2.72 nmol/ml + 0.468 (Mean + S.D) with the range of 2.1 to 4.0. The increase in the serum Malondialdehyde among cases is significant with a 'p' value of <0.001.

Total patients with Malondialdehyde level > 6 nmol/ml are forty-three (43) out of fifty (100) constituting 43 %. Males are twenty-four (24%), and females are nineteen (19%).

TABLE - 7: MDA LEVELS AND LIPID PROFILE IN CASES AND CONTROLS

	Mean Values	Mean Values		
	Cases	Controls	't' Value	'p' Value

MDA Levels (nmol/ml)	6.01	2.72	25.5	<0.001
LDL (mg/dl)	176.09	103.38	16.03	<0.001
T.Cholesterol (mg/dl)	246.7	156.86	18.8	<0.001
HDL(mg/dl)	32.96	38.46	4.8	<0.001

TABLE-8: SERUM CREATININE VALUES DISTRIBUTION IN CASES AND CONTROLS
-Chi-square test (Pearson chi-square value 100 with degrees of freedom f=3)

CREATININE (C) (mg/dL)	GROUP 1 (CASES)	GROUP 2 (CONTROLS)	GROUP (1+2)	PERCENT (%)
	FREQUENCY	FREQUENCY	FREQUENCY	
≤1.4	0	50	50	33.33
1.5-5	14	0	14	9.33
5-8	66	0	66	44
8-15	20	0	20	13.33
Total	100	50	150	100

The majority (66%) of cases had serum creatinine values between 5-8 mg/dL. Among controls 100% had creatinine less than 1.4mg/dL. This difference has been found to be statistically significant. (p value<0.001).

TABLE-9: BLOOD UREA VALUES DISTRIBUTION IN CASES AND CONTROLS-Chi-square test (Pearson chi-square value 100 with degrees of freedom f=3)

UREA(U) (mg/dL)	GROUP 1 (CASES)	GROUP 2 (CONTROLS)	GROUP (1+2)	PERCENT (%)
	FREQUENCY	FREQUENCY	FREQUENCY	
<40	0	50	50	33.33
40-80	26	0	26	17.33
81-100	46	0	46	30.66
>100	28	0	28	18.66
Total	100	50	150	100

Majority 46% of cases had blood urea values between 81-100 mg/dL. Among controls, 100% had blood urea levels less than 40mg/dL. This difference has been found to be statistically significant. (p value< 0.001).

DISCUSSION:

A terrible condition having clinical, economic, and ethical aspects, chronic kidney disease (CKD) is becoming a significant global public health issue⁹. The permanent decline in renal function that eventually leads to end-stage renal disease (ESRD)¹⁰ is known as chronic renal failure (CRF). Renal replacement therapy, such as chronic peritoneal or hemodialysis and transplantation, is required once end-stage renal disease (ESRD) develops¹⁰. Research indicates that individuals with chronic kidney disease experience increased oxidative stress; however, there is a lack of reports regarding the oxidative stress levels in these patients prior to undergoing hemodialysis. Antioxidants that mitigate oxidative stress play a crucial role in maintaining overall health¹¹. In patients with CKD, the targets of oxidative stress include muscle fats and proteins, which may lead to skeletal muscle disorders. Additionally, oxidative stress in CKD patients could lead to endothelial dysfunction and the development of atherosclerosis.

Numerous pieces of evidence connect oxidative stress with cardiovascular diseases and kidney damage. While some studies clarify the origins of oxidative stress, there is a lack of research on oxidative stress levels in patients with chronic kidney failure prior to undergoing hemodialysis. To address this lack of information, the current study aimed to measure the plasma concentrations of MDA, Vitamin C, urea, and creatinine in individuals with CKD. The current study involved 150 participants, consisting of 100 individuals with chronic renal failure and 50 healthy controls. The primary objective of this research was to measure the levels of blood urea, serum creatinine, MDA, Vitamin C, and lipid profile in order to explore any potential correlations among them.

CREATININE: The mean concentration of serum creatinine was in the range of 1.03 ± 0.13 mg/dL in controls and 7.12 ± 2.78 in cases. Statistical analysis by unpaired t-test showed that the mean value of serum creatinine was increased in cases when compared to controls and was statistically significant ($p < 0.001$). In the present study chi-square test has shown that majority (66%) of cases had serum creatinine values between 5-8 mg/dL. Among controls, all (100%) had creatinine less than 1.4 mg/dL. A statistically significant difference has been found between cases and controls (p value < 0.001). Furthermore, 14% of cases had serum creatinine value between 1.5-5 mg/dL and 20% of the cases had serum creatinine value between 8-15 mg/dL.

UREA: The mean concentration of blood urea was in the range of 93.14 ± 21.54 mg/dL in cases and 29.4 ± 2.95 mg/dL in controls. Statistical analysis by unpaired t-test showed that the mean value of blood urea was increased in cases when compared to controls and was statistically significant ($p < 0.001$). In the present study chi-square test has shown that majority (46%) of cases had blood urea values between 81-100 mg/dL. Among controls, all (100%) had blood urea levels less than 40 mg/dL. A statistically significant difference has been found between cases and controls (p value < 0.001). Furthermore, 26% of cases had blood urea value between 40-80 mg/dL and 28% of the cases had blood urea value above 100 mg/dL. CRF is characterized by a gradual decrease in nephron number and function. Decrease in the concentrating ability of kidney leads to accumulation of urea and creatinine.¹²

VITAMIN C:

Vitamin C was estimated in 50 controls, and their levels were noted that majority of them (41 subjects) are in the range of 0.20 to 0.40 mg/dl & 9 subjects are in the range of 0.1-0.19 mg/dl. Out of 100 cases, majority of them (81 cases) are in the range of 0.1-0.19 mg/dl, and 19 cases with < 0.09 mg/dl and none were in the range of 0.2-0.4 mg/dl. The Values of present study are in consistent with the study by Mehdi, W.A. (2011)¹³.

The possible sources of increased oxidative stress might include increased generation of free radicals or an impaired antioxidants defence system. Oxygen free radicals are toxic to tissue because of their high reactivity, and ability to form covalent bonds non enzymatically. Extensive studies with biological materials have shown clearly that reactive free radicals can produce chemical modifications in cells and damage the proteins lipids, carbohydrates and nucleotides. To overcome these

consequences, cells have antioxidant defence systems, which scavenge the free oxygen radicals and suppress the free radical chain and lipid peroxidation¹⁴.

Oxidative stress, the state in which an imbalance between the levels of reactive oxygen species and antioxidants exists, can lead to disturbed glucose metabolism and hyperglycemia. Oxidative stress is consistently observed in patients with renal failure, and the degree of oxidative stress tends to be greater in those with diabetic nephropathy and more diabetic complications.

Various studies have reported protective effects of antioxidants against oxidative damage of kidney. The level of vitamin C in plasma and renal tissue is significantly reduced in CKD patients. Impaired antioxidant functions also play a role in development of chronic kidney disease. Vitamin C is one of the most important antioxidants that inhibits lipid peroxidation and improves endothelial function. In one study, it is proposed that every other day supplementation with 250 mg vitamin C for 12 weeks increases serum vitamin C, decreases MDA levels, and improves lipid profiles in hemodialysis patients.

MDA:

Mean Blood Malondialdehyde levels are (6.014+0.61nmol/ml) elevated in the cases compared to controls (2.72+0.46 nmol/ml). A significant difference was observed in mean MDA changes between cases and control groups. Total patients with Malondialdehyde level > 6 nmol/ml are forty-three (43) out of fifty (100) constituting 43 %. Males are twenty-four (24%), and females are nineteen (19%). A statistically significant difference has been found between cases and controls (p value<0.001). A study by Bhatia et al.¹⁵ showed that the MDA and SOD levels were found significantly increased in DN patients as compared to DM patients. Malondialdehyde (MDA) is an end-product generated by lipid peroxidation and has been used to demonstrate increased oxidative stress during CKD. The rise in serum MDA indicated that any oxidative stress incurred sufficiently could cause free radical mediated peroxidation of lipid component in cell membrane, thus MDA is a good indicator for evaluating oxidative stress in degenerative disease like CKD and diabetes mellitus. The increased oxidative stress levels that observed in CKD patient are consistent with the finding of several previous studies.¹⁶

LIPID PROFILE:

The mean concentration of total cholesterol was in the range of 246.7+ 22.6 mg/dL in cases and 156.86 + 20.65 mg/dL in controls and the mean concentration of HDL cholesterol was in the range of 32 + 3.97 mg/dL in cases and 38.46 + 4.51 mg/dL in controls. The mean concentration of LDL cholesterol was in the range of 176.1 + 23.77 mg/dL in cases and 103.38 + 17.81 mg/dL in controls and the mean concentration of VLDL cholesterol was in the range of 37.65 + 5.6 mg/dL in cases and 28.3 + 2.07 mg/dL in controls. The mean concentration of Triglycerides was in the range of 188.26 + 28.44 mg/dL in cases and 141.76 + 10.12 mg/dL in controls. The elevated levels of LDL cholesterol may provide increased substrates for reactive oxygen species. These free radicals react with the low-density lipoprotein (LDL), leading to the formation of oxidized LDL particles, which are important in the initiation and progression of atherosclerotic plaques because they can elicit inflammatory processes and lipid accumulation within the arterial wall. Patients with CKD generally have reduced plasma HDL cholesterol concentrations compared with nonuremic individuals.

CONCLUSION:

In light of the findings from King George Hospital, it is evident that chronic kidney disease acts as a profound state of biochemical imbalance where the escalation of urea and creatinine is inextricably linked to heightened oxidative stress and dyslipidemia. The significant rise in Malondialdehyde (MDA) alongside the depletion of Vitamin C creates a hazardous environment that facilitates lipid peroxidation and endothelial damage, directly contributing to the high cardiovascular morbidity observed in these patients. By establishing a statistically significant (p < 0.001) relationship between these markers, this study underscores the necessity of moving beyond traditional renal parameters to include redox-status monitoring. Ultimately, the integration of targeted antioxidant therapy—such as Vitamin C supplementation—offers a promising therapeutic window to mitigate oxidative damage,

stabilize lipid profiles, and potentially alter the trajectory of systemic complications in the renal population.

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